A SYSTEM OF MEDICINE

BY MANY WRITERS

8vo. Half-bound.

Second Edition, edited by Sir CLIFFORD ALLBUTT, *K.C.B.,*
M.D., F.R.S., and H. D. ROLLESTON, *M.D., F.R.C.P.*

Original Edition.

Edited by Sir CLIFFORD ALLBUTT, K.C.B., M.D.

25s. net each Volume.

MACMILLAN AND CO., LTD., LONDON.

A SYSTEM OF MEDICINE

A

SYSTEM OF MEDICINE

BY MANY WRITERS

EDITED BY

Sir CLIFFORD ALLBUTT, K.C.B.

M.A., M.D., LL.D., D.SC., F.R.C.P., F.R.S., F.L.S., F.S.A.

REGIUS PROFESSOR OF PHYSIC IN THE UNIVERSITY OF CAMBRIDGE
FELLOW OF GONVILLE AND CAIUS COLLEGE

AND

HUMPHRY DAVY ROLLESTON

M.A., M.D., F.R.C.P.

SENIOR PHYSICIAN, ST. GEORGE'S HOSPITAL ; PHYSICIAN TO THE VICTORIA HOSPITAL
FOR CHILDREN ; SOMETIME FELLOW OF ST. JOHN'S COLLEGE, CAMBRIDGE

VOLUME VI

DISEASES OF THE HEART AND BLOOD-VESSELS

PREFACE

THE contents of this volume—The Diseases of the Heart and Blood-vessels—were partly in Volume V. and partly in Volume VI. of the original edition. The present instalment further differs from Volume VI. in the first edition in not containing the Diseases of Muscles and some of the Diseases of the Nervous System. This change has of course been dictated by the convenience of having the diseases of special parts of the body inclusively considered in separate volumes.

Considerable changes have necessarily been made in the individual articles. Professor Sherrington's original introductory article on Cardiac Physics has been revised by Dr. James Mackenzie, who has added an account of the peripheral circulation, including arterial blood-pressure; this justifies a change in the title of the article to that of Physics of the Circulation. Dr. J. Mackenzie's influence on this branch of medicine is further seen in the numerous tracings which he has generously placed at the disposal of the authors of various articles. An entirely new article on Stokes-Adams disease has been contributed by Professor Osler and Dr. Keith. The article on Over-Stress of the Heart has been rewritten, and contains a section by Dr. R. W. Michell, the out-come of his special opportunities of watching University athletes. The late Professor Dreschfeld's account of Simple Acute Endocarditis has been revised by Dr. T. M'Crae, and the Diseases of the Mitral

Valve have been brought up to date and more freely illustrated by Dr. G. A. Gibson. The account of Functional Disorders of the Heart has been largely re-written ; and a new article on Aneurysm has been contributed by Professor W. Osler.

The Editors are indebted to Dr. A. J. Jex-Blake for a number of corrections in the text, especially in the " References."

<div style="text-align: right">CLIFFORD ALLBUTT.

H. D. ROLLESTON.</div>

CONTENTS

ILLUSTRATIONS

COLOURED PLATES

FIGURES

LIST OF CONTRIBUTORS TO THIS VOLUME

Allbutt, Sir Clifford, K.C.B., M.A., M.D., LL.D., D.Sc., F.R.C.P., F.R.S., Regius Professor of Physic in the University of Cambridge; Fellow of Gonville and Caius College; Hon. Fellow of the Royal College of Physicians of Ireland, and of the Academy of Medicine of New York.

Clutton, H. H., M.C., Senior Surgeon, St. Thomas's Hospital.

Dreschfeld, The late Julius, M.D., M.Sc., F.R.C.P., Consulting Physician to the Manchester Royal Infirmary; and Professor of Medicine, Victoria University, Manchester.

Gibson, George Alexander, M.D., D.Sc., LL.D., Physician to the Royal Infirmary; Lecturer on Medicine and Clinical Medicine, Royal Colleges School of Medicine, Edinburgh.

Humphry, Laurence, M.D., F.R.C.P., Physician to Addenbrooke's Hospital; Lecturer on Medicine in the University of Cambridge.

Keith, Arthur, M.D., F.R.C.S., Conservator of the Museum, Royal College of Surgeons of England.

M'Crae, Thomas, M.D., F.R.C.P., Associate Professor of Medicine and Clinical Therapeutics, the Johns Hopkins University, Baltimore.

Mackenzie, James, M.D., M.R.C.P., Consulting Physician to the Victoria Hospital, Burnley.

Mott, Frederick Walker, M.D., F.R.C.P., F.R.S., Senior Physician, Charing Cross Hospital; Pathologist to the London County Asylums.

Oliver, Sir Thomas, M.D., LL.D., F.R.C.P., Physician, Royal Victoria Infirmary, Newcastle-upon-Tyne; late Medical Expert, Dangerous Trades Committee, Home Office.

Osler, William, M.D., LL.D., D.Sc., F.R.C.P., F.R.S., Regius Professor of Medicine, University of Oxford; Hon. Professor of Medicine, Johns Hopkins University, Baltimore; Student of Christ Church, Oxford.

Pitt, G. Newton, M.D., F.R.C.P., Physician and Lecturer on Medicine, Guy's Hospital; sometime Fellow of Clare College, Cambridge.

Powell, Sir R. Douglas, Bart., K.C.V.O., M.D., LL.D., D.Sc., Physician in Ordinary to H.M. the King; President of the Royal College of Physicians; Consulting Physician to the Middlesex Hospital, and to the Hospital for Consumption and Diseases of the Chest, Brompton.

Roberts, Frederick T., M.D., F.R.C.P., Consulting Physician to University College Hospital, and to the Hospital for Consumption and Diseases of the Chest, Brompton.

Rolleston, Humphry Davy, M.D., F.R.C.P., Senior Physician, St. George's Hospital ;
    Physician, the Victoria Hospital for Children ; sometime Fellow of St. John's
    College, Cambridge.

*Sansom, The late A. Ernest, M.D., F.R.C.P., Consulting Physician, London Hospital ;
    Fellow of King's College, London.*

Sherrington, Charles Scott, M.D., LL.D., D.Sc., F.R.S., Professor of Physiology,
    University of Liverpool.

Welch, William H., M.D., LL.D., Professor of Pathology, Johns Hopkins University,
    Baltimore.

In order to avoid frequent interruption of the text, the numbers indicative of items in the lists of "References" are only inserted in cases of emphasis, where two or more references to the same author are in the list, where an author is quoted from a work published under another name, or where an authoritative statement is made without mention of the author's name. In ordinary cases an author's name is sufficient indication of the corresponding item in the list.

DISEASES OF THE HEART

PHYSICS OF THE CIRCULATION

By Prof. C. S. Sherrington, M.D., LL.D., F.R.S.
Revised by James Mackenzie, M.D.

I. The Heart

The Auricular Systole.—The heart's contraction starts normally at the mouths of the great veins, and sweeps over the auricles and ventricles. There is a perceptible interval between the beginning of the auricular contraction and the beginning of the ventricular, but no interval can be detected between the contraction of the veins and of the auricles. When the auricle contracts the venous orifices are closed, and the contents are emptied through the auriculo-ventricular orifices into the ventricles. The closure of the venous orifices is due mainly to a thick band of muscle which extends over the roof of the auricles — the taenia terminalis — assisted by the circular fibres around the mouths of the veins, and in the case of the inferior cava by the remains of the Eustachian valve (Keith). Besides shutting off the veins, the taenia terminalis assists the pectinate fibres and remaining musculature of the auricular wall in emptying the auricle. The pectinate fibres run transversely over the roof of the auricles from the taenia terminalis to the auriculo-ventricular septum, and in their contraction, being fixed to the taenia terminalis, they draw up the septum to an extent of about half an inch (Keith).

The Cardiac Valves.—1. *Mechanism of the Auriculo-Ventricular Valves.* —At each systole of the ventricles the tongue-shaped valve-flaps pendent from the margins of the auriculo-ventricular orifices are moved together towards those orifices, and meeting together across them block them. By this means the blood in each ventricle is prevented from returning into the auricle, and, under the compression of the contracting ventricle, is forced to take its way into the great arteries. Were it not for these valves not a drop of the blood would enter the arteries, so long as the pressure in the latter possessed a value near the normal; but for the valves its issue would be far easier back into the cavity of the auricles where the pressures are low. During diastole of the ventricle the flaps of the auriculo-ventricular valves lie in the cavity of the ventricle with their long axes convergent towards the central long axis of the ventricle.

Between the valve-flaps and the inner face of the ventricular wall there is always an interval, and therefore always more or less blood (Baumgarten, Krehl). Manometric observations reveal no increase of pressure in the auricle at the moment of closure of the auriculo-ventricular valves. The discharge of its contents by the auricle into the quiescent and already partly-filled ventricle somewhat stretches the slack walls of this latter, and, whether by eddy or otherwise, the valve-flaps are raised toward each other and toward the auricular opening. Then, as the contraction of the auricle passes off, the pressure in the now fully-loaded ventricle becomes higher than in the relaxing auricle. The valve-flaps thus swing together into position, and are moved to meet across the auriculo-ventricular orifice, even before the ventricular systole has thoroughly set in. If the arterial openings of the excised heart be blocked, and through the auricles a momentary rush of water under about twelve inches pressure be allowed to play into the auriculo-ventricular orifices, the valve-flaps rise into the orifice, and come together sufficiently firmly to allow of the inversion of the heart without the escape of a drop of its contents.

The valve-flaps would be forced through the orifice back into the auricle were they not tied down to the ventricle by the chordae tendineae attached to almost all areas of their under surface. Each valve-flap shares in a pair of papillary muscles ; these latter are so placed in regard to the valve-flaps that the resultant of their combined individual directions of pull lies strictly along the long axis of the ventricular chamber, and at right angles to the plane of the auriculo-ventricular orifice itself (Ludwig).

The auricular face of each valve-flap in its position of closure is convex. The thin contiguous edges of the adjacent valve-flaps are bent abruptly downward, side by side, tightly apposed ; the tenuous edges of the membranes bear, therefore, no part of the great strain to which the valve elsewhere is subjected ; for these edges, projecting into the ventricular cavity, are supported on both sides by the fluid pressure of the blood in the ventricle. That this is the position of these parts of the valve is proved by the following among other considerations : the chordae tendineae which are inserted near the free margin of each valve-flap are much shorter than those inserted into the midrib of the flap.

Regarding the use of the papillary muscles, it has been shewn (Roy and Adami) that the papillary muscles begin to contract later than does the rest of the ventricle ; as the ventricle shortens from base to apex during systole, the papillary muscles, if they are to afford the chordae tendineae a suitably placed support, and to prevent retroversion of the valve-flaps into the auricle, must shorten in order to maintain their distance from the auricular orifice.

In the paragraph dealing with the auricular systole it was shewn that the pectinate fibres of the auricle in their contraction pulled up the auriculo-ventricular septum. With the relaxation of these fibres and the contraction of the muscle-fibres of the ventricle the auriculo-ventri-

cular septum is drawn back from the auricle (Chauveau, Keith). In consequence of this and of the contraction of the papillary muscles which draw down the auriculo-ventricular valves (Roy and Adami, Porter) a negative pressure is produced in the auricle during the earlier part of the ventricular systole, the auricular cavities are enlarged, and the blood is drawn into them from the veins (see fall x', Fig. 2).

It must not be forgotten that an important detail in the mechanism of the closure of the auriculo-ventricular orifices is the circularly arranged muscle surrounding those orifices as a true sphincter (Meckel, 1825). This sphincter appears to be important, especially for the tricuspid orifice the valves of which are barely competent to close the orifice (John Hunter). In the heart of the bird the tricuspid orifice is unprovided with valve-flaps, and its closure is effected wholly by a muscular sphincter, whilst in diving animals the incompetent tricuspid valves seem specially provided to permit regurgitation when the animals are under water (Wilkinson King).

2. *Mechanism of the Semilunar Valves.*—So long as the pressure in the ventricle is below the pressure in the great arterial trunk leading from it, so long will the semilunar valve-flaps meet across the arterial ostium and occlude it. When examined under a pressure approximately that of the aorta, the valve-flaps are seen to lie apposed across the orifice ; if one of the flaps be displaced gently towards its attached border, the other two cusps follow it, becoming correspondingly more stretched. The cusps, therefore, in the closed position of the valve mutually support one another. When during the systole of the ventricle the intra-ventricular pressure becomes higher than the aortic (or pulmonary) the valve-flaps yield, are moved apart, and leave between them a triangular opening.

When the valve is open, the position of the cusps is with their free edge convex toward the arterial wall, but the cusp-membrane does not lie apposed to or quite close against the wall. In the open position of the valve the arc formed by the curved wall of the sinus of Valsalva may be said to have its chord approximately represented by the free edge of the cusp. The supposition of Brücke that the cusps when the aortic valve is open are pressed back against the aortic wall, so as to block the entrances to the coronary arteries, is completely disproved.

The closure of the valves seems to be brought about in the following way :—During systole the cavity of the ventricle, where it adjoins the aortic opening, is narrowed by the bulging into it of the contracted muscular wall ; it forms, in fact, a narrow channel which ends in the direction of the aorta in the triangular cleft between the semilunar cusps in the wide root of the aorta with its triple circumferential bays—the sinuses of Valsalva. At the place where the narrow stream suddenly embouches into the wide aortic channel eddies are formed, curving back behind the valve-cusps, and constantly tending to bring these together. The cusps are, however, kept apart by the pressure of the blood flowing between them ; as soon, however, as that flow ceases the cusps rush together, as it were under the force of a spring. Ceradini's account of

the eddies which come into play on closing the valves is as follows :—If in a vertical tube containing water, in which visible particles are suspended, a piston at the lower end of the tube be pushed upward, the water in the axis of the tube is seen to move with nearly twice the velocity average for the whole column ; along the face of the wall of the tube the water moves so slowly that the piston overtakes the particles suspended in it.   As this occurs the particles are seen to be swept from the circumferential zone by a centripetal current conveying them into the axial stream.   Along this they rush upward to the free surface of the fluid, where they sweep outward in a centrifugal eddy to reach the wall of the tube again, there later once more to be overtaken by the piston and swept inwards in a centripetal eddy (inversion).   If the ascent of the piston be suddenly checked, the above currents in the fluid are modified to the extent that an actual back flow sets in downward along the inner face of the tube.   The result is that at the moment the piston stops, the column of water above it is split into two parts—into an axial cylinder moving forwards and a peripheral layer moving backwards, the two being connected above by a centrifugal eddy, below by a centripetal (inversion) eddy.   To this latter is due the bringing together into position the cusps closing the aortic opening.   The cusps thus brought together are held so by a mechanical force measurable in the left heart by the product of the difference between the aortic and ventricular pressures into the area of the valve-flap, excluding their margins.   It is probable that the cusps are partly supported under this strain by the thick bulging myocardium of the ventricular wall on which they may partly rest.

The Cardiac Sounds. — In 1810 Wollaston shewed that skeletal muscle, when it contracts under the will, emits a sound—the muscle-note. William Harvey had pointed out that the heart during its contraction emits sound (*rumor*).   Laennec (1819) was the first to recognise the diagnostic value of the heart-sounds.   The British Association Committee in 1836 declared the first cardiac sound to be the muscle-note of the ventricles, but their observations were not decisive.   Ludwig, in 1868, succeeded in proving clearly that when the heart is so placed as not to convey by its mass-movement a shock to any vibrator and at the same time is so inadequately filled as to exclude the possibility of tension of any of its valve-flaps, the first cardiac sound continues to be distinct.

But it has been shewn (Wintrich) by means of resonators that the normal heart-sound consists of two notes, the lower of which only can be considered a muscle-tone.   The higher is due to the vibration of the auriculo-ventricular cusps and the column of blood they support.   This seems clear because it can be heard if these valve-flaps are suddenly rendered tight in the dissected heart.   The first sound of the heart is therefore found to be due to the vibration of (a) the muscular wall of the ventricles, (β) the auriculo-ventricular valves, and (γ) the mass of blood in the ventricles.

The second cardiac sound has been traced to sudden tightening and subsequent vibration of the semilunar valve-flaps. The vibration of the columns of blood in the aorta and pulmonary artery is also partly answerable for the sound. If the root of the aorta and its valve be cut out and tied to the lower end of a vertical tube filled with blood, and the valve be then rendered slack by gently pushing it up from below, and be then suddenly rendered tense by removing the support from under it, a sound is produced. If next the length of the tube and column of blood be doubled, and the experiment repeated, the sound is lowered in pitch although the tightness of the valve-flap is increased. Hence the resonance of the tube and column of blood rather than that of the valve-membrane is the predominant factor in the sound (Talma). But analysis proves the sound to be compounded of a lower note due to the vibration of the column of blood and a higher note due to the vibration of the valve-membrane. The sudden tightening of the valve-flaps and the production of the second sound occur not at the closure of the semilunar valve-flaps, but quickly after.

Of the sounds emitted from the heart the weakest on the surface of the body is that of the right ventricle; the loudest that of the left ventricle. The aortic sound is usually not so loud as that of the pulmonary artery (Vierordt).

Recently attention has been called to the existence of a third heart-sound (Einthoven, A. G. Gibson). This sound is heard best at the apex. It is usually feeble and low, it follows the second sound, and is of much deeper pitch than it. It is best heard in the expiratory pause of respiration. It is very clearly registered by the string-galvanometer; from his observations with that instrument Einthoven concludes that this third heart-sound is probably due to vibration of the aortic valves. Its intensity is variable from individual to individual; its intensity also varies much from period to period in one and the same person. Dr. A. G. Gibson ascribes the sound to the inrush of blood at the end of the systole into the ventricle floating up the cusps and causing a transient closure of the auriculo-ventricular valves.

In certain circumstances a "mid-diastolic" sound can be heard due to the auricular systole. When there is a delay between the systoles of the auricle and ventricle a clear sharp sound may be heard shortly before the normal first sound. Thus, Dr. Herringham detected a sound synchronous with the first of two beats he felt at the apex. Tracings of the apex-beat and jugular pulse shew that the first sharp beat in the apex tracing (*a*) was synchronous with the wave *a* in the jugular due to the auricular systole (Fig. 3). This separation of the auricular systole from the ventricular, and the occurrence of a sound or a murmur at the time of the auricular systole, occur in mitral stenosis, and as the left auricle is then also hypertrophied, there may be some connexion between the hypertrophy of the auricle and the production of the sound. (*Vide* also pp. 351, 352.)

When the jugular valves are competent, and the right auricle hyper-

trophied, the sudden closure of these valves may produce a sound which can be heard above the clavicles preceding the normal first sound of the heart.

Mass-Movements of the Heart.—The diminution in volume undergone by the heart as its ventricles expel their content of blood is accompanied by a change in its form. If the diameters of the heart *in situ* be measured in the opened chest of a supine animal, it is found that during systole the side to side diameter diminishes much more than the front to back. That is, in systole the heart becomes more or less ellipsoid in cross-section. Probably in the unopened chest and in the erect position its cross-section in diastole as well as in systole is nearly circular. In systole the ventricles are somewhat shortened; but the apex shifts little; it is the base which moves, descending and coming forward towards the apex. This movement of the base is accompanied by a

FIG. 1.—Simultaneous tracings of the heart-movements (upper tracing) and of the radial pulse. The first part of the upper tracing was taken from the apex-beat in the fourth interspace immediately outside the nipple, whilst the latter part was taken in the same interspace near the left border of the sternum. In the first part the cardiogram shews a "systolic plateau" during the ventricular outflow (*E*), in the latter part the cardiogram is inverted, *i.e.* there is a depression during this period (*E*).

lengthening of the aorta and pulmonary arteries. This lengthening causes descent of the base of the contracting ventricles, and the descent compensates the shortening of the ventricles, and retains the apex in contact with the chest wall. The cardiac impulse is a protrusion of the chest wall over the surface of the ventricles at the moment just before the pulse-beat at the wrist. As the ventricles suddenly become hard their long axis becomes more horizontal to the vertical plane of the chest, and is tilted against the resistance of the chest wall. Around the spot where the soft parts of the chest are protruded by the impulse they are found slightly drawn in at the time of each systole (see Fig. 1). This "negative impulse" is caused by shrinkage of the heart in the air-tight chest as it empties itself, being followed inward by the lungs and to a small extent by the soft parts of the chest wall under the pressure of the atmosphere.

Graphic records of the cardiac impulse can be obtained by one or other of the different forms of cardiographs. Cardiograms, however, in spite of much attention bestowed on their elucidation, still remain unsatisfactory, on account of their variability and the difficulty of disentangling their component factors.

The Filling of the Heart.—The factors concerned in the filling of the heart are many. The acceleration imparted by the ventricles to the blood, both mediately through the elasticity of the arterial wall and immediately in the heart, gives the momentum of the inflowing blood. Then there is the excess of static pressure in the great veins over that in the diastolic auricle and ventricle. Contributory is the aspiration by the thorax during the act of inspiring, and also the slighter thoracic aspiration produced by the diminution in volume of the heart itself at each systole (John Hunter). The circulatory effect of the rhythmic decrease in intrathoracic pressure due to these two causes is illustrated by the pulsatile recession of the brain in the cranial fontanelles. Finally it is the general opinion that the ventricles and the auricles during their
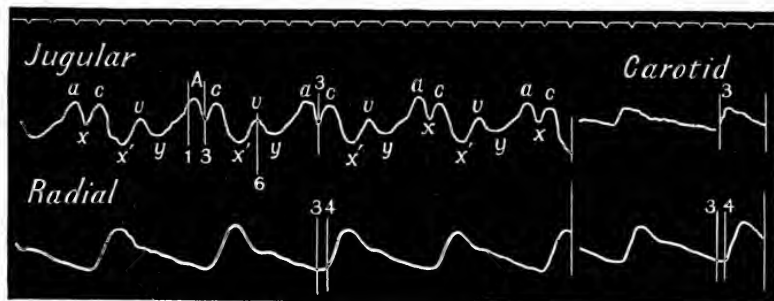
Fig. 2.—Simultaneous tracings of the jugular and radial pulses in the first part of the tracing, and of the carotid and radial in the latter part. The waves in the jugular tracing correspond to periods of increased pressure in the auricle ; a, wave due to the auricular systole ; c, wave due to the impact of the carotid and subclavian pulses ; v, wave due to stasis of the blood in auricle and veins during ventricular systole. The beginning of the fall of wave v corresponds with the time of the opening of the tricuspid valves (perpendicular line 6), and the succeeding fall y is due to the diastole of the ventricle. The fall x is due to the diastole of the auricle, and the fall x' to the increased expansion of the auricle on account of the pulling down of the auriculo-ventricular septum during ventricular systole.

relaxation period generate within themselves pressures lower than the pressure in the veins.

The Intra-auricular Pressure.—The curve of intra-auricular pressure during the cardiac cycle, corresponds with the variations in venous pressure which can be graphically recorded from the jugular veins, as in Fig. 2. When the examination is begun at the outset of the auricular systole, there is seen—(i.) a systolic rise of pressure, which is synchronous with the period of contraction of the auricle (wave a, Fig. 2) ; (ii.) a first diastolic fall of short duration corresponding with the relaxation of the auricle and with the earliest part of the systolic rise of intra-ventricular pressure (fall x, Fig. 2). It is noteworthy that the closure of the auriculo-ventricular valves does not cause even a transient elevation of pressure in the auricle. (iii.) The first diastolic rise of pressure is short, and occurs during the early continuance of the ventricular systole. It may be due to the bulging up of the partition between the auricle and ventricle under the high pressure in the latter. It is absent when by vagus inhibition

the ventricle is prevented from beating.   In tracings of the jugular pulse the wave *c* (Fig. 2) is sometimes said to be due to this first diastolic rise in auricular pressure, but there can be no question that the carotid and subclavian impact is the main factor of the wave *c* in such a tracing as Fig. 2, for the cup which receives the movement of the jugular also covers portions of the carotid and subclavian arteries, and the air in the cup is affected by their movements.   (iv.) A second diastolic fall occurs while the intra-ventricular pressure is still rising (fall x', Fig. 2).   It lasts longer than the former fall, and is more marked.   Its cause may be the factors increasing the size of the auricular cavity described on pages 4 and 5.   (v.) A second diastolic rise occurs as a steady increase of pressure, due to accumulation of blood in the auricle during the ventricular systole (wave *v*, Fig. 2), which continues until the beginning of the diastole of the ventricle.   (vi.) The third diastolic fall (fall *y*, Fig. 2), best marked when the heart is beating slowly, is probably due to a low pressure generated in the common cavity of auricle-ventricle by the suction of the relaxing ventricle.   The opening of the tricuspid valves is shewn in the jugular tracings by the beginning of the fall of the wave *v* (perpendicular line 6, Fig. 2).   In a particular case the values of the pressures were in the dog's auricle systolic rise 9, 5, -10, 5, 5 mm. Hg.

The Filling of the Ventricle.—As the systole of the ventricle ends and relaxation of its muscle occurs, a negative pressure is generated in the ventricle.   Moens supposed that in the latter part of systole the ventricle developed in itself a negative pressure, but his hypothesis is unsupported by subsequent physiological observations.   The negative pressure is at first considerable, but this degree of it lasts for a very short time only (Porter), and is over before the auriculo - ventricular valve-flaps can open ; it does not, therefore, help directly to fill the heart. There succeeds a longer period of much slighter negative pressure ; this assists, the auriculo-ventricular valves being open, to draw blood into the ventricle from the auricle, and into the latter from the veins.   Its importance for the filling of the heart is proportional to its duration.

The Intra-ventricular Pressure.—The rise of pressure in the ventricle which accompanies the systolic contraction of its muscle proceeds gradually though rapidly.   It closes the auriculo-ventricular valves almost at once, but for some $\frac{3}{100}$ of a second, though steadily increasing, it cannot burst open the semilunar valves.   This is the period of "getting up pressure," the "prosphygmic interval" as Sir C. Allbutt terms it.   The pressure reaches its maximum in about $\frac{8}{100}$ of a second, and for more than the latter half of this interval the semilunar valves have been opened.   The pressure continues to rise, therefore, after the opening of those valves has been effected ; nor does it recede far from the maximum until the relaxation of the muscle sets in, about $\frac{20}{100}$ of a second after the opening of the valves. The pressure in the ventricle then drops below the pressure in the aorta, and the semilunar valves close.   If the pressure in the arterial system is high, the pressure in the ventricle runs a course somewhat different from the above, for instead of reaching its maximum soon after the opening of

the semilunar valves it slowly increases throughout the systole, becoming maximal immediately prior to relaxation (Huerthle). In both cases, however, the curve of intra-ventricular pressure is a relatively flat-topped one, shewing a "systolic plateau." As Sir Clifford Allbutt wisely says, "It is the function of a healthy heart and arteries to promote the maximum of blood displacement with the minimum alteration of pressures." In the systolic plateau two minor undulations of pressure are seen ; the causation of these, which are synchronous with two seen in the aortic pressure-pulse, is not clear. On the setting in of relaxation of the ventricle the pressure, in $\frac{4}{100}$ of a second, falls from between 150 and 180 mm. Hg to below zero ; and then for $\frac{4}{100}$ to $\frac{8}{100}$ remains negative. The negative pressure generated varies much in amount, but may reach nearly 20 mm. of Hg. Some careful observers have recently failed to find distinctly negative pressures developed in the ventricle under approximately normal conditions. Experimentalists are at present divided in opinion as to the mode in which the ventricle after its contraction returns to its diastolic form ; one view is that the return is passive, the other (Luciani) that the return is dilatation on the part of the ventricular chamber caused by active elongation of its wall. Gradually the pressure rises to a little above zero, and remains a few millimetres above zero throughout the rest of the diastole, until the auricular systole occurs and drives it slightly up to about 10 mm. of Hg.

	Secs.	Secs.
Systole of ventricle before the opening of the semilunar valves, while pressure is still getting up . . .	·03	
Continued contraction of the ventricle and escape of blood into aorta	·27	
Total systole of the ventricle	—	·3
Diastole of both auricle and ventricle, neither contracting, passive interval	·4	
Systole of auricle (about or less than)	·1	
Diastole of ventricle, including relaxation and filling, up to the beginning of the ventricular systole . . .	—	·5
Total cardiac cycle		·8

It is important to note that with a frequent pulse the frequency is obtained without appreciable shortening of the cardiac systole, and almost entirely by reduction of the resting period of the heart, the diastole. Further, with a high arterial pressure the period of complete ventricular relaxation is somewhat shortened.

The Work of the Heart.—The heart is a machine which converts chemical energy into heat, electrical difference, and mechanical work. Only the last-named form of its output of energy need be considered here. During $\frac{1}{3}$ sec. of the ventricular systole the left ventricle exercises a pressure on its contents often amounting to close on 200 mm. Hg ; that is, a

pressure of 272 grams on each square centimetre of its internal surface ; 100 cubic centimetres is a low estimate of the output of blood.

Experimental observation shews that the heart is a machine which maintains in varying circumstances — so long as its nervous system and its own nutrition are not interfered with — a curiously constant action in two respects : namely, in the duration of the ventricular systole and in the quantity of the output of blood into the aorta. To keep these constant the heart has in varying circumstances to perform very various amounts of work. When the aortic pressure is high, it is found by direct measurement that not only is the maximal pressure produced in the ventricle at each systole much higher than when the aortic pressure is low, but also that the average pressure in the ventricle during systole is much higher than when the heart is beating against a low aortic pressure. The systolic pressure-plateau is much heightened. High arterial blood-pressure involves, therefore, a greater expenditure of energy by the heart at each systole. It is interesting to note that a rise of arterial pressure is in most cases followed by a reduction of the frequency of the heart's rhythm. This is in consequence of excitation of the vagus centre ; the stimulation being in part a reflex started from the wall of the heart itself, and in part a direct effect of the high pressure of the blood circulating in the brain. An important factor determining the work of the heart is the distension of the ventricular cavity in diastole. The pressure on a unit of surface of the cavity remaining the same, the total intra-ventricular pressure will vary approximately as the square of the radius of the cavity if the cavity be taken as approximately spherical. Thus Roy and Adami have pointed out that distension of the ventricle means not only increase of the tension of the muscular fibres, but also increase of the lateral pressure on their surface in proportion as the square of their increase in length. But, as they further pointed out, the content of the cavity increases as the cube, and the muscle-fibres in order to expel the same constant quantity of blood from the dilated as from the undilated ventricle need to shorten to a relatively less extent than was required of them before. The effect of diastolic distension is therefore, if the output from the ventricle at each systole remain the same, to leave a larger residuum of blood in the ventricle at the end of systole. Recent investigations (Roy and Adami, Huerthle) have shewn that to suppose that the ventricle empties itself completely at each systole is erroneous. Not only does it not do so, but the residual quantity of blood varies a good deal, and with it varies generally the amount of distension of the ventricle in diastole. The amount of distension of the ventricle, in other words, the degree of stretch in the muscle-fibres, at the moment when they enter into contraction, is an important determinant of the force of their contraction. All muscles respond by greater contraction when stretched than when unstretched. This increase in contraction is seen chiefly in increase of the work done, but the amount of actual shortening of the muscle is usually less when it is placed under considerable stretch than when it is not. The work done (lift × load) and

the heat given out are, however, greater. The ventricle when well loaded, or even excessively loaded, may from our general knowledge of the effect of tension on all muscular structures be expected to expend more energy and do more work at each contraction than when lightly loaded. But it does not necessarily follow that a largely distended ventricle is during diastole more loaded, that is, under higher tension than one only normally distended. The tonus of the heart-muscle is variable, and its tension will depend on the tonus. Moreover, the heart may be considered an after-loaded muscle, its load only coming into play during its contraction. The amount of blood expelled at each systole will be increased in an enlarged ventricle, and is found by experiment to be increased ; but at the same time the nervous system is likely to be excited to reduce the frequency of repetition of the heart's beat, and in that way to spare the expenditure of energy by the muscle-cells.

The diastolic size of the ventricle also influences the contraction of the ventricle in another way. The mechanical condition of the contraction of the ventricular muscle differs in one respect remarkably from the conditions obtaining in the skeletal muscles : in the skeletal muscle the contractions are in the execution of most movements approximately isotonic ; that is, while the length of the muscle alters, its tension remains approximately constant ; broadly taken, it is only in using the muscles for fixation that the contraction becomes isometric, that is, without change in length. The contraction of the heart during the time of getting up pressure is, on the contrary, practically isometric. The muscle-fibres can only alter their length in so far as the cavity of the ventricle can be altered in its shape, its volume remaining constant. But the larger the chamber of the ventricle the smaller the amount of shortening, which, as explained above (Roy and Adami), is necessary for reducing the size of the chamber by a given volume. The output of the heart remains fairly constant for each systole. The amount of systolic shortening of the cardiac fibres then is less when the diastolic ventricle is largely distended than when it is little distended. The contraction in the former case approximates nearer to the isometric condition than in the latter.

In many morbid conditions increased work is thrown upon the heart. In mitral and in aortic regurgitation the ventricle is not an " after-loaded " muscle to the extent it normally should be ; its load in those cases is applied in diastole owing to the excessive filling of the heart by back-flow. Similar increased diastolic volume of the heart may be brought about by compressing the abdomen and the veins therein (Roy and Adami). As stated above, a certain amount of diastolic loading is favourable to the heart's contraction. In aortic stenosis an extra load is imposed on the ventricle at each systole. The heart is more than normally after-loaded ; and here again high-tension of the muscle is, within limits, a favourable condition for output of energy by the heart. But tension beyond a certain degree, and applied for more than a short period, is harmful here as in the case of other muscles. The heart, as Roy and Cohnheim have so well insisted, offers remarkable examples of the reserve power characteristic of

the mechanisms of the animal body.   By artificially reducing the lumen
of the aorta even greatly, the aortic blood-pressure is but little lowered ;
it is maintained by the expenditure of perhaps a fourfold amount of work
by the ventricle, as has been proved by manometric measurements.   And
furthermore increased activity within limits in the cardiac muscle leads,
as in other muscles, to growth and further development of the muscle.
To a certain extent, therefore, the heart possesses not merely a great
temporary reserve power, but in virtue of its reaction of " hypertrophy "
a high degree of permanent reserve power.

On the other hand, heart failure means the exhaustion of the reserve
power, and the degree of recovery from heart failure depends on the
amount of reserve power the heart is capable of acquiring.   Whilst this
possession of reserve power is true of the heart as a whole, it also holds
good for the individual functions of the heart muscle-fibres, and any one of
these functions may shew an exhaustion of this reserve power while the
reserve power of other functions may be considerable.   Thus in Fig. 3
the long delay between the auricular and ventricular systoles and the
dropping out of the ventricular systole in Fig. 4 imply a great ex-
haustion of the function of conductivity, while the contractile force of the
heart shews no sign of exhaustion.

The Manner of the Heart's Contraction.—In order to appreciate the
nature of the normal contraction of the heart and the departures from
the normal (irregular action of the heart) it will be well to glance briefly
at the changes undergone by the heart in the course of development.
Included in this description is a hypothesis as to the nature of irregular
action, which is not put forward as finally proved, but as offering an
intelligible account of the different forms of irregularity ; irregularity
would otherwise have to be looked upon as a series of bewildering and
disconnected data.

The heart first appears as a tube in which, later, pouches develop,
these becoming ultimately the auricles and ventricles.   This primitive
tube contracts in a very definite manner, the wave of contraction
beginning at the venous end, which is the most excitable part.   Any
part of the remainder of the tube can start the contraction if rendered
more excitable (as by heat) than the venous end.   This power of starting
the contraction, though present in the whole length of the tube, becomes
less excitable in parts farther away from the venous end.   This is seen
when the tube is cut between the auricle and ventricle in the frog's
heart ; the ventricle will then beat of its own accord independently of
the sinus and auricle, but at a slower rate.   In the course of develop-
ment this primitive cardiac tube becomes modified, so that it loses its
tubular form, and in the mammalian heart is found scattered about in
various places.   Dr. Stanley Kent and W. His, jun., first described a
bundle connecting the auricle and ventricle, and Aschoff and Tawara
more recently gave a minute account of this " auriculo-ventricular
bundle."   This bundle arises in the right auricle from a node near the
coronary sinus, passes across the auriculo-ventricular septum and divides

into two branches, one of which goes to the left ventricle and the other to the right, these branches finally breaking up into numerous fine threads which pass to the muscle-fibres. Still more recently Drs. Keith and Flack have found a node of tissue at the mouth of the superior. vena cava, and Dr. A. G. Gibson has described tissues of a very primitive kind distributed over the auricles.

Branches of the vagus and sympathetic nerves are distributed to the node described by Drs. Keith and Flack, and it is well known that stimulation of these nerves has a powerful influence in modifying the rate and rhythm of the heart's contraction. In every portion of the heart numerous nerve-fibrils are present. Normally, the heart's contraction in mammals is assumed to arise in some portion of the primitive cardiac tissue at the mouths of the great veins, as in the sino-auricular node of Keith and Flack. From this point the contraction spreads to the auricles and by way of the auriculo-ventricular bundle to the ventricles. This passage of the stimulus to the ventricle is in the
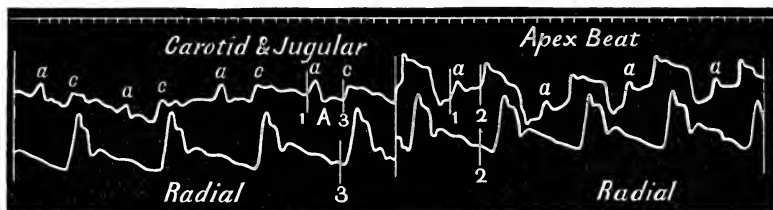
Fig. 3.—In the first part of the tracing the movements of the carotid and jugular above the clavicle are recorded at the same time as the radial, and in the latter part the apex and radial. The interval between the auricular wave, *a*, and the carotid pulse, *c* (the *a-c* interval), is nearly three-fifths of a second (compare with the *a-c* interval in Fig. 2), and indicates a great delay in the transmission of the stimulus from auricle to ventricle. The same delay is seen in the apex tracing.

normal human heart so rapid that the ventricular contraction begins at the completion of the auricular systole. In certain circumstances, as in disease of the auriculo-ventricular bundle, there may be a delay in the stimulus passing from auricle to ventricle (see Fig. 3). By gradually compressing the connexion between auricle and ventricle in the heart of the tortoise Dr. W. H. Gaskell has shewn that a delay may be produced between the auricular and ventricular contractions. By increasing the compression the stimulus from the auricle may be blocked (*i.e.* prevented from passing to the ventricle), so that some of the auricular contractions are not followed by a ventricular contraction. With increasing compression the dropping out of the ventricular systoles becomes still more frequent, until the pressure may prevent all communication between auricle and ventricle. When this occurs, the auricle and ventricle may contract at independent rates, the ventricular rate being much slower than the auricular. Practically identical results are obtained experimentally in the hearts of mammals (Hering, Erlanger). In the human heart the same results have been observed. Thus in Fig. 3 there is a great delay in the transmission of the stimulus from auricle to

ventricle.   In Fig. 4 there are two auricular waves (*a*) to one carotid or
radial pulse, whilst in Fig. 5 there is a discordance between the auricular
and ventricular rhythm, the auricular rate being 75 and the ventricular
31.   The relationship of the auricular wave (*a*) to the carotid (*c*) is seen
to be always varying.

Abnormal Inception of the Cardiac Rhythm.—From the description of
Fig. 5 it will be seen that the heart's contraction may start from two
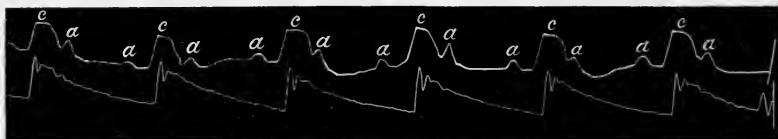
FIG. 4.—In the tracing from the neck (upper tracing) there are two auricular waves (*a*) to one carotid
(*c*).   The radial pulse (lower tracing) is synchronous with the carotid wave.   Pulse in radial 35 per
minute ; auricular wave in jugular 70 per minute.

places, one the normal, as shewn by the contraction of the auricle, and
one somewhere else, as shewn by the independent contraction of the
ventricle.

There are a number of places where an independent contraction may
arise.   It was pointed out that if some portion of the primitive cardiac
tube were rendered more excitable than the venous end, the contraction
would proceed from that part.   In 1892 it was demonstrated that in the
human heart, while the auricle beats regularly, a ventricular contraction
could occur before, and independently of, the auricular contraction
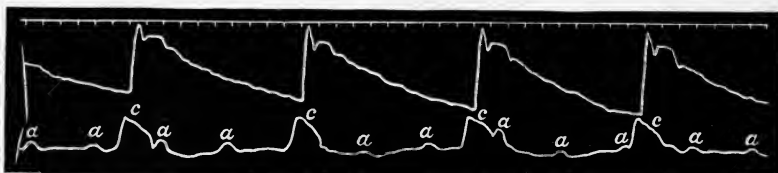
FIG. 5.—Shews a complete dissociation of the auricular waves from the carotid, the auricular waves
occurring at the rate of 75 per minute, while the carotid radial pulse is at 31.

(Mackenzie).   Thus in Fig. 6 the waves due to the auricular systole
(*a* and *a'*) are perfectly regular in their appearance, whilst the wave *c'*
appears before *a'*, that is to say, the wave *c'* is due to a premature
contraction of the ventricle, and arises independently of the auricular
contraction, which pursues unaltered its normal rhythm.   This observa-
tion has been amply confirmed by subsequent experimental and clinical
observation.   This form of premature contraction is now recognised as
the "extra-systole."   Other forms of extra-systole may appear, for
example, when the auricle contracts prematurely and independently of
the normal rhythm, and when auricle and ventricle both contract prema-
turely and simultaneously.   To explain this, it is suggested that some
portion of the primitive cardiac tube has become more excitable than the

venous end, so that a stimulus arising in the ventricular portion causes the ventricular extra-systole, that a stimulus arising in the auricular portion causes the auricular extra-systole, and a stimulus arising in the auriculo-ventricular node causes the simultaneous contraction of auricle
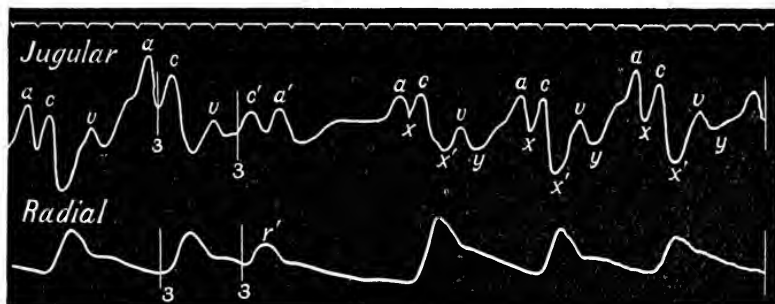
Fig. 6.—Simultaneous tracings of the jugular and radial pulses, shewing a premature contraction of the ventricle (c′, r′), while the auricular rhythm is undisturbed (a and a′). The wave c′ in the jugular tracing corresponds to the premature beat r′ in the radial tracing, and is seen to precede the auricular wave a′,—the latter appearing at the normal interval. The premature beat (c′, r′) is due to a "ventricular extra-systole."

and ventricle (nodal extra-systole). In certain individuals all these forms of extra-systole may be found to occur. Observation of patients with these extra-systoles for a number of years will shew that in a few these extra-systoles may at times become so frequent that a number of
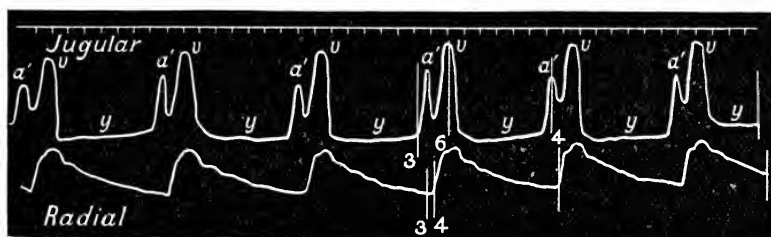
Fig. 7.—Simultaneous tracings of the jugular and radial pulses, shewing the ventricular form of the jugular pulse. There is an absence of the auricular wave (a) before the time of the carotid pulse (perpendicular line 3). The wave a′ occurs during the period that the ventricle contracts, and is supposed to be due to an auricular systole occurring at the same time as the ventricular systole (nodal rhythm).

them may appear in succession, and in rare instances the heart's contraction may continuously start in this abnormal fashion.

In advanced mitral disease the cicatricial lesion affecting the valves may extend to the muscle, and may encroach on the auriculo-ventricular bundle, irritating it, and start the contraction at the irritated place. This change in the inception of the rhythm of the heart is made evident by the disappearance of all evidences of the auricular systole at the normal period of the heart's contraction. Thus a presystolic murmur

disappears, and the auricular wave disappears from the venous pulse, and the venous pulse becomes of the ventricular form (compare Fig. 7 with Fig. 2). As a rule the heart's rhythm becomes irregular (Fig. 8). It is assumed that in instances such as shewn in Figs. 7 and 8 the heart's contraction no longer begins at the normal area, but at some point which starts off auricle and ventricle together, and it is suggested that the node of tissue at the beginning of the auriculo-ventricular bundle is the place
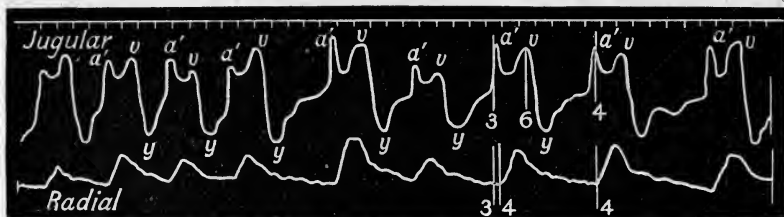
Fig. 8.—Shews the same features as Fig. 7, but the rate is more rapid, and the rhythm is of that disorderly kind characteristic of the inception of the heart contraction at some place other than the normal starting-place (nodal rhythm).

in question; hence this form of cardiac rhythm is provisionally spoken of as the "nodal rhythm." This nodal rhythm has recently been produced experimentally (Cushny); and by means of the electro-cardiogram it has been shewn that in these cases the auricular systole no longer precedes the ventricular (Hering, Lewis). The post-mortem examination by Dr. Keith of a series of cases which shewed this abnormal rhythm during life has revealed changes in the auriculo-ventricular bundle (increase of fibrous tissue, nucleated cells).

II. The Peripheral Circulation

The Peripheral Resistance to the Heart.—Our knowledge of the conditions of resistance offered in the circulation of the blood to the action of the heart can be satisfactorily dealt with from a physical point of view only by use of laws which connect together certain measurable facts concerning the blood-vessels and the blood. We require to know the amount of motive force which, as shewn above, may be taken to be the aortic blood-pressure, the velocity of flow of blood, and the resistance which is overcome by the streaming blood. The last-named—the resistance—is composed of *two factors, the one resident in the dimensions of the channel, the other in the properties of the fluid—the blood.*

It has just been said that of the factor resident in the properties of the vascular channel the *dimensions* only are of account. The resistance which the channel offers to the flow of fluid along it diminishes with the shortness of the tube and with the increase of the bore of it. The nature of the material composing the tube is practically without influence on the flow. A tube of given dimensions offers the same resistance to a

stream of water within it whether it be of metal, of glass, or of any other material. Every moving fluid streams along in a channel lined by its own fluid particles, and the layer of fluid immediately next the wall of the containing channel is practically at rest.

The factor depending on the properties of the blood itself is measurable in terms of standard fluids, and is due to what is called *viscosity,* its internal friction. Fluid flowing along a channel may, of course, be considered as though composed of a number of concentric fluid cylinders ranged round an axial thread of quickest stream, and contained within an outermost sheet where velocity is reduced to zero. In their streaming motion, therefore, the particles of the fluid move over and among their fellows, and this relative movement is opposed in the fluid by its specific coherence or viscosity. As to its degree, this internal resistance is largely influenced in one and the same fluid by temperature. Dr. Graham Brown has proved experimentally that the blood flows with considerably less resistance along tubes when warmed to fever heat than it does at normal body-temperature. The internal friction of distilled water is decreased 250 per cent by raising its temperature to blood-heat as compared with its internal friction at $0.5°$ centigrade.

But the main portion of the work of the heart is expended immediately, not on moving the blood through the vessels, but in *stretching the arterial wall.* The elasticity of this wall is therefore of importance in physical action of the heart. Wertheim and Roy examined the elasticity of the aorta by hanging weights on a strip of uniform cross-sectional area taken from it. Roy, by an ingenious apparatus, obtained continuous graphic records while the load was uniformly increased in weight, and thus obtained curves in which the weights are represented by the abscissae, the elongations constituting the ordinates. Both he and Wertheim agree that the curve obtained (if the strip be fresh and from a healthy vessel) is an hyperbola. Roy and Zwaardemaker have further examined experimentally the increment of cubic content of the vessel obtained under heightened pressure. Starting from a pressure about equal to that normal in the blood-vessel under examination, they found that, under successive equal increments of pressure, the increase obtained in capacity is greatest at first ; and as the pressure is gradually heightened, the increase in capacity obtained becomes less and less. They found also that as the pressure starting from normal (for example 120 mm. of mercury for the carotid of the cat) is reduced by successive equal decrements, the diminution of capacity follows more rapidly at first than later. These observers, therefore, find the extensibility at its greatest at a range of pressures which are frequent and usual in the vessel under examination. Also that above those pressures the curve of extensibility is hyperbolic. It is clear, therefore, that with a high arterial blood-pressure a certain further absolute increase of pressure will distend the vessel less than will the same absolute increase of pressure under a lower arterial pressure. Also that the injection by the heart into the aorta of a certain absolute quantity of blood will raise the

aortic tension relatively more when the pressure is already high than when it is about the mean or is low.   The walls of the smaller vessels have been proved to be more easily distensible than those of the large, so that any increase in the amount of blood in the arterial system will locally distribute that blood in the smaller or larger vessels relatively differently under low than under high arterial pressures.

It is noteworthy that the rupturing strains of the arteries is proved by experiment to be about twenty times greater than any strain the body can put upon them ; this is true, of course, of healthy vessels.

Influence of the Force of Gravity on the Heart.—It might at first sight appear that since the blood in circulation lies practically in a vertical circuit, the effect of gravity as regards the work to be done by the heart in maintaining the movement of the blood would not be affected by gravity, the weights of the blood in the up-stream and down-stream columns balancing one another.   But such a view leaves out of consideration the effect of the static pressure of the fluid columns in the vessels in stretching the walls of the vessels.   Dr. L. Hill has investigated the results of this for the heart and the circulation generally.   In respect of the former he points out that the force of gravity must be regarded as a cardinal factor in circulatory problems.   The splanchnic vasomotor system is entrusted with the important duty of compensating the hydrostatic effects of gravity brought about by changes in the posture of the body.   This action of the splanchnic vasomotor system is far more developed in upright animals, such as the monkey, than in rabbits and dogs ; and therefore is probably very complete in man.   He proves that when the power of compensation is damaged by paralysis of the splanchnic vaso-constrictors, for instance by shock, in asphyxia, or by chloroform, the blood drains into the abdominal veins, the tonus of the splanchnic vessels not being sufficient to resist the hydrostatic pressure if the upright position be assumed ; in consequence the heart empties and the cerebral circulation ceases.   In the horizontal and in the " feet-up " position syncope is avoided or recovered from, the force of gravity acting in the same sense as the heart.   To bandage the abdomen firmly has the same restorative effect.   Chloroform by destroying the compensation for gravity in the circulation can kill an animal if the posture be one in which the abdomen is on a lower level than the heart.

Estimation of Arterial Pressure.—As has already been shewn the full force of the ventricular contraction is not spent on the blood-current merely during the period of its systole, for in throwing the blood into the arterial system sufficient force is exerted to distend to a slight degree the walls of the larger arteries.   So soon as the ventricular systole is over the elastic coats of the arteries compress the column of blood within them.   The smaller arteries and arterioles offer such a resistance to the escape of blood, that the pressure within the larger arteries is kept at a certain height, in order to maintain a continuous flow through the capillaries.   This pressure within the arteries is

spoken of as blood-pressure or arterial pressure, and the factors concerned in its production and maintenance are therefore the contraction of the left ventricle, the peripheral resistance, the elastic recoil of the arteries, and the viscosity of the blood. (For discussion of the use of word "tension" *vide* p. 496.)

The estimation of the condition of the arterial pressure is of great importance in the examination of the circulatory system. Many methods have been employed for this purpose. The trained finger will ever remain a useful instrument for this purpose. It is necessary that the finger should be educated particularly by comparing the impression made on the mind through the finger, with the result of instrumental observation. In palpating the pulse the condition of the arterial wall is observed ; the resistance offered to the compressing finger, the size of each individual pulse-beat, the manner in which it strikes against the finger, the duration of the pulse-wave, and the degree of resistance that remains after the pulse-wave has passed are all features that the trained finger can recognise.

The results obtained by digital examination cannot be expressed in terms of mathematical formulae, nor in language that conveys the exact impression to other minds ; nevertheless to the experienced observer the information is invaluable, and cannot be acquired by any other procedure.

The tracings obtained by means of the sphygmograph also give certain information in regard to the character of the arterial pressure. The character of the wave due to the systole of the ventricle sometimes presents very instructive features. Normally it is well maintained during systole, and the fall during diastole is gradual with a dicrotic wave more or less well marked. In low pressure this systolic wave is not well maintained and the tracing falls rapidly during the diastole. With increased pressure the systolic wave is well marked and distinct, and the fall during diastole slight in amount and the dicrotic wave is not of large size. There are, however, infinite gradations depending on a number of factors, such as the rate of the pulse, the temperature, the condition of the aortic valves and arterial walls, and instrumental defects, so that great care must be taken in discriminating the features in any given case. As a result of these numerous and varied difficulties the use of the sphygmograph for this purpose has, to a great extent, passed out of use. This is to be regretted, because the sphygmograph can reveal variations in pressure of very great significance. Thus Fig. 9 shews a form of pulse-beat in which every alternate beat varies in size while the rhythm is accurately maintained. This alternating variation (*pulsus alternans*) is of great prognostic significance, and it would have been entirely overlooked were it not for the sphygmogram.

However highly trained the finger may become, and however skilfully the sphygmograph may be applied, no trustworthy conception of the pressure within the artery can be obtained. The necessity for some method capable of giving trustworthy information is urgently

felt by clinicians, and numerous attempts have in recent years been made to supply a satisfactory method. At the present time the method most frequently employed is that in which a broad band containing an air-bag is fixed round the upper arm, the pressure in the bag being raised by pumping air into it. The bag is connected with a mercurial manometer so as to indicate the amount of pressure within the bag. A finger is kept on the patient's radial pulse on that side which is being compressed. The pressure within the bag is raised until the radial pulse disappears, and the pressure on the manometer at the time of the disappearance of the radial pulse is noted. Or, the pressure is raised rapidly until the pulse disappears, then the air in the bag is allowed to escape and the pressure noted at which the radial pulse-beats are first detected. By this means the amount of pressure necessary to cause the disappearance of the pulse (obliteration pressure)
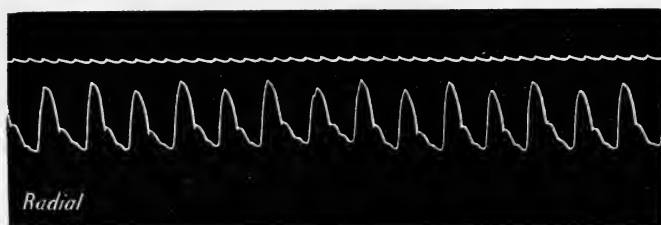
Fig. 9.—Sphygmogram of the radial pulse shewing pulse-beats alternating in size due to variations in pressure recognisable only by means of the sphygmograph. This form of pulse—the *pulsus alternans*—has a very grave significance.

is obtained. It is this obliteration pressure which is meant by "arterial pressure" in the following description. Some speak of the obliteration pressure as "systolic pressure" as it is supposed to indicate the amount of pressure in the pulse due to the systole of the ventricle. There are, however, several factors concerned which make it as yet doubtful whether the pressure that causes the disappearance of the pulse as estimated by clinicians is really equivalent to the systolic pressure. The variable results obtained by different individuals and different instruments indicate a personal or instrumental element in the detection of the time of disappearance of the pulse. It is even doubtful if the pulse has actually stopped when the finger fails to detect it. There is still some uncertainty whether the arterial wall itself may not oppose some resistance to compression, either from changes in the wall or to its degree of contraction.

In the manometer connected with the air-bag oscillation of the mercury can be detected, beginning at first small in size and gradually increasing with a rise in pressure, then diminishing until at the obliteration pressure they sometimes disappear. Attempts have been made to find in these oscillations, periods which would correspond to the mean or diastolic pressure, but no trustworthy evidence has been produced which would justify such an interpretation. There are a number of

instruments used for measuring the blood-pressure. Among the more useful and reliable are Martin's modification of Riva-Rocci's instrument, Hill and Barnard's sphygmometer, Oliver's compressed-air haemomano-meter. Graphic records of the arterial pressure can be obtained by Erlanger's and by Dr. G. A. Gibson's sphygmomanometer; the latter gives an excellent picture of the movement of the mercury column as the air escapes from the armlet.

The most useful method so far has been in observing the pressure in the air-bag which obliterates the radial pulse. Practically all observers agree that this method gives a result which so nearly represents the maximum blood-pressure as to be of definite clinical value. But inasmuch as the results are somewhat conflicting it needs much further careful work, particularly in the observation of individual cases for a long period of years, and in the consideration of the factors present in each individual case, before the true value of blood-pressure observations can be estimated. At present it is most injudicious to base a diagnosis or treatment on the blood-pressure observation alone. It will be found that many individuals have what in our present state of knowledge might be considered an abnormal pressure, but suffer in no way from this, and their subsequent histories demonstrate that there was no serious condition underlying it. When an abnormal pressure is supposed to be obtained, the condition of the patient as a whole should be carefully scrutinised. In the search, some definite cause for the abnormal pressure may be ascertained, and in the absence of some definite cause (as kidney disease), judgment should be suspended as to the import of the abnormal pressure.

As far as our present knowledge goes, the following are what may be regarded as normal blood-pressures :—In children under ten a blood-pressure of 100 Hg may be considered normal. In the young and in most adults up to forty years of age, the pressure varies from 115 to 125 mm. Hg. Above forty a slight rise occurs, which gradually increases with advancing years, so that a pressure of 140 to 150 in people over fifty years of age is so frequent as to be considered within normal limits. A great many people from sixty to seventy years and over may have a pressure of 160 to 180 and do not have any symptoms apart from that limitation of the field of cardiac response which is a natural concomitant of advancing years. An important point brought out by the measure-ment of arterial pressure is, that what is commonly spoken of as heart failure, is not necessarily accompanied by a fall of arterial pressure. In many cases of heart failure with great breathlessness on exertion, it will be found that the blood-pressure has not fallen, and when recovery takes place the blood-pressure has not risen (H. J. Starling). What has happened is that the heart can keep up the necessary pressure when the body is at rest, but that it cannot supply the additional pressure which is called for on exertion—in other words, it is the reserve force of the heart that is exhausted. When the heart failure is also accompanied by dilatation of the heart, the blood-pressure, as a

rule, will be found to fall during the heart failure and to rise during the recovery.

Increased pressure is found in many diseases, most notably in affections of the kidney. It is certain that from obscure causes, individuals are liable at times to periods of increased pressure (hyperpiesis of Clifford Allbutt), and this is often associated with some bodily or mental discomfort. With a return of the pressure to normal the discomfort disappears. Since many transient influences, such as bodily exertion, mental excitement, or smoking, may cause a temporary rise in pressure, care must be exercised in the determination of the cause of the increased pressure in any given case.

It has been remarked that in advancing years there is frequently an increase in the arterial pressure. As it is always accompanied by a diminution of the field of cardiac response, there is manifestly a diminution of the reserve power of the heart. Accompanying these changes the arterial walls become less resilient and the capillary field much diminished. This latter condition is shewn by the attenuation of the skin and the absence of capillary oozing from freshly-made incisions in the elderly. These two factors, the arterial and capillary changes, probably impede the flow and hence the heart responds by more vigorous contractions, encroaching in consequence on its reserve power. It is held, on the other hand, that these two factors do not affect the arterial pressure, but that the conditions producing temporary hyperpiesis become constant, and that the pressure is raised permanently (Allbutt). In consequence of this rise in pressure degenerative changes take place in the walls of the arteries. In certain cases hypertrophy of the muscular walls of the smaller arteries have been demonstrated (Savill, W. Russell), and from this it is inferred that there has been some material circulating in the blood, which stimulates directly or reflexly the muscular coats of the smaller arteries.

The pressure may be lower than normal, and certain definite symptoms, as faintness and giddiness, may be associated with it. The most characteristic cases are those in which the blood accumulates in the large abdominal veins. By compressing the abdomen the arterial pressure may be raised (Oliver), and attacks of syncope due to lowered pressure may be obviated by wearing a tight abdominal belt (Langwill).

The viscosity of the blood may be measured by observing the rate at which the blood flows down a capillary tube, as by the viscosimeter (Denning and Watson, M'Caskey, Hess, Bachmann). It can scarcely be said, however, that the estimation of the viscosity of the blood has been of much clinical use so far.

Electro-cardiograms.—Dr. A. D. Waller, using Lipmann's capillary electrometer, was the first to demonstrate the possibility of obtaining graphic records, from the human body, of the changes in electrical potential resulting from cardiac systole. Einthoven has recently introduced a new and delicate " string galvanometer," with which valuable curves can be obtained without difficulty. By means of his instrument the variation

in potential in the limbs following auricular and ventricular systole can be identified, and the method allows of an exact analysis of irregularity in the sequence of contraction of the various chambers of the heart.

C. S. Sherrington, 1898.
James Mackenzie, 1909.

REFERENCES

The Cardiac Valves: 1. Ceradini. *Der Mechanismus der halbmondförmigen Klappen*, Leipzig, 1872.—2. Hesse. *Arch. f. Anat. u. Physiol.*, 1880, 344.—3. Hunter, J. *Works*, edit. by Palmer, 1835, iii.—4. Keith, A. "The Evolution and Action of certain Muscular Structures of the Heart," *Lancet*, 1904, i. 556.—5. King, W. "The Safety-Valve Function in the Right Ventricle of the Human Heart," *Guy's Hosp. Rep.*, 1837, ii. 132.—6. Krehl. *Arch. f. Anat. u. Physiol.*, Physiol. Abt., 1889, 288.—7. Luchsinger. *Arch. f. d. ges. Physiol.*, 1884, xxxiv. 291.—8. Morus. *Arch. f. d. ges. Physiol.*, 1879, xx. 531.—9. Roy and Adami. *Phil. Trans.*, London, 1893.—10. Sächs. *Gesellsch. d. Wiss.*, 1891, 358.—11. Sandborg und Worm-Müller. *Arch. f. d. ges. Physiol.*, 1880, xxii. 412.—12. Sée, Marc. *Recherches sur l anatomie et la physiologie du cœur*, Paris, 1875. **The Cardiac Sounds:** 13. Einthoven, W. *Arch. f. d. ges. Physiol.*, 1907, cxvii. 117; *Ibid.*, 1907, cxx. 31.—14. Gibson, A. G. *Lancet*, 1907, ii. 1380.—15. Haycraft. *Journ. Physiol.*, 1890, xiii. 486. —16. Herroun and Yeo. *Journ. Physiol.*, 1885, vi. 290.—17. Huerthle. *Deutsche med. Wchnschr.*, 1894, xx. 267.—18. Kasem-Beck. *Arch. f. d. ges. Physiol.*, 1890, xlvii. 56.—19. Ludwig und Dogiel. *Ber. d. Sächs. Gesells. d. Wissens.*, 1868, 96.—20. Talma. *Arch. f. d. ges. Physiol.*, 1880, xxiii. 275.—21. Vierordt. *Die Messung der Intensität der Herztone*, Tübingen, 1885. **Mass Movements of the Heart:** 22. Bramwell (Byrom) and Murray. *Brit. Med. Journ.*, 1888, i. 10.—23. Colin. *Physiol. comp.*, Paris, 1888, 420.—24. Edgren. *Skand. Arch. f. Physiol.*, 1889.—25. Filehne und Penzoldt. *Centralbl. f. d. med. Wiss.*, 1879, 482.—26. Fredericq. *Travaux du labor.*, 1888.—27. *Idem. Centralbl. für Physiol.*, 1891, 587.—28. Frey, v. *Die Untersuchung des Pulses.*—29. Frey, v., und Krehl. *Arch. f. Anat. u. Phys.*, 1890, 31.— 30. Gad und Cowl. *Centralbl. f. Physiol.*, 1888, ii. 264.—31. Guttmann. *Arch. f. path. Anat.*, 1879, lxxvi. 534.—32. Huerthle. *Arch. f. d. ges. Physiol.*, 1891, xlix. 92.— 33. Knoll. *Sitzungsber. d. Wien. Akad. d. Wiss.*, 1890.—34. Magini. *Arch. ital. de biol.*, 1887, viii. 127.—35. Martius. *Ztschr. f. klin. Med.*, 1889, xv. 536.—36. Rolleston. *Journ. Physiol.*, 1887, viii.—37. Roy and Adami. *Practitioner*, 1890, 82.—38. *Idem. Phil. Trans.*, 1892.—39. Scheiber, D. *Arch. f. klin. Med.*, 1891, xlvii. 368.—40. Stefani. *Memoria da Ferrara*, 1891. **The Filling of the Heart:** 41. Ebstein. *Ergebnisse der Physiologie*, 1905, iii. pt. 2. p. 123.—42. Haycraft and Edie. *Journ. Physiol.*, 1891, xii. 426.—43. Luciani, L. *Fisiologia del l' uomo*, Milano, 1904, i.—44. Martin, Newell, and Donaldson. *Studies from the Physiol. Lab.*, Baltimore, 1887.—45. Mink. *Centralbl. f. Physiol.*, 1890, iv. 569.—46. Porter. *Journ. Physiol.*, 1892, xiii. 513.—47. Roy and Adami. *Brit. Med. Journ.*, 1888, ii. 1321.—48. Stefani-Ferrara. *Cardiovolume, etc.*, 1891. **The Work of the Heart:** 49. Roy and Adami. *Phil. Trans.*, London, 1893.—50. Stolnikow und Ludwig. *Arch. f. Anat. u. Physiol.*, Physiol. Abth., 1886, 1.—51. Tigerstedt. *Skand. Arch. f. Physiol.*, 1891, iii. 145.—52. *Idem. Die Physiol. des Kreislaufes*, Leipzig, 1893, 146.—53. Zuntz. *Deutsch. med. Wchnschr.*, 1892, 109. **The Manner of the Heart's Contraction:** 54. Erlanger. "Physiology of Heart-block in Mammals, etc.," *Journ. Exper. Med.*, N.Y., 1906, viii. 41.—55. Gaskell. Art. "The Contraction of Cardiac Muscle," Schäfer's *Textbook of Physiology*, ii. 169.—56. Gibson, A. G. "On the Primitive Muscle-Tissue of the Human Heart," *Brit. Med. Journ.*, 1909, i. 149. —57. Hering. "Nachweis dass das His'sche Uebergangsbündel Vorhof und Kammer des Säugethierherzens functionell verbindet," *Arch. f. ges. Physiol.* cviii.— 58. His, Jun. "Dritter intern. physiol. Congress in Bern," *Centralbl. f. Physiol.*, 1895, ix. 469.—59. Keith and Flack. "The Form and Nature of the Muscular Connexions between the Primary Divisions of the Vertebrate Heart," *Journ. Anat. and Physiol.*, London, 1907, xli. 172.—60. Kent. "Researches on the Structure and Functions of the Mammalian Heart," *Journ. Physiol.*, Cambridge, 1893, xiv. 233.— 61. Mackenzie, J. "The Extra-Systole," *Quart. Journ. Med.*, Oxford, 1908, i. 131, 481.—62. *Idem. Diseases of the Heart*, Oxford Univ. Press, 1908.—63.

TAWARA. *Das Reizleitungssystem des Säugethierherzens*, Jena, 1906. **Peripheral Resistance to the Heart's Action** : 64. BOIS-REYMOND, R. DU, BRODIE, T. G., und MÜLLER, F. *Arch. f. Physiol.*, Berlin, 1907, Suppl. Bd. 37.—65. BROWN, GRAHAM. *Edinb. Hosp. Rep.*, 1893, i. — 66. HUERTHLE. *Deutsche med. Wchnschr.*, Aug. 1897.—67. NICOLLS. *Journ. Physiol.* xx. 407.—68. ROY. *Journ. Physiol.*, 1881, 1888. —69. WERTHEIM. *Ann. de chim. et phys.*, 1847.—70. ZWAARDEMAKER. *Nederl. Tijdsch. v. Geneesk.*, 1888. **Influence of Gravity on the Heart's Action** : 71. BLUM-BERG, HERMANN, und WAGNER. *Arch. f. d. ges. Physiol.*, 1885, xxxvii. 467, and 1886, xxxix. 371.—72. HILL, LEONARD. *The Physiology and Pathology of the Cerebral Circulation*, London, 1896. — 73. OLIVER, G. *Pulse-Gauging*, London, 1895. — 74. ROY and ADAMI. *Brit. Med. Journ.*, 1888, ii. 1321. **Arterial Pressure** : 75. ALLBUTT, Sir CLIFFORD. "Rise of Blood-pressure in Later Life," *Med.-Chir. Trans.*, London, 1903, lxxxvi. 323.—76. *Idem.* "Clinical Remarks on Arteriosclerosis," *Brit. Med. Journ.*, 1906, ii. 1004.—77. DAWSON, P. M. "The Systolic Output and Work of the Heart and their relation to the Blood-pressure in Man," *Brit. Med. Journ.*, 1906, ii. 996.—78. ERLANGER. "A New Instrument for Determining the Minimum Blood-pressure in Man," *Johns Hopkins Hosp. Rep.*, 1904, xii. 53. — 79. DENNING and WATSON. "A Simple Form of Clinical Viscosimeter," *Lancet*, 1906, ii. 89. — 80. GIBSON, G. A. "A Clinical Sphygmomanometer yielding absolute Records of the Arterial Pressure," *Quart. Journ. Med.*, Oxford, 1908, i. 103.—81. JANEWAY. "The Clinical Study of Blood-pressure," 1904.—82. MACKENZIE, J. "The Nature of some forms of Heart Failure in consequence of long-continued high Arterial Blood-pressure," *Brit. Med. Journ.*, 1906, ii. 1907,—83. M'CASKEY. "The Viscosity of the Blood. Its Value in Clinical Medicine," *Journ. Am. Med. Assoc.*, Chicago, 1908, li. 1553.— 83A. MARTIN, C. J. "Determination of Arterial Blood-pressure in Clinical Practice," *Brit. Med. Journ.*, 1905, i. 865.—84. MUMMERY, P. L. "Comparison of Blood-pressure Readings obtained simultaneously with a Manometer and with a Sphygmomanometer," *Journ. Physiol.*, 1905, xxxii. 23.—85. OLIVER, G. "Studies in Blood-pressure," 2nd ed., London, 1908.—86. RUSSELL, W. "Arterial Hypertonus, Sclerosis, and Blood-pressure," Edin., 1907.—87. SAVILL. "On Arterial Sclerosis especially in regard to Arterial Hypermyotrophy," *Trans. Path. Soc.*, London, 1904, lv. 375.—88. STARLING, H. J. "Observations on the Arterial Blood-pressure in Heart-Disease," *Lancet*, London, 1906, ii. 846. **Electro-cardiograms** : 89. EINTHOVEN, W. "Le télécardiogramme," *Arch. internat. d. physiol.*, 1906-7, iv. 132 ; "Weiteres über das Elektrokardiogramm," *Arch. f. d. gesammte Physiol.* (Pflüger), 1908, cxxii. 517.—90. WALLER, A. D. "On the Electromotive Changes connected with the Beat of the Mammalian Heart, and of the Human Heart in Particular," *Phil. Trans. Roy. Soc.*, 1889, clxxx. 169.

J. M.

DISEASES OF THE PERICARDIUM

By FREDERICK T. ROBERTS, M.D., F.R.C.P.

THE NORMAL PERICARDIUM.—Before proceeding to discuss the morbid changes affecting the pericardium, it is desirable to offer a few remarks concerning this structure in health. The pericardium is a fibro-serous or bursal sac, which surrounds the heart and the adjacent portions of the great vessels. It is of a somewhat conical shape, the base of the sac being connected with the diaphragm ; whilst its narrower portion is directed upwards. The external or fibrous layer is dense and resistant ; it is attached firmly to the central tendon of the diaphragm, more loosely to its muscular structure, especially towards the left, by areolar tissue.

The fibrous layer is continued for some distance along the large blood-vessels in the form of tubular prolongations, which become gradually lost upon and incorporated with their sheaths, being thus fixed to the cervical fascia. The inferior vena cava passes through the floor of the pericardium at its right inferior angle, to reach the heart, which is tethered to the sac by the attachment of the vessel to the foramen quadratum.

The serous membrane lines the fibrous sac, and is reflected over the surface of the heart, thus constituting its parietal and visceral portions. These portions are continuous along the great vessels, about an inch to an inch and a half above the base of the heart ; the aorta and pulmonary artery being enclosed in a common tubular sheath, and a passage, named the "transverse sinus," being formed between these vessels and the auricles. The serous layer is also reflected on the superior vena cava and pulmonary veins. The heart is thus bound to the pericardium by two mesocardia—arterial and venous. The inferior vena cava has a very scanty covering. The "oblique sinus" covers the posterior aspect of the left auricle, and forms a serous diverticulum between the heart and oesophagus. A triangular fold—the "vestigial fold" of Marshall—formed by a duplicature of the serous layer enclosing areolar tissue and fat, with vessels and nerves, passes between the left pulmonary artery and the subjacent pulmonary veins. The pericardium has an abundant supply of vessels, lymphatics, and nerves, the last being derived from the phrenic, vagi, and sympathetic nerves.

In relation to the anterior thoracic walls, the pericardium occupies a triangular area, the normal limits of which are as follows :—To the right a line one inch from the right margin of the sternum extending from the insertion of the second to that of the sixth costal cartilage ; below a line corresponding to the diaphragm, from the insertion of the sixth costal cartilage to the fifth left intercostal space $3\frac{1}{2}$ inches from the margin of sternum ; to the left a line from this point to below the second left costal cartilage half an inch from the sternum. The apex lies behind the sterno-manubrial joint between the second costal cartilages. In the greater part of its extent, however, the pericardium is separated from the anterior wall of the chest by the pleurae and lungs, with which it is in contact in front and laterally. It only approaches the surface below in an angular space behind and to the left of the sternum, a space which varies in extent and shape in different instances. Under perfectly normal conditions the uncovered portion is somewhat triangular in outline, with the base below ; and is bounded on the right by a line along the middle of the sternum from between the fourth cartilages, on the left by a line from the same point to the apex of the heart. The pericardium is attached slightly to the sternum by sterno-pericardial ligaments. Behind it is in relation with the contents of the posterior mediastinum ; and the structures to be more especially remembered on this aspect are the oesophagus, descending aorta, bifurcation of the trachea and left bronchus, and the other structures which form the root of the left lung.

The phrenic nerves pass down, one on each side of the pericardium, on their way to the diaphragm.

In health the contiguous surfaces of the pericardium are kept moist by the usual serous secretion. This never collects in such quantity as to be capable of detection by physical examination during life, though at
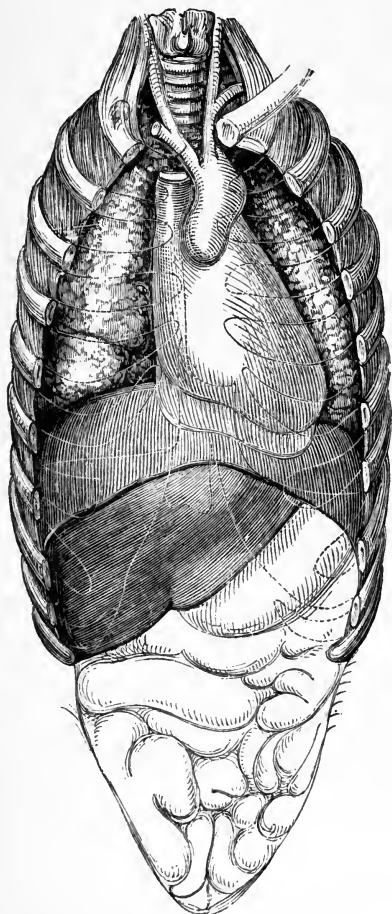
Fig. 10.—Pericardium not distended. (Sibson).
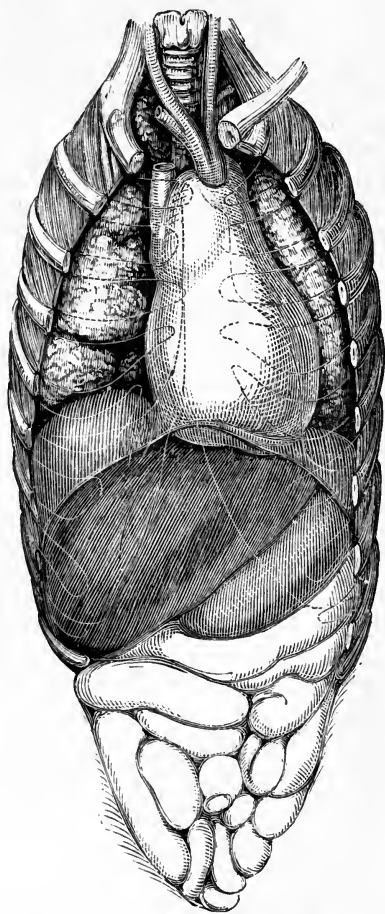
Fig. 11.—Pericardium artificially distended with fifteen ounces of fluid. (Sibson).

post-mortem examinations more or less fluid is always found in the sac, and it may amount to an ounce or two, or even more. Part of this, however, and in some cases most of it, has certainly exuded after death. The rubbing together of the surfaces during the cardiac movements does not give rise to any appreciable external sign.

Sibson found by experimental injections that the pericardium of

an adult man with a healthy heart is capable of holding from 14 to 22 ounces of fluid ; that of a boy between six and nine years old, about six ounces. The heart does not completely fill the sac, and is capable of more or less movement within it, which may be considerable. According to Dr. L. Hill and Mr. Barnard, the normal function of the fibrous pericardium is to restrain dilatation of the heart. Dr. T. Fisher, however, disputes this on the ground that the sac is usually far too capacious to exert any such influence ; whilst clinical experience frequently shews that there is ample room within it to allow of great displacement of the organ.

SUMMARY OF MORBID CONDITIONS OF THE PERICARDIUM.—The pericardium is liable to certain very definite morbid changes ; but, before discussing the more important of these, it will be convenient to refer briefly to certain conditions of this sac, which, although morbid, are in the large majority of instances more of pathological than of clinical interest or consequence, being indeed usually only revealed when a necropsy is made.

1. The pericardium in exceptional cases is the seat of more or less extensive *congenital defect*. (*Vide* p. 292.)

2. *Diverticula* or hernia-like pouches have been met with very rarely in connexion with the pericardium. They are the result of pressure from within ; usually by chronic pericardial effusion, exceptionally by blood. The fibrous layer becomes thinned or yields at a spot, and the serous lining protrudes as a sac, with a wider or narrower opening ; it is generally of small size, but has been found sufficiently large to contain three to four ounces of fluid.

3. In the case of a greatly enlarged heart, the pericardium will of necessity become more or less *stretched* and *distended* in proportion to the size of the organ, and it may become *thinned* in the process. I am not aware that such a condition in itself gives rise to any discoverable signs or injurious consequences, but it may be assumed to exist under such circumstances. An aneurysm of the heart wall, or of the intra-pericardial portion of the aorta, would also tend to push out the sac locally, and might even perforate it. Should pericardial effusion occur in such cases the signs might be unusual.

4. At post-mortem examinations certain *white spots* or *patches* (*maculae albidae*) are frequently observed associated with the pericardium, the nature and origin of which have given rise to far more controversy than their importance demands. They are also known as *tendinous* and *milk-spots* (*maculae v. insulae tendineae v. lacteae*), and as " corns " or " callosities." These conditions certainly cannot be regarded as normal ; and the main discussion has turned on the question whether they are or are not the result of inflammation. It cannot be doubted that the great majority of the pericardial white spots and patches are not of acute inflammatory origin at any rate ; and the meanings attached to " chronic inflammation " by different pathologists are so totally at variance, that it really does not matter whether we attribute them to such a process or not. My personal

opinion is that these changes are in the large majority of cases directly due to the constant mechanical attrition or irritation to which certain parts of the pericardium are subjected during the cardiac movements. They are met with in progressive frequency as age advances ; it has been affirmed, indeed, that they do not occur in children at all, but this state- ment is incorrect, though they are extremely rare in such subjects. They are decidedly more common in males than females ; and also in persons in whom, from their occupation, much friction between the pericardial surfaces might be expected.   Moreover, the white spots are by far most frequently observed on the visceral pericardium, over the portion of the front of the heart which, being uncovered by lung, comes chiefly into contact with the inner surface of the chest wall, that is to say, the base or middle of the right ventricle ; and they are not uncommon at the apex of the left ventricle.   They do occur, however, on other parts of the surface ; at the origin of the great vessels ; as white stripes on the auricles ; and along the course of the coronary arteries.   They are met with very exceptionally on the parietal pericardium.   Some of these changes are similar to those which affect other serous membranes, and cannot be very well explained : others are no doubt the remnants of a definite past pericarditis, when they present special characters, and are occasionally accompanied by adhesions or their remains in the form of filamentous fibrous bands : or there may have been a localised and trifling " dry " inflammation, which has not been detected during life.

Milk-spots are most common on large, hypertrophied and strongly acting hearts, but they are by no means confined to organs of this descrip- tion.   In character and structure they are whitish and more or less opaque, being in some cases of a dead white or pearly colour ; generally circular in outline ; of varying size, being usually about half an inch in diameter ; and, as a rule, cannot be detached from the serous membrane, with which they seem to be intimately incorporated.   Indeed they then consist merely of a local fibroid thickening or sclerosis of this structure, due to a hyperplasia of the connective tissue ; rather perhaps to a con- densation of fibres previously existing than to a development and increase of new fibres.   Occasionally patches are met with presenting a smooth or granular surface, decidedly opaque, and of some degree of thickness and firmness, which can be peeled off from the underlying membrane, with which they are more or less loosely connected.   Such patches are undoubtedly inflammatory in origin.

Clinically these conditions are generally regarded as of no con- sequence.   Certainly they do not give rise to any cardiac symptoms whatever, and as a rule are not revealed during life by any signs.   From personal observation, however, I feel sure that some pericardial white spots or patches are capable of originating a limited friction-sound which, in certain circumstances, might, without due care, be mistaken for an early sign of acute pericarditis.   Some very curious loud friction-sounds, varying in character—crunching, scraping, brushing, etc.—have been described by several observers as audible over the lower sternal region,

which have been attributed to these conditions, but I have never met with such pronounced phenomena in my own experience.

5. In rare instances what may be called *foreign bodies*, lying free in the pericardial sac, have been found at necropsies. Some of them have been soft and smooth, varying in size from a pea to a bean ; others firm, fibrous, occasionally stratified, or calcified, either in a central nucleus or throughout—the so-called *pericardial calculi*. These bodies have been regarded as polypi detached from the inner surface of the pericardium ; or as results of fibrinous or calcareous deposits around some foreign substance. They have never been diagnosed during life.

6. Very occasionally the pericardium, from no known cause, is the seat of *melanosis*, in which black pigmentation of the internal parietal surface is observed. The pericardial fluid exhibits at the same time cells containing particles of similar pigment.

7. It may be mentioned, lastly, that, as a consequence of prolonged chronic pericarditis in extremely exceptional instances, the pericardium becomes the seat of extensive *calcareous deposit*, which may actually convert it into a complete *calcified shell* surrounding the heart ; and the change may even encroach upon the cardiac walls, constituting the so-called "bony heart." Calcified spots or patches in connexion with this sac are not uncommon. Although these conditions might be suspected in certain circumstances, it is very doubtful whether they can be demonstrated clinically ; yet it has been affirmed that a calcified pericardium may give rise to a peculiar percussion-sound of an osteal quality.

Having thus disposed of changes of the pericardium which are almost exclusively of pathological interest, I now proceed to deal with those diseases which are clinically important ; and, taking a comprehensive survey, they may be indicated as follows :—I. Acute fibrinous and sero-fibrinous pericarditis. II. Suppurative pericarditis — Pyopericardium. III. Chronic pericarditis—Chronic effusion—Pericardial adhesions and thickening. IV. Hydropericardium—Dropsy of the pericardium. V. Haemopericardium—Blood in the pericardium. VI. Pneumopericardium and its effects—Gas in the pericardium. VII. Tuberculosis, Malignant growths, and Hydatids.

The diseases just enumerated are attended with pathological effects which give rise to well-recognised abnormal conditions, often of a very pronounced character. These conditions not only affect the pericardium and its contents, but also frequently influence neighbouring structures ; whilst in most cases they are revealed clinically by well-marked and characteristic physical signs. It is very desirable at the outset to have a definite general knowledge of their nature, and of the signs to which they severally give rise. They may be comprehensively summed up as :—(i.) abnormal states of the pericardial surfaces ; (ii.) accumulations of fluid or its remains in the pericardial sac ; (iii.) accumulations of gas, or of gas and fluid together ; (iv.) pericardial adhesions of various kinds ; (v.) thickening of the pericardium, usually associated with adhesions, whether of inflammatory origin, or due to new growths. It must be remembered

that these abnormal physical conditions may be variously combined in particular cases.

I. Acute Fibrinous and Sero-Fibrinous Pericarditis. Acute Inflammation of the Pericardium

Acute inflammation and its results constitute by far the most frequent and important morbid conditions of the pericardium with which we have to deal in medical practice ; and they often lead to serious consequences, both immediate and remote. As an acute affection pericarditis varies considerably in different cases, whether as regards its intensity and extent, the rapidity of its progress, the nature and amount of its pathological products, or its terminations and ultimate effects ; but the complaint must always be looked upon with concern. In some instances it may be described as subacute rather than acute, but there is no line of demarcation between the two groups.

Etiology and Pathology.—The valuable article on "Pericarditis" contributed by Sibson to Reynolds's *System of Medicine*, founded on extensive personal observations, still claims attention, and I shall refer to it in relation to several points in the following discussion of the subject. It will be convenient to discuss its etiology and pathology under two main headings.

Bacteriology.—It is now generally recognised that acute pericarditis, like other inflammatory affections, is immediately due in the large majority of cases, if not in all, to the action of micro-organisms. Certain of these microbes have been frequently demonstrated in the inflammatory products and in the pericardium itself, and the disease has been produced experimentally. Where they have not been found, their presence may usually be reasonably inferred on general grounds. The relation of particular organisms to the several etiological varieties of acute pericarditis will be considered in their appropriate connexion, and it will suffice to state here that those which have to be chiefly borne in mind are the different kinds of streptococci and staphylococci, pneumococcus, *Bacillus tuberculosis*, *B. coli* in certain cases, and the gonococcus in connexion with gonorrhoea.

Etiological Classification.—Formerly it was customary, from an etiological point of view, to divide cases of acute pericarditis into *primary* or *idiopathic* and *secondary*. There is, however, no valid foundation for such a division, and it will be more useful to arrange them in certain well-recognised groups, as they come under observation in ordinary practice.

(*a*) *Rheumatic Pericarditis.*—This is by far the most common and important variety. The definite connexion between acute rheumatism and pericarditis has long been recognised ; and the pericardial inflammation is not to be looked upon as a mere complication, but as an essential part of the disease. The morbid changes are believed by some to be

immediately set up by the *Micrococcus rheumaticus*. The existence of this organism, however, has not been satisfactorily established (*vide* Vol. II. Part I. p. 606), and at any rate other pathogenetic organisms probably take part in the process. The frequency of the association has been very differently stated by different writers, and doubtless it varies in different circumstances. In rheumatic cases pericarditis is not nearly so common as endocarditis, and Sibson noted that, in the large majority of cases of pericarditis, endocarditis was also present. Sturges drew special attention to this association in children, and he applied the names *peri-endocarditis* or *carditis* to the combination, which he regarded as exclusively rheumatic. Modern experience is fully in accord with these observations, and the combined effects of pericarditis and endocarditis come before us in a considerable proportion of the cases of chronic heart disease which can be traced to one or more rheumatic attacks in early life. More recently several writers have laid special stress upon the association of myocarditis as an essential part of the rheumatic carditis in children ; this point will be more conveniently dealt with later on (p. 43).

Contrary to what was formerly believed, it is now generally recognised that rheumatic pericarditis is by far most frequent during childhood and early life. Sturges pointed out that pericarditis is very common in children. He noted that " out of 100 fatal cases of heart disease occurring at the Children's Hospital, Great Ormond Street, of which 54 were of rheumatic origin, and 46 due to other causes, in 6 only was there no evidence of pericarditis." Dr. Cheadle has also spoken of pericarditis as less and less frequent with the advent of puberty. According to Dr. Poynton the incidence of rheumatic pericarditis increases from the age of five years up to ten, and thus contrasts with pneumococcic pericarditis ; in a series of cases of pneumococcic pericarditis in children he found that 84 per cent occurred before the age of four years (69). So far as my own experience goes, while prepared to meet with rheumatic pericarditis at any age, it is in children, growing boys and girls, and young adults that I have found it necessary to be more particularly on the look-out for the complaint. It is comparatively infrequent after twenty-five to thirty years of age.

Rheumatic pericarditis is on the whole more common among males than females, but this does not apply to its occurrence in childhood. Sibson explained the difference in part by the influence of age and occupation on acute rheumatism and its complications. Domestic servants formed fully two-thirds of the female patients under his care affected with pericarditis ; and three-fourths of these subjects were below the age of twenty-one. Women who at mature age followed occupations as laborious as that of the young servants were affected with pericarditis in but a moderate proportion, and in a comparatively mild form.

With regard to males Sibson observed the following facts :—Of laborious workers out of doors attacked with pericarditis only 1 in 10 was below the age of 21 ; whilst of indoor workers thus affected fully three-fourths were below that age. The scale was entirely reversed in

those of older age. Of those labouring out of doors four-fifths were above 25 ; whilst of those working indoors only one-sixth were above that age. Sibson writes : "We here, I consider, find the explanation of the twofold fact, that the male cases of pericarditis usually combined with endocarditis outnumber the female cases by one-fifth, and that the number of the men so affected above the age of 25 is three times as large as that of the women. I think we may infer that excessive labour in men of mature age is a frequent cause of acute rheumatism having a strong tendency to pericarditis."

The relation between rheumatic arthritis and acute pericarditis must next be considered. Sibson noted that in servants attacked with pericarditis the severity of the joint affection in the great majority of cases bore a strict relation to the severity of the heart affection. This rule, however, by no means applies to a considerable proportion of rheumatic cases, and it is highly important to remember that pericarditis may set in and become very pronounced while the articular complaint is comparatively or actually mild ; and it may even occur alone, or come first of the rheumatic series. This statement applies particularly to children, who are liable to cardiac inflammation of rheumatic origin with little or no joint affection or pyrexia, though there may be other rheumatic manifestations, such as chorea. The number of articulations involved, and the implication of particular joints, bear no relation to the frequency of acute pericarditis. The disputed question whether it is more prone to occur during first or subsequent rheumatic attacks is not of much practical significance, though the general experience is in favour of first attacks. The development of the affection must be watched for during every attack, whether it has or has not previously occurred, unless indeed it has left behind universal adhesion.

As to the time at which acute pericarditis supervenes in the course of a rheumatic attack, it appears in a certain proportion of cases at the very beginning, being coincident with the joint affection ; or, as already stated, it may even precede such a manifestation. Not uncommonly it supervenes between the third and the sixth day ; and, according to the late Dr. George Balfour, most cases occur within the first week of the rheumatic onset. In nearly one-half of Sibson's cases signs of pericarditis were observed on or before the eleventh day of the illness. On the other hand, the complaint may not be revealed for two or three weeks or even a longer period. Moreover, it may follow a relapse of articular rheumatism, the pericardium having been quite unaffected during the primary attack. In the case of children pericarditis may arise at any stage of the rheumatic series, but, according to Cheadle, it most often comes late, in association with recurrent endocarditis, when the heart is already hypertrophied and dilated.

The opinion has been advanced that excessive action of the heart, set up by the rheumatic condition, may help in the production of acute pericarditis. This was evidently Sibson's opinion in explanation of his view as to the relative severity of the joint affection and that of pericarditis.

Very cold, damp, and changeable climate and season have necessarily an indirect disposing influence upon the frequency of cases of rheumatic pericarditis.

(*b*) *Renal Pericarditis.*—The association of acute pericarditis with Bright's disease is well established, though statistics seem to shew that the frequency of this form depends on circumstances, and it differs materially in different countries. In renal disease pericarditis has been chiefly attributed to streptococci or other organisms acting upon tissues, the resistance of which is lowered; formerly it was thought to be due to uraemic toxins (*vide* Vol. IV. Part I. p. 617). With regard to its relative frequency in the several varieties of Bright's disease, it appears from Sibson's statistics to be uncommon in connexion with acute scarlatinal nephritis in young subjects, but frequent in adults who suffer from acute Bright's disease, as well as during the transitional stage to the large white kidney. When the latter has become established, however, the tendency to general pericarditis disappears almost entirely ; yet it may occur in a partial or circumscribed form. The complaint is most common in connexion with the chronic contracted or granular kidney. It may also occur in cases of fatty and lardaceous kidney. Renal acute pericarditis is more common in elderly persons, over fifty years of age, and is usually a late complication, being often a precursor of fatal uraemia. Sibson believed that over-action of the heart increases the tendency to pericarditis in Bright's disease, as well as the enlargement of the organ associated with the granular kidney.

(*c*) *Pericarditis from Extension or Irritation.*—The occurrence of pericarditis as the result of extension from neighbouring structures is now generally recognised. In most instances it follows pneumonia or pleurisy, more particularly when the inflammation is on the left side, being then usually set up by the pneumococcus, but other organisms may take part in the process. It must be noted, however, that in some cases in which these combinations of acute inflammatory diseases are met with in the chest, the pericardium has been first involved, and from it the inflammation has spread to other structures ; or the whole of them may be implicated so rapidly that it is difficult or impossible to determine where the inflammation started. The pneumococcus may originate pericarditis as a primary and independent complaint. Here it will suffice to mention that cases are now and then met with in which pericardial inflammation follows some neighbouring morbid condition apart from the inflammatory diseases just considered, such as abscess, aneurysm, enlarged glands or tumours, or bone disease. In exceptional cases the process may extend, through the diaphragm, from the peritoneum to the pericardium, without any direct communication between the two cavities.

(*d*) *Traumatic and Perforative Pericarditis.*—These very exceptional forms may be considered together. The chief injuries from without which may cause pericarditis are a blow or contusion over the precordial region ; fractured ribs ; penetrating wounds by sharp instruments or gunshot wounds ; and lesions produced by way of the oesophagus, especially

by foreign bodies, purposely or accidentally swallowed, which may actually perforate the pericardium, or even gain access into its cavity, or, remaining lodged in the gullet, injure the adjacent pericardium—examples of such bodies are false teeth, needles, or fish-bones.   Perforative pericarditis may result from the bursting of any neighbouring abscess into the sac ; or, in very exceptional instances, a communication may be established from an empyema, from a phthisical cavity, or from the oesophagus if it be the seat of ulceration or new growth.   Still more rarely the contents of an abdominal abscess find their way through the diaphragm into the pericardium ; and even a gastric ulcer has perforated its walls.   In all these cases definitely irritating or septic materials gain access to the pericardial sac, with abundant organisms of different kinds, including the *Bacillus coli* in certain cases of abdominal origin.

(e) *Pericarditis secondary to Cardiac or Aortic Disease.*—A separate group may be recognised of cases in which acute pericarditis is secondary to some affection of the heart itself, or of the arch of the aorta.   Its association with endocarditis and myocarditis has already been referred to, and it may follow these forms of cardiac inflammation.   Very rarely pericarditis is due to the bursting of an abscess in the walls of the heart into the sac. Among very exceptional causes may be mentioned cardiac aneurysm or intra-pericardial aortic aneurysm.   With regard to chronic diseases of the heart, pericarditis has now and then appeared in cases of valvular affection, chiefly aortic, especially when associated with cardiac hypertrophy ; but the connexion between these conditions is not very clear, and careful investigation in such cases would probably reveal some more definite cause of the pericardial inflammation.

(f) *Pericarditis associated with New Growths* (vide p. 101).

(g) *Tuberculous Pericarditis.*—When the inflammation is set up in connexion with chronic pulmonary tuberculosis, apart from the bursting of a vomica into the sac, it is essentially a slow process ; but in acute pulmonary or very active tuberculosis it may certainly be acute.   In very exceptional cases pericarditis seems to be the main tuberculous manifestation, and it is then rather subacute in its onset and mode of progress. The tubercle bacillus is the chief organism concerned, but other infective microbes also take part in setting up the inflammatory process.

(h) *Septic Pericarditis.*—This variety may arise in all kinds of general septicaemia and pyaemia ; though in such cases the pericardium is far less frequently affected than the pleura.   Septicaemia associated with puerperal conditions and acute necrosis of bone have to be especially remembered in this connexion.

(i) *Pericarditis associated with Miscellaneous General Diseases and Blood-states.*—It will suffice under this heading to draw attention to the fact that in exceptional instances acute pericarditis occurs as a complication of some of the acute specific diseases, particularly scarlatina (most commonly during the period of desquamation, when it has been attributed to rheumatism or renal disease), measles, influenza, and small-pox ; rarely of enteric fever, typhus, diphtheria, erysipelas, cholera, severe malarial

fevers, and gonorrhoea (due to the gonococcus). It has also been met with in scurvy, purpura, and haemophilia, where it is probably secondary, in some cases at any rate, to pericardial haemorrhage ; and may occur exceptionally in the gouty state, and in diabetes. The supposed idiopathic form of pericarditis has been associated with alcoholism, exposure to cold, and privation, but it is easy to understand how readily in these conditions pathogenetic organisms act injuriously upon the pericardium.

Morbid Anatomy.—The changes which occur during the progress of acute pericarditis are similar in their general nature to those which characterise inflammation of other serous membranes. It is customary to describe the disease as following successively the stages of—(i.) Hyperaemia or increased vascularity ; (ii.) fibrinous exudation ; (iii.) fluid effusion ; (iv.) absorption ; and (v.) adhesion. These stages, however, cannot always be definitely recognised, and in many instances they run more or less concurrently. Moreover, the fibrous pericardium itself is usually more or less invaded by the inflammatory process. It will be expedient, in the first place, to describe in succession the changes which take place during the progress of a pronounced case of acute pericarditis ; and afterwards to point out the more important aspects under which they are presented in practice.

(i.) Hyperaemia, or increased vascularity, no doubt constitutes the earliest change in acute pericarditis. It involves the serous lining of the sac and the subserous tissue, both parietal and visceral, and is accompanied with more or less parenchymatous swelling of the membrane, which loses its normal polish or glistening appearance, and becomes dull and opaque or velvety. In its lesser degrees the hyperaemia is revealed by a fine network of vessels ; but in its more pronounced form the surface is extensively and uniformly red, the redness being either bright or dark. The network is most marked over the large coronary vessels at the base and septum of the ventricles. Increased vascularity may be evident on the external surface of the pericardium in some cases. Sometimes minute haemorrhages are observed, especially around newly-formed vessels. The hyperaemic condition is of short duration, it may last but a few hours, and then either subsides or is concealed by exudation. As a matter of fact it is seldom seen at necropsies, and usually only in pericarditis associated with Bright's disease. Under the microscope changes are observed in the endothelial cells, which become swollen and granular, and some of them are cast off.

(ii.) The deposit of fibrinous exudation or inflammatory lymph is an invariable accompaniment of acute pericarditis ; though its quantity, extent, mode of arrangement, and exact characters vary much in different cases. As a rule it is observed both over the surface of the heart and the interior of the pericardial sac. In some instances there are merely a few shreds about the roots of the great vessels ; in others a thin film or coating forms at different spots, especially on the visceral surface ; or a more or less thick and stratified layer covers both surfaces extensively or universally, and is often very abundant. Owing to the

incessant movements of alternate contraction and expansion of the heart, the arrangement of the exudation is often peculiar. It very rarely presents a smooth surface ; and in the large majority of cases exhibits an alveolar, reticular, or honeycomb pattern. Laennec's oft-quoted comparison likens the appearance to that presented on suddenly separating two smooth pieces of wood between which a small pat of butter has been forcibly compressed. It has also been called the "bread-and-butter-like" appearance ; or has been compared to tripe. It must be noted, however, that the fibrin does not always present this kind of arrange-ment ; it may exhibit a shaggy or villous surface, or peculiar characters, to which such names as *cor hirsutum, cor tomentosum*, have been applied. When abundant, it is said to accumulate in large masses in the auriculo-ventricular groove and about the auricles. Should there be much fibrin associated with fluid its surface is covered with floating shaggy processes, which sometimes have a mammillated aspect. Occasionally fibrinous papillae or bands pass across between the opposing surfaces of the pericardium, and these may even form partitions.

The lymph exuded in pericarditis is usually of a whitish-yellow, yellowish, or reddish colour ; but it may be brownish. In a very short time a fine network of vessels is developed in its substance, and not uncommonly spots of haemorrhage are noted, or the whole exudation may be deeply stained. In connexion with purpura, scurvy, and allied diseases alternating layers of blood and lymph are now and then observed. In consistence the material is, as a rule, somewhat firm and elastic, but it may present different degrees of softness down to that of an almost liquid jelly. Not infrequently it is intermingled with serous fluid. In exceptional instances of a low type it has been described as granular, crumbling, or boggy. At first the exudation can be readily separated and peeled off from the surface of the membrane, but after a while it becomes more adherent and difficult to detach. In structure it consists of coagulated fibrin and cell-elements, the latter chiefly occupying the deeper layers. When the material is very soft the cells are in great abundance, and at the same time molecular disintegration has taken place. Micro-organisms of different kinds are usually found in the exudation.

It will be convenient to refer here to the changes in the pericardium in the course of acute pericarditis. When the fibrous structure is invaded by the inflammatory process, it becomes more or less swollen and thickened, sometimes to a considerable degree, as well as softened and sodden. The softening effect of inflammation upon fibrous tissue appears to be more marked in children than in adults. Microscopically, the pericardial sac is found to be infiltrated with cells, which are either leucocytes or derived from connective-tissue cells.

(iii.) There can be no doubt that in not a few cases of acute pericarditis there is little or no fluid effusion ; a form of "dry or plastic pericarditis" being met with, which can be recognised clinically. In such cases very rapid adhesion may take place between the visceral and parietal surfaces, even over an extensive area ; the lymph being thick,

sticky, gelatinous, and specially agglutinative. This course of events has been particularly noticed in children. Occasionally a kind of network of fibrinous strings passes between the adjacent surfaces, the meshes of which are filled with serum. As a rule, however, during the progress of an attack of acute pericarditis, where there are no adhesions, a definite effusion of fluid takes place into the pericardial sac, separating its parietal and visceral layers. Effusion may indeed supervene after the formation of early soft adhesions, sometimes limited to one side ; or when the sac is partially filled with heavy gelatinous masses of lymph. The average quantity of fluid is from 8 to 12 ounces, but it may range from an ounce or two to two or three pints or more. The amount of effusion is by no means in proportion to that of the fibrinous exudation, and the result of the inflammatory process may chiefly be evidenced by either one or the other product. According to Sibson, it is in rheumatic pericarditis that large accumulations usually occur, and the effusion then generally collects and increases rapidly, often reaching its acme in two, three, or four days. In opposition to this statement Sir John Broadbent affirms that "it is the exception rather than the rule to find effusion of any extent in cases of pericarditis of rheumatic origin." Sir William Church seems to be of the same opinion (*vide* art. "Rheumatic

FIG. 12.—Case of pericarditis in which the sac contained 3¼ lbs. of fluid. (Sibson.)

Fever," Vol. II. Part I. p. 622) ; and Dr. Cheadle has stated that in children the effusion, though fluctuating in amount, is never very large, and is usually reabsorbed quickly. In my experience, cases of rheumatic pericarditis have differed very much in the quantity of effusion. In Bright's disease the quantity is often very small. Abundant effusion is likely to be met with in scorbutic cases, in which as much as five pints have been recorded.

The effusion in acute pericarditis is generally of a serous or serofibrinous character, and yellowish or greenish in colour ; it is most commonly bright, clear, and transparent ; but may present small fibrinous particles or flakes in suspension, or be opalescent, or even more or less cloudy and opaque. Occasionally it is brownish or reddish. The specific gravity averages about 1018. Pericardial effusion is highly

albuminous, and often coagulates spontaneously. In certain circumstances, as when pericarditis is associated with purpura or scurvy, the fluid is obviously mixed with more or less blood or its colouring matter— "haemorrhagic pericarditis." The cases in which the inflammation leads to the formation of pus will be separately discussed (*vide* p. 71). In very rare instances the contents undergo a putrefactive change, and become "ichorous," foul in appearance and odour, or actually stinking. Pneumococci in great abundance have been found in pericardial effusion, and other microbes are present in other forms of the complaint.

Effects of Pericardial Effusion.—It will be convenient in the present connexion to discuss briefly from a general point of view the immediate effects of pericardial effusion upon the sac itself and its contents, as well as upon neighbouring structures, effects which are met with by far most frequently in cases of acute pericarditis. Obviously they must vary considerably in nature and degree, according to the amount of the fluid accumulation, and the rapidity of its collection. It must be acknowledged that a certain quantity of effusion is sometimes found in the pericardium at the autopsy, it may be as much as 6 or 8 ounces, which had not given rise to any evident disturbance, and was not detected during life. In all such cases, however, which have come under my personal observation, there has been every reason to believe that the effusion had taken place shortly before death, from obvious causes, and usually in circumstances rendering adequate physical examination impracticable ; and no doubt it is often increased by transudation of serum from the vessels after death.

Beginning with the *pericardium* itself, when a collection of fluid exceeds a certain quantity the sac necessarily becomes more and more distended, in proportion to its amount, and at the same time stretched and thinned, so far as the normally tough and firm parietal pericardium will permit. When the sac itself, however, undergoes the changes already described, it becomes capable of far greater distension than in its natural state. As the fluid accumulates in increasing quantity the pericardium undergoes changes in form, which were well described and figured by Sibson. When artificially distended with 15 ounces of fluid, he noted that the pericardium became pyramidal or pear-shaped, being formed of a larger and a smaller sphere, the smaller one resting on top of the larger, and reaching up almost to the top of the sternum. The distended sac occupied the whole centre of the chest, filling up the space between the sternum in front and the spinal column behind, and extending across the chest from a little within the right nipple to a little beyond the left nipple. Its floor presented a large spherical prominence bulging downwards into the epigastrium, and reaching as low as the tip of the ensiform cartilage and the lower edge of the sixth costal cartilage. This description will apply to the shape which the pericardium usually assumes when distended with fluid from pathological causes ; but when it collects in larger quantity, the form alters considerably. The sac yields sideways and backwards, and widens to the right and especially to the left. Its

width thus becomes decidedly disproportionate to its height, and it loses its pyramidal outline, becoming in extreme cases almost globular.

What is the mode in which a pericardial effusion collects, and what position does the *heart* assume within the sac? These questions have been the subject of special controversy; and although to some writers they present no difficulty, and are unhesitatingly answered in a particular way without reserve, I must confess that in my own clinical experience of individual instances I have not always found them easy of solution. It may be of interest to explain Sibson's later views on this subject. Describing the mode in which fluid collects in the pericardium, he writes: "At first it falls into the back part of the sac, but as it increases in quantity it makes a space for itself between the floor of the pericardium, which it depresses, and the lower surface of the heart, which it elevates; . . . and the result of this is to displace the apex and body of the organ and its great arteries upwards and forwards." He adds: "The heart, . . . leaves the broader part of the chest below, and ascends into the narrower part of the chest above." In another place he writes: "The distension of the pericardium with fluid produces two other effects on the heart. (*a*) The heart is heavier than the fluid in which it plays, and its ventricles consequently tend to sink backwards, so that the left ventricle rests upon the posterior wall of the pericardium. (*b*) The other effect of pericardial distension on the heart is the lifting or tilting upwards of the organ within the sac. The heart is attached by its great vessels to the posterior and upper part of the sac, and the whole organ therefore tends to shrink upwards and backwards towards its points of attachment."

The displacement of the apex of the heart upwards and outwards in cases of pericardial effusion was formerly taught as an indisputable rule. Most authorities at the present day, however, are opposed to this doctrine. The general opinion is that the fluid collects towards the front, and that the heart, being heavier than the fluid, falls or sinks backwards, away from the anterior thoracic wall; the ventricles, right auricle, and great vessels being successively covered from below upwards, and thus separated from the parietal pericardium. Some writers have maintained that an effusion first collects about the base, which is turned downwards, the heart lying rather more horizontal than normal, and the apex turned outwards; but this part is described as descending when the diaphragm is pushed down by the effusion.

Another opinion is that the position of the heart is not altered. Dr. William Ewart (31) affirms that the apex will be found in the usual situation in any necropsy on a case of uncomplicated pericardial effusion; and that whilst the heart has preserved its normal situation the floor and the sides of the pericardium have receded from it. He regards the impossibility of any elevation of the apex as almost self-evident; and argues that the anatomical relations of the heart to the pericardium must tend to depress the apex, far from allowing it to rise. This observer has in some cases detected a lowering of the heart's apex in pericardial effusion, and with it a more median position of the heart, which then tends

to hang more vertically from the aortic arch, the latter becoming slightly straightened.

Sturges believed that " the heart may be moved either forwards, upwards, or backwards in effusion ; or it may remain where it was ; and of the factors that determine its conduct, pericardial adhesion, here or there, temporary or permanent, is the chief." He further stated : " I have repeatedly in fatal cases of pericardial effusion inserted needles, just before the post-mortem examination, into the proper apex place, and above the fifth right costal cartilage, close to the sternum, without being able to detect upon opening the chest any dislocation of the heart. . . . There are clinical facts to shew that the early pushing forward of the heart, . . . although it may be the rule, is not without exception. The fluid may cover the heart from the first." My personal opinion is, that when dealing with particular cases it is well not to have too fixed or positive an opinion as to the position of the heart in pericardial effusion. Should the sac be quite free, there can be no doubt that in very abundant effusions the organ is covered by the fluid.

The next question is what effects, if any, are produced by pericardial effusion upon the walls of the heart and great vessels, when it becomes so considerable as to interfere directly with these structures ? Sibson writes on this point : " The muscular walls of the ventricles are so thick, and their action is so powerful, that the direct effects of the fluid pressure upon them cannot be very great. But the pressure of the fluid tells inwards upon the weak and unresisting walls of the auricles, the vena cava descendens within the pericardium, and the pulmonary veins, so as to compress and lessen the cubic contents of those vessels and the auricles, and to resist and impede the currents of blood, on the one hand from the system along the cava, and on the other from the lungs along the pulmonary veins. This partial blocking of the double stream from the system and the lungs to the heart lessens the contents of the organ, and tends to diminish the size of its cavities. At the same time the supply of blood to the aorta is lessened, and the ascending aorta is therefore also compressed by the fluid. The pulmonary artery, however, owing to the obstacle to the flow of blood through the lungs, tends to resist the pressure of the fluid in the swollen sac, and to remain distended." This seems to be a correct description in the case of large effusions. He was further of opinion, however, that in cases of pericarditis the compressing influence of pericardial effusion is counteracted by the protecting and sustaining covering of lymph, which to some extent shields the weaker parts of the heart, and strengthens the naturally feeble walls of the auricles and veins.

As regards the effects of pericardial effusion upon the action of the heart, it is believed that the systole of the auricles and ventricles is not restrained by such a collection ; indeed, according to Traube, the systolic motion of the organ is greater than normal, the fluid being less resistant than the pericardium. The compression of the walls already referred to may, however, interfere with the diastolic distension, and thus

diminish the flow of blood into the cavities, especially into the auricles. The direct interference with the entrance of the blood from the veins into the auricles, and impairment of the normal elastic traction of the lungs upon the walls of the heart, add to this difficulty.

Cardiac Changes.—Apart from the frequent presence of endocarditis along with pericarditis, there are certain conditions affecting the heart itself which demand special notice. It is well recognised that myocardial changes are commonly associated with this complaint, more particularly the rheumatic variety occurring in children and young subjects. These are either the result of an inflammatory process—myocarditis or carditis; or of an acute degeneration, which may be pronounced and extensive. According to one view such changes are secondary to the pericarditis, being, speaking generally, proportionate to its intensity and duration, and affecting primarily and chiefly the superficial layers of muscle, immediately below the serous visceral layer, but gradually extending until they may ultimately involve the entire thickness of the cardiac walls. This is the view maintained, among more recent writers, by Dr. Sequeira, who states that general myocarditis is rare. On the other hand, several observers (Sturges, Cheadle, Lees, Poynton, Fisher, and Coombs) regard the myocardial lesions not as a sequel of pericarditis, but as part of a general carditis due to rheumatic infection, and they consider that these lesions may probably occur independently, without any pericardial inflammation. Moreover, myocardial changes are believed to constitute the most important factor in an individual case of rheumatic carditis in early life, as regards the immediate effects and danger to life; whilst their remains may be met with at a later period, in the form of fibrosis or fatty change, when recovery takes place. In support of this view Dr. Poynton has demonstrated by microscopical sections of a specimen that the changes in the heart-walls commence by numerous scattered foci, some of them far from the pericardium, and that they are general—involving also the interstitial tissues. He attributes them to the action of toxins derived from micro-organisms gaining access through the circulation. The coronary arteries may be the seat of acute arteritis or other infective lesions; and it has been suggested that pressure on these vessels by inflammatory lymph might cause degeneration of the myocardium, by impeding the normal supply of blood. Pericardial effusion, however abundant, does not seem to have any direct influence upon the nutrition of the cardiac muscle. It may be noted that the nerves distributed to the surface of the heart and great vessels may be implicated in the inflammatory process in cases of pericarditis.

Acute ventricular dilatation is another dangerous condition of the heart very liable to be associated more particularly with rheumatic pericarditis in early life, and it may become extreme (Sturges, Lees and Poynton, J. F. Broadbent); it not uncommonly occurs in dry or plastic pericarditis. How the dilatation is brought about is disputed. It is probable that in some cases the rheumatic toxin may so act upon the myocardium as to produce mere impairment of function, and diminished

resisting power, without any obvious morbid changes in the tissues. The commonly accepted explanation is that the dilatation follows, and is the result of the myocardial lesions just referred to. Dr. Sequeira is opposed to this view, and maintains that the cardiac enlargement is secondary to dilatation of the fibrous pericardial sac, which is a consequence of the softening of this structure due to the inflammation. He agrees with Dr. Hill and Mr. Barnard that the normal function of the pericardium is to prevent dilatation of the heart beyond a certain point ; and insists that not only may the pericardium be acutely dilated with effusion to a remarkable extent, but that the plastic form of pericarditis also softens it, thus diminishing its resisting power. " The heart dilates and distends the yielding pericardium, which, while still inflamed, becomes adherent to the chest-wall and diaphragm." He further believes that dilatation of the pericardial sac is helped by the latency of pericarditis in children, and its liability to recur ; but considers the too early resumption of muscular effort after an acute attack, before the fibrous sac has become consolidated, as the most potent cause of such a condition. Moreover, Dr. Sequeira regards the movements of chorea, when violent and of long duration, as deserving of special consideration, as a factor in dilating the pericardium. The condition may be extreme from the outset, or progressive in its development.

Neighbouring Structures.—It will be obvious that distension of the pericardium with fluid must interfere with neighbouring structures in proportion to its amount, and such consequences are chiefly seen in connexion with the respiratory apparatus. Some observers maintain that the portions of the lungs in front of the sac are pressed at first against the inner surface of the anterior wall of the chest. The ordinary effects of pericardial effusion upon these organs are complex. It necessarily embarrasses them more or less, and large collections of fluid also press upon the bifurcation of the trachea and the main bronchi, especially the left bronchus. Hence it is found in many cases that the upper lobes of the lungs, particularly the right, are in a state of inflation, and in time become the seat of catarrh also, while other portions are collapsed in various degrees. As the effusion increases, and becomes excessive, it pushes these structures to either side and backwards, at the same time compressing them more and more, the left lung especially, which in extreme cases may become almost or even completely collapsed and airless. Rapid and repeated serous effusion may take place into one or both pleurae in connexion with great pericardial distension, especially during the later stages, and it has been described as among the most common complications. When not inflammatory the condition is regarded as of mechanical origin, being attributed to pressure on the vessels in the roots of the lungs.

A very abundant pericardial effusion may press upon the oesophagus and descending aorta sufficiently to interfere with their channels. Whether the phrenic or other nerves within the thorax may be affected by the mere physical consequences of such an accumulation it is difficult

to say ; but some observers are of opinion that this may be the case, and it is highly probable, especially if the effusion be rapid.

A considerable pericardial effusion will tend to cause more or less protrusion of the corresponding portion of the thoracic walls, particularly in young subjects. When these walls have become rigid no such protrusion can take place. In a downward direction the diaphragm is not only embarrassed, but often considerably depressed, as well as the contiguous viscera, as chiefly evidenced by the liver.

(iv.) The course of events and the ultimate pathological results in acute pericarditis differ much in different cases. The natural tendency is for any serous or sero-fibrinous effusion to become absorbed sooner or later, sometimes very rapidly. There is every reason to believe, moreover, that even fibrinous exudation, up to a certain amount, can be absorbed completely, after undergoing a molecular fatty change ; some degree of pericardial thickening or opacity at the most being left behind. The probability of such absorption is in inverse ratio to the extent and thickness of the lymph deposited, and to the duration of the inflammation. In respect of the " white spots " on the pericardium, it may be well to note again that those resulting from pericarditis are usually distinguished by greater thickness and extent, irregular distribution, and special characters, and as a rule by the coexistence of adhesions. Very rarely irregular knob-like projections or pedunculated outgrowths are formed, and the latter may even become detached, and lie loose in the pericardial sac.

(v.) In most cases, after absorption of the fluid, or where only lymph has been exuded, adhesions of various kinds and degrees are formed. At first these are soft and easily broken down, and on account of the movements of the heart firm and permanent adhesions are much less easily established than in the case of other serous membranes. Loose adhesions of connective tissue are probably torn by the repeated pulling and stretching ; and it has been suggested that the cardiac action considerably interferes with the circulation in the newly-formed vessels.

I have a strong impression that there is still a general tendency to make light of the conditions remaining after acute pericarditis, or at any rate not to regard them as of much consequence ; and I feel it necessary, therefore, to insist that well-marked pericardial adhesions not uncommonly persist, particularly in young subjects, and subsequently often become of decided importance. A new growth of connective or fibrous tissue takes place, originating mainly in the cells present in the exudation ; the fibrinous portion taking no part in the process, but being absorbed after undergoing fatty degeneration. In severe cases the changes affecting the tissues of the pericardium contribute to the fibrous growth. As the subject of pericardial adhesions is separately discussed in this article no further reference need be made to it here. It must be noted that in exceptional cases an ordinary inflammatory effusion into the pericardium does not undergo absorption, but remains

as a chronic collection, or may become haemorrhagic or purulent. These conditions are referred to more fully on p. 74.

According to the extent of the disease, cases of pericarditis have been divided into *circumscribed* or *local,* and *diffuse,* the latter being in many instances *general* or practically universal. Local pericarditis may be met with in any part, but is chiefly observed at the base, about the origin of the great vessels ; and the inflammation may thence extend to the coats of the arteries, so far as they are covered by pericardium, and subsequently give rise to thickenings and callosities.

In the preceding discussion pericarditis has been dealt with only so far as it affects the sac internally. It must be mentioned, however, that in not a few instances the external surface of the pericardium is acutely involved at the same time, or alone ; though it is more commonly implicated in a chronic process. The condition has received the names of *external pericarditis, mediastino-pericarditis,* or *pleuro-pericarditis* when the contiguous surfaces of the pleura and pericardium are affected. This form of disease and its results are more conveniently dealt with elsewhere.

Clinical History.—Acute pericarditis presents considerable differences in its clinical history, depending upon a number of circumstances ; and this must be always borne in mind in practice. At the same time the phenomena to be watched for and studied are definite, and when at all pronounced bear an obvious relation to the morbid changes which are associated with the disease. The signs revealed by physical examination are of special clinical value, for the symptoms are not uncommonly far from characteristic, whilst the more important of these signs can be investigated as a rule without much difficulty, and it is only by their aid that we can positively determine the pathological conditions affecting the pericardium. Indeed, it must never be forgotten that, when symptoms are practically absent or latent, they may reveal the presence of even serious acute pericarditis ; and this statement applies still more to cases in which the inflammation is localised. Moreover, physical examination gives the only trustworthy information as to the progress of the morbid changes.

Taking a comprehensive survey of the circumstances in which acute pericarditis usually supervenes, it might be anticipated that an attack is not ushered in, as a rule, by any striking premonitory symptoms, such as rigors and the like ; and experience confirms this conclusion. In certain classes of cases, however, the illness may begin with phenomena of this nature ; nor must it be forgotten that even rheumatic pericarditis may appear as a primary acute disease, before the joints or any other structures reveal the presence of the rheumatic condition.

Discussion of Symptoms.—From what has just been stated, it may be gathered that it is useless to attempt to give a definite clinical picture of acute pericarditis, and it will be more practical in the first instance to consider individually the several symptoms which may be associated with this disease ; remembering that they differ much in their exact nature, severity, and combinations in particular cases.

(i.) *Subjective Sensations.*—Pain is a symptom to be looked for in the early stage of acute pericarditis; but it is by no means always present, nor does it bear any necessary proportion to the seriousness of the attack. Severe pain may certainly be associated with a limited dry pericarditis of short duration; whilst, on the other hand, it is well recognised that in cases of large effusion no such sensation may have been complained of from first to last, or it may have been so slight and transient as not to have attracted any attention. In young children pain seems to be generally absent. In the majority of cases in which pain is present it is referred to the precordial region, extending usually from the right of the sternum at its lower two-thirds to the left nipple. This pain is more or less continuous, but varies in severity, being in exceptional instances very intense. In character it is described in different cases as dull, aching, shooting, stabbing, burning, or tearing. Sibson noted that it came on, as a rule, at an early stage, afterwards diminishing; and usually relief, which was permanent, came when the effusion was at its height. Occasionally a return of the pain occurs with a relapse. The suffering is often increased by deep pressure or percussion; and now and then there is tenderness without spontaneous pain. In many cases there is superficial hyperaesthesia over the precordial region, which may be so pronounced as to forbid the slightest manipulation of the chest, and to make a full physical examination impossible. In other cases the structures of the intercostal spaces seem to be tender.

Another not uncommon seat of pain or tenderness, or both, is the epigastric region. The tenderness is said to be most marked at one or other of the costal angles, and is particularly brought out when upward pressure is made. Epigastric pain comes on, as a rule, later than that over the heart, and in a considerable proportion of Sibson's cases it appeared when the effusion was at its height. Both varieties are likely to be increased by the act of respiration and by bodily movements. Sometimes painful sensations radiate in different directions from the central points. A deep pain in the chest, between the shoulder-blades, was noted in a few cases by Sibson, who supposed it to be seated in the back of the inflamed pericardium; it was increased by swallowing or eructation, and occasionally was only thus brought out. In exceptional instances pain of an anginal character, shooting up the left side of the neck, to the ear, to the shoulder, or down the arm, is associated with acute pericarditis; but endocarditis has almost always been present at the same time, and generally chronic valvular disease as well. The sensations just discussed are believed to be located mainly in the sentient nerves distributed to the surface of the heart, the pericardial sac itself and the portion of diaphragm incorporated with it, or the pleura covering the pericardium. They are often associated together in different combinations. Moreover, there may be pain in one or other side, evidently of pleuritic origin; or referred indefinitely to the chest, without any particular localization.

Other subjective sensations besides those actually painful are not uncommonly complained of in acute pericarditis, as the disease progresses; and especially if a large accumulation of fluid takes place. They are described in different cases as feelings of precordial uneasiness; oppression or pressure; a weight or load over the heart; tightness; or ill-defined distress and anxiety.

(ii.) *Disorders of the Cardiac Action and Pulse.*—It might naturally be expected that acute pericarditis would affect the action of the heart in various ways. In the early stage the heart is excited and irritable, as evidenced by increased rapidity and force of the beats, the movements in some instances being more or less tumultuous. Subsequently, not only as the result of large effusion, but also of the implication of the myocardium and its nerves, as well as of other factors, the cardiac action becomes more or less embarrassed and ineffectual, and this may culminate in marked feebleness or exhaustion, with irregularity and intermittence. The patient may be conscious of the disturbed cardiac action, and sometimes there is a distinctly painful form of palpitation. Moreover, faintness or actual syncope may occur, which, in exceptional instances, has come on suddenly or very rapidly, and proved fatal. With regard to the frequency of the pulse, according to Sibson, "it rises in number as the disease rises in intensity, is at its greatest rapidity when the disease is at its acme, and falls in number as the disease declines." "During the early stage the pulse usually mounts up to 90, 100, or even 120; but later on it tends to become more rapid, and in rare cases reached 160." It may, however, not be much changed from the normal, or from what it was before the pericarditis supervened; or after an initial acceleration it may soon subside. In exceptional instances the pulse is retarded in the course of the disease. A much-quickened pulse-rate, 120 or 130, without adequate rise of temperature, is said by Dr. Cheadle to be very characteristic of the subacute pericarditis of early life. In the early stage the pulse is generally full and strong, and tension may be increased; as the case progresses it becomes small, weak, often dicrotic, and of very low tension. Dr. Ewart has drawn special attention to the large, slapping, and suddenly collapsing pulse which he has frequently observed in pericardial effusion. Irregularity or intermittence may accompany a similar disturbance of the cardiac rhythm; occasionally this is an early phenomenon, but usually comes on later. It has been stated that in some cases of copious pericardial effusion the left carotid and radial arteries are smaller and pulsate less forcibly than the corresponding arteries on the right side (Traube). The sphygmograph has been much used to investigate the pulse in cases of acute pericarditis, but I venture to doubt whether it has proved of much practical value. Speaking from personal experience of this disease, I think it must be acknowledged that no definite or precise description of the pulse can be given; but at the same time its study in individual cases affords most useful information, and it needs to be constantly watched. In grave cases it may become almost imperceptible. The *pulsus para-*

doxus has been observed occasionally in connexion with large pericardial effusions.

(iii.) *Respiratory System.*—Some disturbance of breathing is noticed in the great majority of cases of acute pericarditis, varying much in its degree and exact characters, but often well marked or even decidedly grave. In the early period respiration is rendered quick and hurried, but restrained and shallow, on account of pain ; and this cause may also modify the movements later, when the physical effects of pericardial effusion, as well as other influences, especially the myocardial changes, come into play. If there be much fluid, actual dyspnoea supervenes, the respirations increasing in frequency, with marked activity of upper costal breathing, but more on the right side than the left. As it accumulates, the breathing becomes more and more difficult and laboured ; the alae nasi work ; the extraordinary muscles are called into play ; there is a corresponding sense of oppression, distress, and air-hunger ; and the patient may have to be propped up more or less. In extreme cases the dyspnoea is very urgent, the respiratory movements are greatly impeded, and there is persistent orthopnoea, or the patient instinctively bends forwards to seek relief. As a rule it is more comfortable to lie on the left than on the right side, but dorsal decumbency is usually preferred. Occasionally the dyspnoea is intensified paroxysmally. As the fluid is absorbed the respirations diminish in number, and the breathing improves ; but a relapse may cause fresh disturbance. The pulse-respiration ratio is changed, and even at the early period may be $3 : 1$; later the proportion may come to be $2\frac{1}{2}$ or $2 : 1$. The difficulty of breathing interferes with the act of speaking ; and changes in the voice have been noted in exceptional instances, attributed mainly to pressure upon or implication of one or both recurrent nerves. A short, irritable, spasmodic cough is not uncommon with a large pericardial effusion, and there may be a small quantity of mucous frothy expectoration. Bäumler noted painful sensibility of the left side of the larynx, increased by every movement of the heart. . Distressing and painful hiccup is an occasional symptom, attributed to implication of the phrenic nerve in the inflammatory process.

(iv.) *Dysphagia.*—Difficulty or pain in swallowing is occasionally noticed, mainly the result of the pressure of a large pericardial effusion upon the oesophagus ; but sometimes it appears to be due to nerve-irritation. Deglutition is more difficult in the recumbent posture, and is made easier by raising the shoulders and bending forwards. In exceptional cases the difficulty is only associated with swallowing solids ; or is brought on by oesophageal spasm induced by an attempt to drink. Rarely a feeling of spasmodic choking in the throat or along the gullet is complained of.

(v.) *General Symptoms and Appearance.*—More or less pyrexia may be expected in cases of acute pericarditis, but it does not present any special course or characters. In rheumatic cases the onset of pericarditis may not be attended with any increase of temperature previously raised ; it

seldom rises above 102° or 103° F. at any time, and may soon subside. Sometimes it is practically normal throughout, or only reaches from 99° to 100° or 101°, especially in children. It is affirmed that rapid absorption of inflammatory products may occasion some rise of temperature. As a rule, strength is fairly maintained ; but in some instances, particularly in children, there is marked prostration. In severe cases of acute pericarditis, especially when associated with endocarditis and myocarditis, the expression generally indicates anxiety, distress, or depression ; and the face is flushed, dusky, or pallid, or presents alternating hues. Rarely it has a muddy or glazed appearance. The eyes at the same time are dull, heavy and injected. Sibson attached much importance to the appearance of the patient ; as the complaint subsided he found that the aspect quickly improved, the eyes becoming bright and clear, the cheeks rosy, and the expression often quite suddenly cheerful.

The most striking general symptoms in the graver forms of acute pericarditis are those indicative of interference with the aeration of the blood, and of general venous obstruction. The patient then presents a more or less livid or cyanotic appearance ; with sweating, often profuse ; fulness of the veins of the neck, sometimes with pulsation ; and in extreme cases coldness of the extremities. Possibly oedema of the legs may supervene. A large effusion in children is said to affect the action of the heart more rapidly than in adults, and to lead to an earlier interference with the circulation. In these subjects progressive anaemia and wasting are in some instances pronounced symptoms. The amount and characters of the urine will depend very much upon the condition with which the pericarditis is associated. It tends to be deficient in quantity, and to present the usual changes associated with the rheumatic and febrile states. Albuminuria may occur altogether independent of renal disease.

(vi.) *Nervous Symptoms.*—Patients suffering from pronounced acute pericarditis are generally very restless, but movements may be checked by the rheumatic condition. Headache and sleeplessness are frequent symptoms, and slight delirium is not uncommon. Vomiting is sometimes a marked symptom in acute pericarditis, and is regarded as of nervous origin. In exceptional cases nervous disturbances become very prominent, and may be grave, such as delirium, either active and noisy, or even violent and maniacal, chiefly nocturnal ; or low and muttering : sometimes a transition from one to the other variety takes place. The condition may resemble delirium tremens, the patient being strange in manner, excited, and incoherent ; or there may be a tendency to stupor, semi-unconsciousness, temporary insensibility, or actual coma. Other phenomena which may be met with are motor disorders, such as subsultus tendinum and jactation, "risus sardonicus," clonic or tonic spasms, rolling of the head from side to side, choreiform movements, general convulsions ending in extreme exhaustion, or tetanic rigidity ; curious emotional attacks in early life, in which the child is moved to tears or laughter by a word (Cheadle) ; or temporary insanity, usually with taciturn melancholy, and often with hallucinations, which derange-

ment may last some time, but is ultimately recovered from. The particular symptoms of this class and their combinations differ much in different cases, and delirium may pass into coma. They cannot, as a rule be referred directly to the pericarditis, but depend rather on the disease to which it is secondary ; its associated complications ; hyperpyrexia in some instances ; the state of the nervous system ; want of oxygenation of the blood ; the previous habits of the patient, or other circumstances. Some authorities, however, have attributed the phenomena to the influence of the pericarditis upon the nervous system ; and Bright believed that such an influence can be communicated through the phrenic nerve to the spinal cord, and is the cause of choreic and tetaniform disorders. G. W. Balfour wrote : "The occurrence of delirium in the course of rheumatic fever ought at once to direct attention to the heart ; and the sudden occurrence of spasms or coma in chronic renal disease is only too frequently found to be associated with pericarditis." Nervous phenomena may be very prominent in grave forms of pericarditis in children. It is important to note, however, that even in cases of acute pericarditis ending fatally, and accompanied with other intrathoracic inflammatory affections, there may be no marked nervous symptoms throughout, the patient being perfectly clear to the last.

Physical Signs.—In discussing the physical signs of acute pericarditis, it is convenient to recognise certain stages corresponding to the progress of the morbid changes already described ; although it must be clearly understood that there is no actual line of demarcation between them, the conditions which give rise to these signs being commonly present at the same time. It may be remarked that the excited or turbulent action of the heart which often occurs at the onset of the disease will be evident on examination, but there is nothing characteristic in this disturbance.

First Stage.—During the early period the signs to be looked for are those indicative of abnormal states of the contiguous pericardial surfaces, which are pressed and rubbed against each other during the movements of the heart. They are commonly known as *pericardial friction-fremitus* or *thrill*, and *friction murmurs* or *sounds.* Many deny that any phenomena of this kind can be produced by mere increased vascularity and dryness of the surfaces, but in my opinion a faint friction-murmur may certainly be thus originated. It is, however, to the fibrinous exudation that the more pronounced and characteristic signs of the early stage of acute pericarditis are due. It appears to me that they can only be brought out when the conditions producing them exist on the anterior aspect of the heart, although some writers have made a contrary statement ; and it is highly probable that when the inflammatory lymph is of a very soft consistence, it may not give any definite sign perceptible on physical examination.

(i.) *Pericardial Friction-Fremitus or Thrill.*—The tactile sensation thus named is practically only recognisable in a comparatively small proportion of cases of acute pericarditis, and when present it is always accompanied with a loud friction-sound. For the detection of this sign careful palpa-

tion with the finger-tips may be needed, and I believe that it can thus be made out more frequently than is generally supposed. It depends more immediately upon the amount and characters of the exudation, though it is also influenced materially by the force of the heart's action.

When any abnormal sensation is felt over the precordial region, the chief point to be determined is whether it is a pericardial fremitus or an endocardial thrill. It must suffice to summarise here the more characteristic features of a pericardial fremitus, and to any one practically acquainted with the usual endocardial thrills the points of difference between them will be at once apparent.

(a) A pericardial friction-fremitus has no definite "focus of intensity," and varies much in its seat and extent. As a rule its area is circumscribed, and it is felt more towards the base of the heart or over the middle of the precordia ; sometimes it is limited to the apex. Now and then, however, the sensation is perceptible over a considerable extent of surface, or in more than one spot. (b) It always gives the impression of being peculiarly superficial, as if the condition producing it were close under the finger. (c) The rhythm is practically systolic, the fremitus being associated with the cardiac impulse ; it usually begins and ends rather abruptly, and there is no shock at the close : sometimes it is irregular in rhythm, differing in exact time in successive beats. (d) In quality a pericardial friction-fremitus gives more or less the impression of the rubbing together of rough surfaces, and in different cases it is described as harsh and grating, rasping, vibrating, thrilling, or creaking. (e) As a rule this sign is short-lived and transient ; and, should it last any time, often changes from day to day in its situation, extent, and characters. It must not be forgotten that pericardial friction-fremitus may be simulated by one of pleuritic or mediastinal origin, brought out by the movements of the heart.

(ii.) *Pericardial Murmur or Friction-Sound.*—It is by the adventitious sounds heard on auscultation that, in the large majority of cases, the early stage of acute pericarditis is recognised. Sibson and other writers have distinguished between a pericardial murmur and friction-sound ; but there is no practical line of demarcation between them. In the following remarks, therefore, I shall employ the term pericardial friction-sound inclusively, merely remarking that the so-called murmur may be regarded as representing the minor degrees of this sign, and that now and then an adventitious sound of pericardial origin may no doubt closely resemble an endocardial murmur in quality.

It is requisite to have a comprehensive and intelligent conception of the more characteristic features of pericardial friction-sounds, so as to be able to contrast them with those of endocardial murmurs ; but as a rule they are easily distinguished. Moreover, by careful attention to the special qualities of the sounds heard, it is practicable in many cases to arrive at a tolerably definite notion of the conditions of the pericardium upon which they depend. It must be noted that pericardial friction-sound may unquestionably be simulated by one of pleuritic origin, or

by a sound originating in the mediastinal cellular tissue over the peri-cardium.

(*a*) Whilst usually more or less circumscribed in extent, pericardial friction-sound does not correspond, as regards its situation or its point of maximum intensity, to any of the recognised endocardial murmurs. In some cases it is audible extensively, though not of the same loudness throughout its area ; but even then it is generally defined with remarkable abruptness, and is never conducted in the directions peculiar to the several intracardiac murmurs ; nor, according to my experience, can it ever be heard over the back of the chest. During the early stage of acute pericarditis friction-sound never extends beyond the normal region of the heart, but in the later period it may do so in exceptional instances. When associated with a fremitus it usually spreads, as from a focus, in all directions more or less beyond the area where this sensation can be felt.

(*b*) As a rule pericardial friction-sound has a double or to-and-fro rhythm, being both systolic and diastolic ; but in some instances, or over certain parts of the heart, it may be confined to the systole. In pro-nounced cases the two parts are of about equal duration, each sound seeming to fill up its respective space, with a short intermission between them. They may, however, occupy the whole time of the cardiac move-ment, thus often giving at first a confused impression to the ear. As regards the heart-sounds, the pericardial murmur seldom corresponds exactly in rhythm with either, and is prolonged beyond them, whilst they are often distinctly audible through it ; though, on the other hand, the friction-sound may be so loud as to drown them entirely. Moreover, its precise time is frequently irregular, varying with successive beats of the heart. This is more especially noticed in connexion with the diastolic portion, which is usually not so loud as the systolic. A double " to-and-fro " adventitious sound heard in connexion with the cardiac movements, of maximum intensity at the same spot, is regarded as highly characteristic of pericardial origin. It has been stated that four murmurs may be audible, the two sides of the heart each producing a systolic and diastolic murmur of different duration ; but that most frequently three are heard, one presystolic, belonging to the systole of the auricles, and two longer sounds, corresponding to the systole and diastole of the ventricles. Rarely pericardial friction is divided into several parts. A so-called triple rhythm has been regarded as very typical, produced by the two normal cardiac sounds, along with a single friction-sound.

(*c*) Whilst varying much in its intensity, pericardial friction-sound strikes the ear as being peculiarly superficial ; and this feature is more pronounced in proportion to its loudness.

(*d*) The precise characters of a pericardial friction-sound vary con-siderably within well-recognised limits, according to the nature of the conditions upon which it depends. In the large majority of cases it conveys to the ear a distinct impression of the rubbing together of con-tiguous surfaces during the cardiac movements ; in short, it is of the

quality of a "friction-sound." At first and in its lesser degrees it is soft or grazing, whiffing, brushing, or rustling; but its more pronounced varieties are described by such terms as harsh, rough, grating or vibrating, and creaking, like the bending of new leather. Sometimes it resembles the rubbing of sand-paper. In certain circumstances the sound is more of a crackling (as of paper or parchment), clicking, churning, or rumbling character; or it may be scraping, scratching, or sawing. It has also been described as "sticky." Whilst thus varying in character, pericardial sounds are as a rule entirely different in quality from endocardial murmurs. Moreover, the double pericardial friction-sounds never begin with an accent or shock, but begin, continue, and end, as a rule, with the same tone throughout (Sibson). When pericardial friction-sounds and endocardial murmurs exist together, the combinations may be very peculiar and difficult to unravel.

(e) *Tests.*—In certain cases in which a pericardial friction-sound is not distinctly audible, but its presence is suspected, or in which it is doubtful whether an adventitious sound heard on auscultation be pericardial, endocardial, or pleuritic, the difficulty may be cleared up by the judicious application of certain recognised tests. These may also help in affording a more correct knowledge of the conditions of the pericardial surfaces upon which a friction-sound depends.

(a) *Pressure Test.*—Firm but not too forcible pressure with the stethoscope over different parts of the region of the heart has long been known as an important and useful test of pericardial friction-sound. It may bring out this sign when not previously audible, especially over the lower two-thirds of the sternum (Sibson). Its effect upon the sound, when present, may be to intensify it and make it louder; to enlarge the area over which it is heard; to modify its duration and rhythm, rendering it more prolonged and continuous, or making it double—systolic and diastolic—when previously only systolic; to alter its character, tone, and pitch, causing it to become more harsh and rough, and especially grating or creaking, or making these qualities come out more prominently under pressure; or to silence the natural cardiac sounds previously heard, or even mask endocardial murmurs.

(β) *Respiration Test.*—The act of respiration may unquestionably produce a definite influence upon pericardial friction-sound, especially as regards its extent, less frequently as to its intensity and quality; and possibly some help in diagnosis might thus be afforded in doubtful cases. It is generally stated that inspiration always increases pericardial friction-sound. Sibson observed that the area of the friction-sound increased below during inspiration in a large number of cases; whilst in a much smaller number it increased above during expiration. It became more loud or harsh sometimes during expiration, sometimes during inspiration; and in one instance it disappeared at the end of a deep breath. Pleuritic friction simulating pericardial can as a rule be distinguished by its situation at the left border of the pericardium, and by its cessation when breathing is stopped, but certainly not always.

(γ) *Effects of Exertion and Posture.*—Should a pericardial friction-sound not be heard at all or but feebly, in consequence of weak action of the heart, it might possibly be brought out or made louder by exciting the organ by some kind of effort. Moreover, it certainly may be intensified or increased in area by bending the body forwards ; and occasionally it is audible in the recumbent but not in the sitting posture. Change of position may affect the locality and extent of this sign in certain cases. Personally I doubt whether the tests mentioned under this head are of much practical value, and at any rate special discretion and caution are demanded in carrying them out.

(δ) *Variability.*—Marked changes in the site, rhythm, intensity, and characters of pericardial friction-sound from day to day, or within shorter periods, constitute most important tests in a large number of instances.

Stage of Effusion.—When fluid collects in the pericardial sac in any quantity, a very definite group of physical signs may be expected, varying in their degree according to its amount and other circumstances. It must not be forgotten, however, that rapid adhesion may take place without any effusion, so that the phenomena of this stage may be entirely wanting, especially in children. Conversely, it occasionally happens that a large quantity of fluid accumulates very rapidly and insidiously without preceding friction-signs, or at any rate without their detection. The possibility of considerable cardiac dilatation must also be borne in mind, lest a wrong diagnosis of pericardial effusion be made.

We shall first consider how pericardial effusion may modify the friction-phenomena. According to Sibson's observations the tendency of the effusion is to shift the whole region of actual friction, and with it the friction-sound, upwards ; and steadily to increase its area in this direction and to the right and left. In the large majority of cases he found the area of friction-sound greater at the time of the acme of the effusion than before ; he also observed that the tendency is for the sign to increase in intensity. It may be stated with certainty that even large effusions do not necessarily obliterate the friction-phenomena ; indeed there may be an abundance of fluid, at least as much as two pints, in the pericardium, while these signs are pronounced. G. W. Balfour went so far as to affirm that if a friction-sound be once heard over the base of the heart in front, no amount of subsequent effusion suffices to efface it. I do not think that this statement will hold good absolutely ; and friction-sound over other parts of the precordia is likely to be completely silenced as the rising tide of fluid separates the two pericardial surfaces.

I proceed now to discuss the more positive signs which are associated in various degrees with pericardial effusion.

1. The tendency of pericardial effusion, when in sufficient quantity, is to cause proportionate bulging or prominence of the corresponding portion of the front of the chest, and occasionally this is a very striking sign. Some writers have asserted that this condition leads to a uniform enlargement of the left side ; but although there may be a certain degree of general distension the prominence is always greater in front. In the

case of a large effusion the margin of the sternum and the left costal cartilages are pushed forwards, while the ribs are raised bodily upwards, and the intercostal spaces widened. In extreme instances the fulness may extend from the second to the sixth or seventh cartilages, but chiefly from the fourth to the sixth. The spaces are sometimes felt to be quite smooth, and an obscure sense of fluctuation may possibly be detected in them. Bulging is naturally more easily produced in children and growing subjects, on account of the yielding condition of the chest-walls; whilst it may be entirely prevented by rigidity of these walls, which thus adds seriously to internal embarrassments by the fluid. The enlargement has been partly attributed by some writers to inflammatory paralysis of the intercostal muscles.

Dr. William Ewart (31) regards what he calls the "first rib sign" as important in the diagnosis of considerable pericardial effusion. He states that owing to the raising of the clavicle, and relaxation of the ligament between it and the first rib, the upper edge of the latter can be felt as far as its sternal attachment.

A prominence of the epigastric region may be noticed in cases of abundant pericardial effusion, due partly to the fluid itself pressing down the diaphragm, partly to the liver, which is also depressed and congested. Dr. S. West has described the rare phenomenon of a peculiar elastic semi-fluctuating depression in the epigastrium, which he regards as additional evidence of pericardial effusion. Sir Clifford Allbutt has met with a similar phenomenon.

2. Certain signs of pericardial effusion, associated with the cardiac movements, as revealed by the impulse and apex-beat, demand careful study:—

(*a*) There can be no doubt that one of the obvious effects of a free and uncomplicated accumulation of fluid in the pericardium is a real or apparent elevation of the impulse, which seems at the same time to be carried towards the left, it may be as far as, or beyond, the nipple-line. Moreover, the movement becomes unusually extensive in an upward direction, its diffusion being often easily recognised by inspection and palpation. According to Sibson's observations there is, as a rule, a relation between the extent of the effusion and the height of the impulse. This he found raised so that its lower boundary corresponded to the fourth or even the third space or cartilage. In exceptional cases the impulse was diffused from the fourth to the second spaces, but generally it was confined to the fourth and third, or the third and second spaces. Sibson believed that it is the actual apex-beat which is felt, displaced upwards and to the left. At the present time, however, most writers regard this opinion as erroneous, and consider that the impulse is communicated by a higher portion of the heart, nearer the base of the ventricles, which is actually in contact with the chest-wall. My personal observations lead me to agree with the latter view, though there may be conditions present in certain cases which cause actual uplifting of the apex-beat.

Series of figures (Nos. 13 to 20), from cases described by Sibson, illustrating the morbid conditions in pericarditis and the physical signs associated therewith. The black spaces correspond to the pericardial dulness, the curved lines to the impulses, and the zig-zags to the friction-sounds. In Fig. 18 there is complete adhesion of the pericardium to the heart.
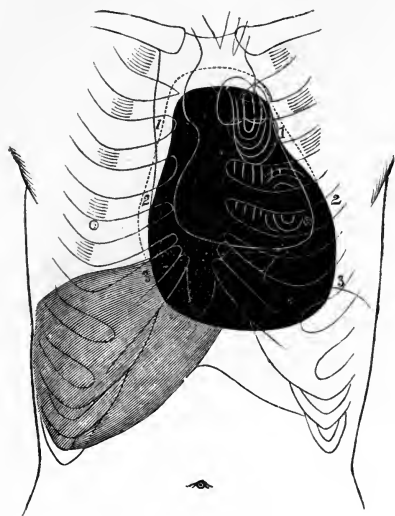
Fig. 13.
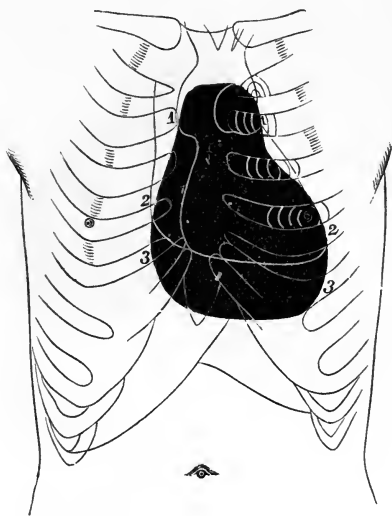
Fig. 14.
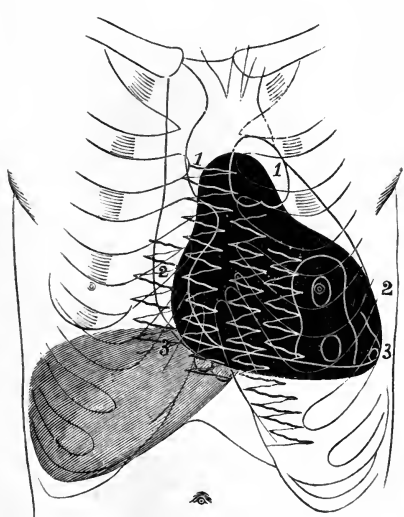
Fig. 15.
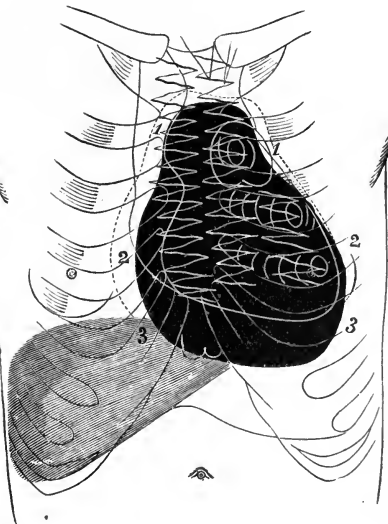
Fig. 16.

Occasionally it has been noticed in acute pericarditis with effusion that the apex-beat is somewhat lower than normal. This may be due to enlargement of the heart; but it has also been attributed to the presence
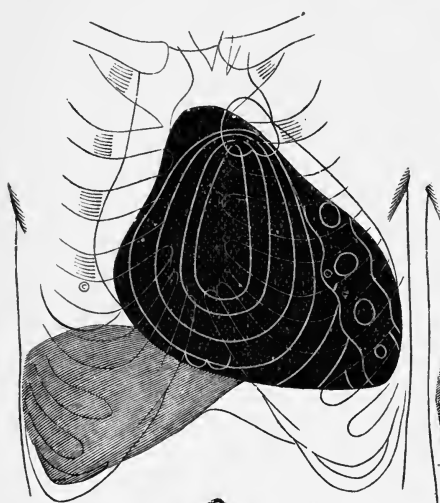
FIG. 17.
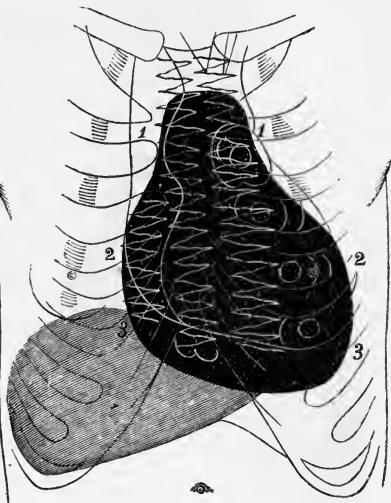
FIG. 18.
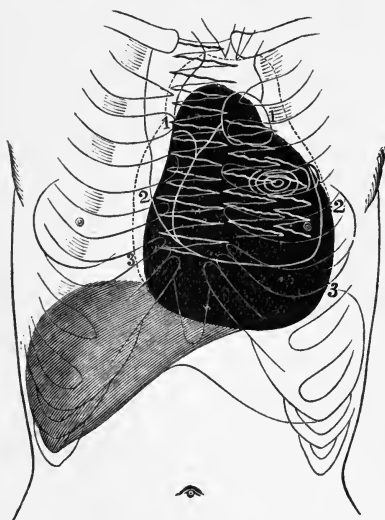
FIG. 19.
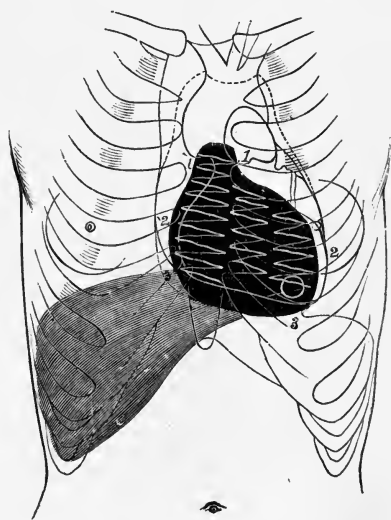
FIG. 20.

of a large quantity of fluid pressing down the diaphragm; or it may be associated with a more median and vertical position of the heart, the aortic arch becoming slightly straightened (Ewart).

Over the pulmonary artery at the base of the heart a double beat is sometimes felt, the second being the diastolic shock due to the closure of its valves.

(*b*) The next change to be noticed in pericardial effusion is a progressive weakening of the cardiac impulse from below upwards. This depends mainly upon the amount of the effusion, but partly upon feebleness of the heart's action. When the fluid is in moderate quantity there is often, as just stated, a strong impulse over the upper spaces, its lower and outer boundaries being also well defined. As it accumulates, however, in increasing abundance, and separates the heart from the chest-wall, the cardiac movements become more and more obscured, until finally they may be wholly lost, and not perceptible over any portion of the precordial region. This sign is occasionally very striking in a case of inflammatory pericardial effusion when it first comes under observation.

(*c*) It is a disputed question whether pericardial effusion can produce any definite change in the character of the cardiac movements, tactile or visible. Certainly the impulse observed over the upper part of the chest may be more or less undulatory, associated with the movement of the exposed heart. A wave-like motion has been described, which can be seen but not felt, supposed to be communicated to the fluid by the action of the heart; but I have never been able to recognise this phenomenon positively. Some authorities regard an undulatory impulse as a sign, not in favour of pericardial effusion, but against it.

(*d*) In some cases of pericardial effusion the rhythm of the impulse has been described as lagging behind the ventricular systole in a peculiar way. Irregularity, with or without inequality in the strength of the beats, may become very pronounced as the result of embarrassment of the heart by a large collection of fluid, and of associated changes in the myocardium.

3. One of the most frequent and characteristic signs of pericardial effusion of any extent is an increase in the area of the normal cardiac dulness, with change in its shape and outline; and not uncommonly these alterations are so pronounced as to attract immediate attention in cases of acute pericarditis. The exact quantity recognisable by percussion cannot be definitely stated, and no doubt it varies in different circumstances; but I believe that methodical and careful determination of the cardiac dulness may afford valuable information in cases in which the fluid is present in comparatively small quantity. It is necessary to study systematically and thoroughly both the superficial or absolute, and the deep or relative cardiac dulness. As the patient lies on his back the increase of dulness is first observed towards the base of the heart. The limits ultimately reached vary much in different cases. The extension takes place chiefly in a lateral and upward direction, the length and breadth of the dulness being thus increased; the former usually preponderating. In most instances it reaches the third cartilage or space, but may extend as high as the second cartilage or first space,

or even above the clavicle. Sansom maintained that whenever marked dulness extends above the third rib there is a strong probability of pericardial effusion. Over the sternum, which is absolutely dull, as the fluid increases the dulness gains a higher level than over the costal cartilages, and in extreme cases it may reach its upper margin. From side to side at its greatest width the dulness may extend from an inch or more to the right of the lower part of the sternum, or the right mammary line, to an inch outside the left nipple, or even to the left axilla. In a downward direction it seldom passes below the sixth rib, but in extreme cases it may be made out as low as the seventh or eighth rib, and be indistinguishable from the hepatic dulness. Rotch regards the presence of dulness in the fifth right intercartilaginous space, due to the accumulation of the fluid in the right corner of the sac, as a valuable aid in the early diagnosis of effusion into the pericardium— "Rotch's Sign."

A notable feature of the dulness in cases of considerable pericardial effusion is its shape, which corresponds with that of the sac itself. Thus it narrows from below upwards, assuming a more or less triangular, pyramidal, or, more strictly speaking, pyriform or pear-shaped outline, with its truncated or "peaked" apex above, and its base below, at the level of the lowermost limit of the fluid. The left border has been described as usually somewhat curved, or indented at its upper part, while the right is more nearly vertical. Dr. Ewart well describes the outline of a large effusion as "that of a bag of fluid spreading out at the base"; and lays stress upon the projection of the lower angle of the dulness to the right, as well as to the left. When the pericardium becomes extremely distended, the characteristic shape is more or less modified, and may ultimately be altogether lost.

In cases of pronounced pericardial effusion the extreme degree of the dulness is very striking. Sansom insisted on the importance of the well-defined transition from the resonance of the lung to such dulness as a factor in the diagnosis of this condition, and in many cases the contrast is certainly very remarkable. It must not be forgotten, however, that the distended pericardium may be overlapped by the margins of the lungs, which yield a superficial resonance ; and that its full extent can then only be made out by very careful percussion beyond the limits of absolute dulness. A large effusion imparts an increased sense of resistance to the fingers.

Another important point is that the dulness of extensive pericardial effusion can be made out distinctly towards the left, considerably beyond the position of the apex-beat, which is then only to be recognised by auscultation.

The rapid development of increased precordial dulness while a patient is under observation is strongly in favour of accumulation of fluid in the pericardium, and in circumstances in which acute pericarditis might be anticipated this sign must be specially looked for. It may soon become quite pathognomonic, but acute dilatation must not be forgotten.

4. The auscultatory signs which may directly result from effusion into the pericardium demand brief notice. The tendency of the fluid itself, as it increases in amount and rises higher and higher, is to weaken the heart-sounds in a progressive manner from apex to base; or they may seem deep and distant. These effects may be due both to imperfect transmission of the sounds through the intervening fluid, and to embarrassment with enfeeblement of the cardiac action. Most commonly in pronounced pericardial effusion the sounds are weak or perhaps inaudible over the region of the normal apex-beat, and for some distance upwards, but become gradually more perceptible towards the base of the heart, where they may be well heard; over the pulmonary artery the second sound may actually be intensified. In cases of extreme effusion the sounds may be practically absent over the whole precordial region.

Some observers have described a basic systolic murmur as a sign of pericardial effusion, the result of pressure by the fluid upon the great arteries. I have never met with such a murmur within my own experience, but it may possibly occur. On the other hand, pericardial effusion may certainly obscure or render inaudible endocardial murmurs previously heard.

5. *Signs connected with Neighbouring Structures.*—The effects produced on the lungs, especially the left, by a large pericardial effusion, are likely to be indicated by more or less pronounced signs, which, however, will vary in different cases according to their exact nature and degree. The respiratory movements over the upper part of the chest are often obviously excessive, especially on the right side; should the fluid be very abundant, a striking contrast will probably be observed between the activity of the two sides, the movements on the left being very deficient. Over the region of absolute cardiac dulness there will be entire absence of breath-sounds, as well as of vocal fremitus and resonance. Beyond its limits there may be hyper-resonance and puerile breathing; and towards the left the percussion-sound is occasionally somewhat tubular, and the breathing bronchial or tubular, with increased vocal fremitus and resonance. Dr. Ewart (31) has noted in severe cases of pericardial effusion tubular breathing below the right mamma, situated usually in the nipple-line, and sometimes restricted to expiration. Dry rhonchi of various kinds may be audible in severe and protracted cases, the result of catarrh of the bronchial tubes.

In considerable pericardial effusion the condition of the left lung may give rise to a definite group of signs at the back of the chest on that side; namely, a limited area of deficient resonance or actual dulness, about the size of a crown piece, generally localised in the vicinity of the angle of the scapula, with increased vocal fremitus, bronchial or tubular breathing, and bronchophony or aegophony. Sansom regarded these as valuable signs in children and young subjects. I have in a few instances noted them in older persons, in a pronounced degree. Dr. Ewart attaches special diagnostic importance to a patch of marked dulness at the left inner base, which he describes as of square shape, with abrupt boundaries,

extending for a variable distance from the spine outwards and upwards, but limited.   Over the dull patch respiratory sounds are absent and voice-sounds feeble.   He attributes these signs to the altered dorsal relation of the liver; and states further that partial dulness extends for a short distance to the right of the corresponding vertebrae, and that, when the effusion is considerable, the extension of the patch in the right chest may become almost absolutely dull.

As previously stated, pleural effusion on one or both sides is not uncommon as a consequence of a large collection of fluid in the peri-cardium; in which case the signs will be modified accordingly.   When it begins on the right side the contrast may be helpful in diagnosis.   Signs indicative of downward displacement of the liver are very pronounced in cases of extensive pericardial effusion; and there may also be some degree of enlargement due to venous congestion.

6. *Effects of Change of Posture.*—The study of the effects produced by changes of posture upon the chief signs just discussed has been regarded as important in the diagnosis of pericardial effusion.   In a large propor-tion of cases, however, these signs are so definite that it is quite unnecessary to test them in this way, and such a procedure may be highly dangerous.

The following are the chief modifications in the signs produced by changes of posture, which are regarded as of more or less diagnostic value. It may happen that the impulse is not perceptible in the recumbent position, but becomes evident when the patient is made to sit up or bend forwards.   Increased mobility of the apex-beat with change of posture has also been looked upon as important, but certainly this is very untrust-worthy, to say the least.   The effects of position upon the dulness have been more particularly insisted upon as evidence of pericardial effusion, and in doubtful cases may be worth studying.   It is increased in extent, especially at its upper part, in the sitting posture, and still more if the body is bent forwards.   It may also be modified in a lateral direction, as the patient turns to either side.   The relative loudness of the cardiac sounds or of endocardial murmurs might also possibly be similarly influ-enced.   Sansom described modifications of the signs observed in connexion with the left lung posteriorly, produced by making the patient bend well forward, or assume the knee-elbow position; but it seems to me that such a procedure may be extremely dangerous, and is only warranted in exceptional cases of very doubtful diagnosis.

Examination by the x-rays has been employed in the investigation of pericardial effusion, but there is still much to be done in this direction. Dr. H. Walsham and Mr. Orton, writing in 1906, say, "The appearances will vary with the amount of effusion present.   When large amounts of effusion are present, the shadow of the cardiac area is increased and its outlines are somewhat rounded and different from the normal outlines. The pulsations of the left border are notably diminished, and may be obliterated.   The cardio-phrenic space also is obliterated in small effusions. An inclination of the patient to one or other side might modify the cardiac outlines; the patient should, therefore, be examined in different positions.

A careful study of the retro-cardiac triangle in the left lateral examination should not be omitted, as, if this triangle is well marked, there is probably not much, if any, effusion present."

Stage of Absorption.—During the progress of absorption of inflammatory pericardial effusion the signs indicative of this condition progressively diminish, until the phenomena become practically normal, or point to the formation of adhesions. The friction-signs, if they have been obscured by the effusion, return for a while, or they alter in their situation, intensity, extent, and characters. Friction-sound in most cases increases in a downward direction as the fluid declines (Sibson). It lasts a variable time. Friction-fremitus may at this period be noticed for the first time ; and the friction-sound is often rough and creaking or churning. The dulness diminishes more or less rapidly from above and laterally ; while at the same time the cardiac sounds become more distinct. Should one or more relapses take place, with further increase of the fluid, the signs return, again to subside as the fresh effusion becomes absorbed. What the ultimate position of the heart and its apex-beat will be depends on the course of events. As a rule, in simple and uncomplicated cases of pericarditis the organ returns to its normal situation, but this return may be prevented by adhesions, by the effects of endocarditis, or by other causes. The signs indicative of adherent pericardium will be separately considered, but it may be remarked that in not a few instances, if carefully watched for, they can be traced in process of development during the period of convalescence.

Course and Terminations.—As already stated, acute pericarditis presents much diversity in its clinical history, and it does not follow any uniform course. When, however, the symptoms and physical signs just discussed have been adequately and intelligently mastered, they can be studied with advantage in individual cases on the lines indicated. Among the chief circumstances which influence the nature, severity, and combinations of the symptoms, may be mentioned the causation of the pericardial inflammation, and the character of the disease to which it is secondary ; its intensity and rapidity of progress ; the characters and amount of the inflammatory products, especially of the effusion ; the presence of previous organic changes affecting the heart or pericardium, or of other chronic intrathoracic diseases ; and the association of the pericarditis with endo-carditis or myocarditis, or with pleurisy or pneumonia.

Attempts have been made to classify cases of acute pericarditis into groups, according to the intensity of the symptoms, and the morbid changes affecting the pericardium and heart associated therewith ; but distinctions of this kind are quite arbitrary, and have no practical founda-tion or value. It may be affirmed that as a rule the clinical phenomena are not so pronounced or so grave as is commonly supposed, or as the older writers used to describe. Not uncommonly the symptoms are not at any time prominent ; they may be practically latent, or quickly attain some degree of severity, and as speedily subside. In some instances one or more relapses or recurrences take place, with corresponding increase

of the symptoms after their subsidence. Acute pericarditis may run a favourable course in a few days, even when there is considerable effusion which then undergoes rapid absorption. The entire duration of the majority of cases is from eight or ten days to a fortnight, but not uncommonly longer. Convalescence may not be established for three to six weeks or more, or the disease, after beginning more or less acutely, may afterwards assume a subacute or chronic course. The course of rheumatic pericarditis in children is described by Dr. Cheadle as usually subacute, chronic, recurrent. As a rule the complaint terminates in recovery, so far as the immediate result is concerned, and no doubt in a considerable proportion of cases the restoration is practically complete ; but in not a few instances definite organic changes are left behind, the effects of which are sooner or later revealed, it may be within a short period. Sometimes the patient can hardly be said to recover, a condition of obvious chronic pericarditis being established, with well-marked symptoms and physical signs, which will be considered later on. It is impossible to make any definite statement as to the direct fatality of acute pericarditis, and the more important points bearing upon this matter will be more conveniently referred to under prognosis. It may be affirmed, however, that death is seldom due solely to this affection, though evidences of pericardial inflammation may not uncommonly be found at post-mortem examinations, or it may partly contribute to the fatal result. Occasionally acute pericarditis assumes a very grave aspect from the first, advancing with great rapidity, exhibiting extremely severe symptoms, and ending in death within a short time, it may be even in less than twenty-four hours ; but such a course of events only occurs in special circumstances, and mainly in haemorrhagic cases.

Diagnosis.—Several important matters bearing upon the diagnosis of acute pericarditis have been sufficiently dealt with under its clinical history, especially in the discussion of its physical signs ; and in further consideration of this part of the subject, I propose merely to draw attention to its more prominent aspects.

An ordinary case of acute pericarditis arising in the course of definite rheumatic fever ought to present little or no difficulty in diagnosis, if due attention be paid to the symptoms and physical signs. Remembering, however, that the inflammation may supervene very insidiously in this complaint, and when the joint-symptoms are absent or not pronounced, it is necessary, whenever any rheumatic condition is suspected, to be constantly on the watch for its appearance. Nor must it be forgotten that pericarditis may be the first manifestation of such a condition. From these points of view it is a disease to be particularly watched for in children, though in such subjects its symptoms and signs, as well as its mode of progress, may be very anomalous, even where there is well-marked or perhaps a large pericardial effusion, a state of things, however, which ought not to occasion much difficulty to an intelligent and practised clinical observer. The occurrence of acute pericarditis in other than rheumatic cases may easily be overlooked, but it should always be borne

in mind at any rate as a possible complication of Bright's disease, or not uncommonly of pneumonia or pleurisy.

Assuming that the diagnosis of pericarditis has been made, it is obviously very important to determine, within due limits, and without endangering or needlessly distressing the patient, the actual morbid conditions present, and more especially the amount and characters of fluid effusion, as well as the changes which take place during the progress of the case. Most of these points can be positively made out by physical examination only, conducted on the lines already explained. It must not be forgotten that extensive friction-sound is not incompatible with a considerable effusion. The rapid development of general pericardial adhesion in some cases is also worthy of note, especially in children. The probability of the fluid being haemorrhagic, suppurative, or ichorous is mainly founded on the conditions with which the pericarditis is associated, and on the general symptoms ; yet these may be in no way characteristic.

What other conditions of the pericardium, or of the heart itself, are apt to be confounded with pericarditis ? A dropsical accumulation—*hydropericardium*—may certainly be mistaken for an inflammatory effusion, especially if it be abundant. However, the circumstances in which it occurs ; the fact that it usually follows hydrothorax ; the absence of symptoms of pericarditis and of any friction-phenomena ; and, as a rule, the comparatively small amount of the effusion, will usually enable a diagnosis to be arrived at readily. A morbid growth involving the pericardium has more than once been mistaken for pericarditis with effusion. The distinction of pericarditis from endocarditis at an early stage is mainly founded on the differences between the tactile and auscultatory signs already discussed, but the symptoms may also help. When marked effusion occurs, any previous difficulty is cleared up. Of course the frequency with which the two diseases are associated together, especially in children and young subjects, must always be borne in mind. Implication of the myocardium is indicated by evidences of serious embarrassment and feebleness of its action, and when grave symptoms arise in the course of pericarditis, changes affecting this structure may be regarded as highly probable. Much has been written about the difficulties of distinguishing between pericardial effusion and cardiac enlargements, especially dilatation, but in my opinion they have been greatly exaggerated, due consideration being given to all the facts of an individual case. Of course it is possible that a much dilated heart, especially if associated with extensive adhesions, might be mistaken for an effusion ; and such a mistake has actually been made several times, the heart having been punctured in an operation for the removal of a supposed pericardial collection of fluid. Difficulty might also arise when acute dilatation with rapid adhesion occurs in pericarditis, instead of effusion. Special value has been attached to the projection of the lower angle of dulness to the right, as well as to the left, in the diagnosis of effusion from dilatation. Should inflammatory effusion supervene where the heart is already enlarged, and the pericardial sac distended, the diagnosis might be obscure ; as well as when

acute inflammation involves a limited area of the pericardium, the rest of the sac having been obliterated by previous adhesions. Possibly the x-rays might afford help in diagnosis in difficult cases.

The diagnosis of acute pericarditis from neighbouring conditions is, as a rule, quite easy. Occasionally the distinction between this complaint and pleurisy might be difficult, and certainly this applies to the friction-sound. A superficial exo-pericardial sound, or even a fremitus produced in the mediastinal cellular tissue, might also simulate pericardial phenomena. The only circumstance in which a pleural effusion is at all likely to resemble one in the pericardium is when it happens to be peculiarly limited by previous adhesions. It has been stated that such conditions as pneumonia, phthisis, aneurysm, accumulation of fat, or intrathoracic tumour might be mistaken for acute pericarditis, but I have certainly never met with any difficulty of this kind. It must not be forgotten that this disease may be associated with other inflammatory affections within the chest, or be secondary to certain adjacent morbid conditions.

Prognosis.—Acute pericarditis must be regarded as a serious disease, though in uncomplicated cases the immediate prognosis is usually favourable. The mortality is comparatively small, but it is not practicable to give any definite percentage of deaths. Much depends upon the conditions with which the disease is associated, rheumatic cases being seldom immediately fatal, except in early life. It is far more dangerous when it supervenes in connexion with Bright's disease or other grave chronic maladies, and is then extremely likely to end fatally. Septic cases of all kinds are also very grave. Seeing that pericarditis and endocarditis so often go together, the prognosis in such circumstances must be guided by a due consideration of the effects of the combination in each particular case ; but obviously it must always be more serious, especially if the myocardium is also involved. When there are, in addition, other acute inflammatory affections within the chest, the danger is very imminent.

Among the factors influencing the immediate prognosis in individual cases the following are worthy of note :—Pericarditis is very serious in infants and young children, and the mortality is very high. Many are of opinion, however, that the carditis and acute dilatation of the heart are the actual causes of death in these subjects. The danger is also decidedly greater in advanced age. Previously impaired health, or a weak condition of the patient, and particularly the presence of old heart trouble or other chronic diseases, especially intrathoracic, may further complicate matters. The character and amount of the morbid products in acute pericarditis greatly affect the prognosis. The danger is obviously more serious in proportion to the quantity of fluid effusion ; as well as if there be reason to believe this to be of a haemorrhagic, purulent, or ichorous nature. Due observation and study of the symptoms may afford important indications as to prognosis. Among those of more or less grave import are serious dyspnoea, especially if amounting to orthopnoea, with signs of ,

cyanosis or asphyxia ; greatly embarrassed or very feeble or irregular cardiac action, with corresponding pulse, and tendency to faintness or syncope ; hyperpyrexia ; dysphagia ; severe vomiting ; marked prostration ; and pronounced cerebral or other nervous disturbances. The general appearance of the patient, and the expression of the face and eyes, are often useful guides as to the immediate prognosis. It must never be forgotten that sudden death from syncope may happen in cases of large effusion into the pericardium, especially if the patient is made to sit up, or to change his posture for the purpose of physical examination. Finally, the mode of treatment materially influences the immediate prognosis in acute pericarditis. Undue activity may certainly do much mischief ; but, on the other hand, a dread of energetic measures, when circumstances demand them, may as certainly lead to a fatal result.

The remote prognosis in a case of acute pericarditis always demands special attention, though it is often impossible to give a positive opinion on this point until the course of events has been watched for some time. I believe that the general tendency is to take too favourable a view of the ultimate prognosis, and not adequately to recognise the importance of the after-effects of the inflammatory changes. Such after-effects are frequently met with, and may be very serious, as will be pointed out in relation to pericardial adhesions. In cases of ordinary pericarditis they are more likely to give trouble in proportion to the amount of lymph effused ; to its presence over the exterior as well as the interior of the pericardium ; and to the slow or subacute progress of the disease.

Treatment.—The treatment of each individual case of acute pericarditis demands careful and intelligent consideration, and it is decidedly a mistake to follow any regular routine plan, or to adopt needlessly active measures. When it occurs in connexion with rheumatism it may not be requisite or desirable to change the previous treatment in any way, but much will depend upon the nature and degree of the morbid changes which the pericardial inflammation produces. The administration of salicylates is not contra-indicated, and it has been claimed for these agents that they help in averting the complaint or preventing large effusions ; but certainly their use requires caution. Dr. Lees, however, has great confidence in sodium salicylate, in adequate doses, with sodium bicarbonate, and states that children bear the drug well. Whether it be possible to prevent the development of pericarditis in rheumatic cases is a doubtful question, but at any rate complete rest, avoidance of chill, and due protection of the precordial region may help in this direction. Should there be a tendency to much cardiac excitement, I believe it is a good plan to administer opium or morphine as a preventive measure in suitable cases, the effects being of course duly watched.

When acute pericarditis has actually developed, the treatment must be guided by circumstances. In every case the patient must be kept as much as possible at rest, and must not be unduly disturbed or moved for the purpose of physical examination. Posture must be intelligently studied in relation to the pericardial conditions, the symptoms, and the

feelings of the patient.   As fluid accumulates it is often necessary to have
the head and shoulders raised ; but, if so, the patient should be propped
up comfortably, and effectually supported.   The judicious administration
of nourishment constitutes an important part of the treatment in many
instances.   Alcoholic stimulants, especially brandy and champagne, are
often needed ; the quantity must be determined by the requirements of
each individual case, as judged chiefly by the degree of general weakness
or depression, and the cardiac action and pulse.   In bad cases a consider-
able amount may be called for.

The treatment of acute pericarditis in the early stage has for its
objects the relief of pain and restlessness, the calming of the heart's action,
and the arrest or control of the inflammatory process.   The practice of
indiscriminate bleeding and administering calomel, formerly adopted by
many as a matter of routine, need only be mentioned to be absolutely
condemned ; nor in my opinion can anything favourable be said for the
use of cardiac depressants, such as antimony, aconite, and the like.
In suitable cases advantage may be derived sometimes from the appli-
cation of a few leeches, or even a small venesection.   As a rule, however,
efficient poulticing over the front of the chest gives most relief at first,
cotton-wool being afterwards applied.   Fomentations or spongiopiline
are also convenient applications.   I have thought that the application of
a blister over this region at an early period has occasionally checked the
progress of the inflammation, but it is easy to be deceived in this matter.
The application of cold, especially by means of ice-bags over the pre-
cordia, is strongly advocated by Dr. D. B. Lees and others, but this
treatment certainly requires caution ; and the feelings of the patient must
be taken into account.   Dr. Lees regards as necessary precautions to keep
the patient quite warm, by means of hot bottles before and while the ice-
bag is applied, and to prevent the right ventricle from being distended.
Further details may be obtained from his writings on the subject.
Should the pain be severe, opium may be given, Dover's powder being
a useful preparation ; or it may become necessary to administer morphine
subcutaneously, and repeat it as occasion demands.   There is no harm in
judiciously applying anodynes, such as belladonna, over the precordial
region ; but I doubt whether they have really any beneficial effect.

The treatment of pericardial effusion must be guided by its quantity
and mode of progress.   If it is not abundant, and shews the natural
tendency to become absorbed quickly, no special measures are needed.
Otherwise it might be desirable in rheumatic cases to apply a blister, or
even two or more in succession.   Some prefer applications of liniment of
iodine as counter-irritants ; others advocate the inunction of mercurial
ointment or oleate of mercury ; but personally I object to these measures
as being either useless or injurious.   The internal administration of
iodide of potassium or sodium may be of service, combined with tincture
of digitalis.   Iron preparations may also be helpful, especially the tincture
of the perchloride ; and a combination of tartrate of iron with the iodide
has been recommended.   Very active measures to promote absorption are

certainly to be deprecated ; and when the effusion is large, special care must be taken not to make the patient sit up suddenly, lest fatal syncope should ensue.

In all cases of acute pericarditis it is necessary to watch carefully the action of the heart and the pulse from the point of view of treatment. In my opinion, at no time is it desirable to give cardiac depressants. Some authorities recommend the administration of tincture of digitalis from the outset, but I do not think that a routine use even of this drug is desirable. However, should there be any indication of cardiac weakness, or a marked fall of arterial blood-pressure, with dicrotism of the pulse, the tincture should be given every three or four hours in 10-minim doses, its effects being duly watched. Strychnine affords valuable help in bad cases, and may be combined with digitalis ; or it may even be thought desirable to employ hypodermic injections of strychnine and digitalin. Strophanthus and other cardiac tonics have also been recommended in pericarditis. As temporary stimulants, ammonia and ether might be of decided service in some cases ; or possibly subcutaneous injection of ether might be urgently called for. Of course alcoholic stimulants are often of the greatest assistance, and large quantities of champagne or brandy may be demanded. The administration of the agents mentioned in the preceding remarks needs the most careful supervision, and they must not be employed indiscriminately or rashly, for it may be desirable at any time to diminish the dose, or to stop them altogether. Special care must be taken in the treatment of children.

Pericarditis not of rheumatic origin must always be treated as a part of the condition with which it may be associated, such as septicaemia, tuberculosis, or renal disease ; in the last-mentioned condition blisters and opium are contra-indicated. When it is accompanied with endocarditis, or with other intrathoracic inflammatory affections, the knowledge, experience, and judgment of the practitioner will often be severely taxed. Much difficulty may also be experienced in the treatment of symptoms, which must be conducted on ordinary principles, though considerable discretion and caution are demanded in carrying them out. Among the most important symptoms which may need attention are dyspnoea, especially if accompanied with a tendency to cyanosis or apnoea ; dysphagia ; severe vomiting ; restlessness and sleeplessness ; delirium or other cerebral disturbances ; motor disorders ; and high fever. Should the right auricle become distended, removal of blood is indicated, either by leeches or venesection. G. W. Balfour recommended chloral hydrate as a sedative and antiphlogistic along with digitalis ; it is, however, a depressant of the heart, and must at any rate be cautiously used. Want of sleep is a very trying symptom, but such remedies as sulphonal, trional, veronal, or paraldehyde in suitable cases may afford valuable help. Hypodermic injection of morphine may be imperatively demanded in spite of all risks. Dr Cheadle speaks highly of nepenthe for children. Inhalation of oxygen may be helpful in some cases. The measures to be

adopted to bring down temperature, especially hyperpyrexia, must be determined by circumstances. Difficulty in swallowing may, perhaps, be diminished by making the patient bend forward, so as to relieve the oesophagus from the pressure of the distended pericardium; but special care must be exercised in adopting this posture. The bowels need due regulation; and in bad cases it is important to see that the bladder is properly emptied.

The quantity of a serous effusion, and the imminent danger to life resulting therefrom in exceptional cases, as indicated by intense dyspnoea and grave signs of cardiac embarrassment, may raise the question of surgical interference, but I cannot agree with those who are too ready to resort to paracentesis for ordinary pericardial effusion, at any rate in cases of rheumatic pericarditis. This measure has also been recommended when the effusion shews no tendency to become absorbed. Sir Clifford Allbutt was the first to introduce as a practice the operation of paracentesis pericardii into this country in 1866, when it was successfully performed on a moribund patient of his by the late Mr. Wheelhouse. In 1883 Dr. S. West gave a summary of eighty cases thus treated. Since then it has attracted much attention, and numerous cases have been recorded. For a fuller discussion of the question of operative interference for pericardial effusion, and the methods employed, reference must be made to *Surgery of the Chest* (Stephen Paget) or other surgical works, and it will only be necessary to refer here to the more prominent practical points. As a dilated heart has on several occasions been perforated for a supposed pericardial effusion, it seems desirable always to make an exploratory puncture with a very fine needle in the first instance, in order to determine whether fluid is really present in the pericardial sac. It has even been advised to make a preliminary incision down to the pericardium. The fluid is generally removed by a small aspirator or glass syringe fitted with a needle; but some operators are in favour of using a trocar and cannula. It must be taken away very gradually, the effects being closely watched; and as much as possible should be removed. Of course strict antiseptic precautions must be taken. The spot usually chosen for the paracentesis is the fourth or fifth left interspace exactly at the border of the sternum, so as to keep clear of the internal mammary artery; and it has been recommended to keep close to the upper edge of the cartilage. Some prefer the opening to be made farther out, two inches or more from the sternum, but then there is a probability of wounding the pleura. Other spots favoured by individual operators are the sixth intercostal space, in or a little outside the vertical mammary line; below in the costo-xiphoid space; and the fourth or fifth space to the right of the sternum. The exact site to be selected may be influenced by the results of physical examination; and if fluid cannot be obtained in one spot, it may be necessary to try another. The patient should be in the recumbent posture, propped up with pillows; but it may be requisite to change the position if there is no result, and a case has been reported in which no fluid came away when the patient was

lying down, but 10½ ounces were withdrawn in the sitting posture. When performing paracentesis pericardii every available means of stimulating the heart should be at hand, ready for immediate use in case of threatening cardiac failure. Whether a repetition of the operation is called for must depend upon the course of events. It will suffice to mention that open incision of the pericardium has been advocated even in cases of serous effusion, as offering less risk and a speedier cure than aspiration; and also the method of reaching the sac by trephining through the sternum. When pericardial is associated with pleural effusion, the removal of the latter may sufficiently relieve all urgent symptoms, at least temporarily, but it may afterwards become necessary to operate on the pericardium.

The management of cases of pericarditis during convalescence is a matter requiring due consideration, especially in relation to the formation of adhesions. Personally I have always been disposed to enforce prolonged rest, and such is the general view. Some years ago, however, Mr. Cantlie suggested the desirability of encouraging exercise after an attack of acute pericarditis in young subjects, with the view of exciting the cardiac action, and thus helping to make the adhesions loose and filamentous. This question has usually to be considered in relation to the presence or absence of endocarditis and its consequences, as well as the state of the myocardium; so that no general rule can be laid down, and every case must be studied on its own merits.

II. Suppurative Pericarditis; Pyopericardium

The formation of pus within the pericardium has already been mentioned under acute pericarditis, but it will be expedient briefly to consider this condition separately, including also those cases in which the fluid is of an ichorous nature.

Etiology and Pathology.—Pyopericardium is occasionally acute in its manifestation, but is much more commonly the result of a subacute or chronic process. In very rare instances it is the outcome of an ordinary acute pericarditis, being then a late or secondary phenomenon, a serous or sero-fibrinous effusion gradually changing into a more or less purulent collection. In the large majority of cases, however, the circumstances in which such a collection is met with are peculiar, and it may not only be formed within the pericardium, but in some instances is partly due to the bursting of a neighbouring accumulation of pus into the sac. Pathologically it is associated, of course, with pyogenetic organisms of various kinds. The longer a pericardial effusion remains unabsorbed the more likely is it to become purulent.

Pyopericardium occurs most frequently in cases of pyaemia or septicaemia of all kinds; in this way it may appear as a complication of certain of the eruptive fevers. It has been said to be associated particularly with injuries and diseases of bones, such as osteomyelitis and acute necrosis.

Purulent pericarditis is more likely to supervene if an abscess has previously formed in the myocardium, but this is by no means necessary. Very rarely it has been secondary to infective endocarditis. In another class of cases pyopericardium is due to the rupture of a neighbouring collection of pus, especially of an empyema, into the sac ; or it may even be set up by infected air, which has entered through a perforation. Exceptionally it results from the extension of empyema, low forms of pleuro-pneumonia, neighbouring ulcerative or gangrenous diseases or abscesses, or possibly peritonitis. The pericarditis associated with Bright's disease is believed to have a special tendency to the formation of pus ; and a similar tendency has been attributed to the tuberculous variety. Among the cases of operation collected by Dr. Samuel West (95), however, in no instance of tuberculous pericarditis was the effusion purulent. Such a condition may be associated with pulmonary tuberculosis, owing to the rupture of a cavity into the sac. Pyopericardium is far more common in young subjects ; and in males. Purulent pericarditis in childhood, probably usually pneumococcal, being specially associated with empyema, pleurisy, or pneumonia, is seldom seen combined with endocarditis, thus contrasting with rheumatic pericarditis. It is difficult to recognise during life (Coutts) ; of 100 fatal cases it was detected with certainty in 6 only (Poynton).

Anatomical Characters.—As the name indicates, the essential change in pyopericardium is the presence of pus in the sac. It may be in small amount, or the accumulation may be considerable—as much as from 14 to 20 oz. or more ; in the latter case it will produce the same mechanical effects upon the heart and neighbouring structures as other forms of effusion. It may collect entirely in the posterior portion of the pericardium, the anterior surfaces being adherent. The pus is usually sweet and inodorous, but may be shreddy, flocculent, curdy, or be mixed with fibrin. Exceptionally and in particular circumstances it is offensive, and may be of an "ichorous" nature, or very foul. It may also become fetid after operation. Occasionally there is an admixture of blood. In most cases the surface of the membrane becomes like that of the granulating surface of a wound. Rarely part of the parietal pericardium becomes destroyed, and perforation takes place, which has even terminated in a superficial fistula ; but at the present day such a termination could hardly be permitted to occur. There seems to be good reason to believe that a purulent collection in the pericardium may in exceptional instances be absorbed, leaving dense and thick adhesions ; or some of it may remain in an inspissated condition as a yellowish-white paste, limited and encapsuled by adhesions, consisting of caseous material, in which calcareous particles may afterwards form ; thus it may ultimately be converted into a chalky pulp, or even into a hard calcified mass.

Clinical History, Diagnosis, and Prognosis.—Speaking generally, the symptoms and physical signs of pyopericardium will be more or less like those of serous effusion, modified not only by the quantity of the pus, but also by the circumstances in which it has formed. It will only

be necessary, therefore, to draw attention to certain special points in the clinical history of this condition. When it supervenes in an ordinary case of acute pericarditis, there are no trustworthy indications of a change from a serous or sero-fibrinous effusion to one of a purulent nature; but if the course of the case happens to be prolonged, such a deterioration would be suggested should fever, perhaps of a septic type, persist. Pyrexia may, however, be entirely absent. Considering the circumstances in which pyopericardium occurs, it is easy to understand how insidiously it may set in; its symptoms, if any, being entirely overshadowed by those of septicaemia. Hence it often remains undiscovered until the necropsy, especially if the amount of pus be small. In cases of this kind symptoms of serious interference with the respiratory and circulatory functions may appear suddenly; being found on examination to be due to a large but previously latent purulent collection in the pericardium. From an analysis of 100 fatal cases in childhood, Dr. Poynton found that there were three groups—the acute running a course of about a month, the subacute lasting from one to six months and including the majority of the cases, and the chronic cases with insidious onset lasting six to twelve months. General symptoms are of little or no value in the diagnosis of pyopericardium. In some of the most pronounced cases neither rigors, pyrexia, nor sweating have been present. Oedema of the legs seems to be not uncommon, but probably is not more frequent than in connexion with other large pericardial effusions and their consequences. It may be noted here that oedema over the precordial region may suggest the purulent nature of such an effusion.

With regard to the *physical signs*, the absence of friction-sound throughout in cases of purulent pericarditis has been noted by careful observers; or it may be very indefinite and transient. In 100 fatal cases in childhood collected by Dr. Poynton, friction was detected in two only. At any rate this sign cannot be relied upon in diagnosis. The ordinary signs indicative of pericardial effusion will be evident on examination, in proportion to the amount of the pus. Should gas be present at the same time, the phenomena associated with this combination will probably be noted (*vide* p. 98).

From the foregoing remarks it will be gathered that the *diagnosis* of pyopericardium is extremely uncertain, and often impossible. Should there be evidence of effusion into the sac, its purulent nature can only be determined positively by the aid of the exploring needle or other suitable apparatus, by which a specimen can be obtained for examination. Some such instrument should be used without delay if there be any reason to suspect the presence of pus. The frequency with which purulent pericarditis is not diagnosed in childhood has already been referred to.

The *prognosis* of pyopericardium is necessarily grave, especially on account of the conditions with which it is associated. In suitable cases, however, efficient operative interference gives reasonable hope of recovery; and some remarkable results have been thus achieved by modern surgery.

Treatment.—The treatment of pyopericardium is entirely surgical, and it would be quite beyond the province of this article to attempt to discuss the important questions involved. Suffice it to say that, according to almost universal experience, paracentesis is of no use; and the operative procedures adopted must be thorough and bold, and should be carried out as promptly as possible. Pericardiotomy is performed; and in order to expose the pericardium adequately it is necessary to resect one or more of the left costal cartilages, from the fourth to the sixth, or even part of the ribs. The sac is then freely but carefully incised, the contents evacuated, and a small soft drainage-tube inserted at the lowest part of the cavity. Strict antiseptic measures must be carried out. In exceptional cases a pyopericardium has been evacuated and drained from behind. It has also been urged that the pericardium should be opened from below through the diaphragm; this gives better drainage, enables the front and back of the heart to be explored, and does away with any risk of opening the pleura (Ogle and Allingham); this operation has been successfully performed (Pendlebury).

III. Chronic Pericarditis ; Chronic Effusion ; Pericardial Adhesions and Thickening

The cases which come within the category of chronic pericarditis may be arranged for practical purposes under two main primary groups, namely, those of—(1) Chronic effusion; (2) Pericardial adhesions and thickening. These conditions are in exceptional instances more or less combined, but it is needless to make an independent class of such complex cases.

1. Chronic Pericardial Effusion.—It occasionally happens that acute or subacute inflammatory effusion into the pericardium remains chronic, though fluctuating in amount; or it may return again and again after paracentesis. In rare instances even a simple pericarditis is chronic from the outset; but this course of events is observed chiefly in elderly persons, and there is reason to believe that in some of these cases the effusion is originally a mere hydropericardium. Chronic pericarditis is more likely to be of a haemorrhagic or purulent nature; or it may be associated with tuberculosis or new growths, especially malignant disease. Dr. Samuel West has recorded a remarkable case of supposed mediastinal cyst, which was tapped several times during a period of four years; on post-mortem examination it proved to be a chronic pericardial effusion. In very exceptional instances an accumulation of this nature originates a diverticulum of the pericardium.

Clinically, chronic pericardial effusion does not, as a rule, give rise to any prominent symptoms; practically it is only recognisable by the physical signs already described. In prolonged cases, owing to the changes produced in the pericardium and the walls of the heart, the

circulation becomes more or less seriously obstructed, with the usual symptoms, including dropsy.

The treatment of this condition must be conducted on the general principles applicable to different kinds of pericardial effusion, some operative procedure being generally required ; but each case must be dealt with on its own merits.

2. PERICARDIAL ADHESIONS AND THICKENING.—The conditions coming under this head are so common and often of such pathological and clinical importance that to make light of them, as some writers have done, is a serious mistake. They are frequently met with in various degrees at necropsies, when they have not been diagnosed during life, and it must be acknowledged at the outset that their diagnosis is often, for obvious reasons, impracticable, or may be a matter of great difficulty or mere surmise ; not uncommonly, indeed, there is no reason whatever even to suspect their presence. On the other hand, to teach that the diagnosis of adherent pericardium is impossible is, in my opinion, absolutely wrong and misleading. If pericardial changes of this nature were always borne in mind and systematically looked for, they would be recognised much more frequently than is even now the case. As a matter of fact, experience has taught me that they are seldom even suspected in the ordinary routine of practice, and are therefore necessarily overlooked. Not uncommonly they can be positively demonstrated by physical examination ; whilst in other cases their presence may be reasonably inferred. Sir John Broadbent, in his valuable monograph on " Adherent Pericardium," duly recognises this truth, and draws special attention to the liability to overlook this condition when it is associated with valvular disease.

Etiology.—The various conditions of the pericardium now under discussion are always of inflammatory origin, and in the large majority of cases they are the remains of one or more acute or subacute attacks of pericarditis, especially rheumatic, of which there is often, but not necessarily, a definite history. As was mentioned in relation to this disease, extensive adhesions may rapidly form in the stage of fibrinous exudation, particularly in children ; and if the termination be not fatal, they become organised and permanent. Most commonly, however, they are formed after the absorption or removal of fluid effusion. As might be anticipated, adhesions are likely to be more firm and extensive in proportion to the number of attacks of pericarditis, and to their duration. After a first attack partial adhesions may form, which after recurrent and repeated attacks become extensive or general. When a pericarditis beginning acutely assumes a prolonged and chronic course, they are usually well-marked ; as well as when the effusion becomes purulent. The occurrence of acute inflammation over the external surface of the pericardium leads to the formation of adhesions between this structure and the chest-wall, the pleurae, and sometimes the posterior mediastinal structures or the spinal column.

An important group of cases in which pericardial adhesions and thickening occur are those which are chronic from the outset, and in these cases they are particularly liable to be overlooked. They may naturally be expected when an inflammatory effusion runs a chronic course throughout; but the cases which must be more especially borne in mind are those in which there has been no such effusion, but the morbid changes leading to the pericardial conditions have taken place slowly and imperceptibly. Some of the " white patches " are of this nature; but the most striking cases are those in which a chronic inflammatory process extends from neighbouring structures, particularly in connexion with pleurisy or pulmonary tuberculosis. Adhesions are also usually associated with new growths invading the pericardium. Chronic pericardial changes may be present in a case belonging to the group known as *polyserositis* or *polyorromenitis*, in which the peritoneum and pleurae are also affected. When the changes leading to adhesions have once started, it seems highly probable that they may extend and increase considerably, as the result of a continued chronic process, which leads to a progressive hyperplasia of fibrous tissue. In this way it may possibly happen that an adhesion, as it were, grows through the parietal portion of the pericardium from within outwards or from without inwards, and thus ultimately fixes it more or less extensively on both aspects.

Pericardial adhesions may be met with at all ages. They have been observed in very young infants, and even in new-born children, being then attributed to pericarditis occurring during fetal life. Dr. T. Fisher has well stated that "adherent pericardium is the most serious form of cardiac disease in children," but the influence of age will be more conveniently considered in relation to prognosis (p. 92).

Anatomical Characters and Effects.—It would not serve any useful purpose to describe in detail the numerous and varied aspects under which pericardial adhesions present themselves, but a comprehensive knowledge of the more important groups of cases in which changes of this kind are met with is of decided practical advantage. Before attempting any such classification it will be well to point out that the adhesions are either partial or general; internal or external, or both; differ much in length, toughness, and firmness; and are often accompanied by more or less pericardial thickening, which may reach an extreme degree. In exceptional instances there is much thickening, with little or no adhesion between the surfaces. Structurally the morbid formations now under consideration consist either of cellular or fibrous tissue—*pericardial fibrosis*. Sometimes they are associated with the encapsuled remains of fluid, thickened pus, soft caseous or chalky pulp, or dry, brittle, calcareous concretions, which may attain a considerable size. As already stated, an adherent pericardium may undergo calcification.

The groups under which I propose to arrange the cases, as they have come under my personal observation, are as follows :—

(*a*) In a large proportion of instances there are merely partial and small adhesions between the contiguous surfaces of the pericardium, it

may be involving different portions of the sac at the same time. Usually such adhesions assume the form of filaments or threads, or of bands, often of considerable length, stretching between the two surfaces. They may be delicate and cellular, or firm and fibrous, sometimes attaining the thickness of a finger or more. Occasionally adhesions occur in circumscribed closely adherent spots or patches. Ultimately the bands often give way by stretching and attenuation, their remains hanging loosely within the sac, especially near the apex of the heart. The situation, extent, and characters of localised pericardial adhesions are affected by the degree and range of the movements of different parts of the heart and arteries ; the relation of the heart to the pericardium ; and the position the organ assumes within the sac in cases of effusion. According to Sibson, they are more frequent a little above and to the left of the apex, and along the line of the ventricular septum ; at the outer border of the left ventricle, and the outer side of the right auricle ; along the posterior surface of the left auricle and of the ventricles which rest upon the sac ; and over the great arteries at their higher part.

(*b*) A second group of cases may be made to include those in which an extensive or general internal adhesion exists between the pericardial surfaces, the external surface being quite free ; and this group may be subdivided into cases without and with thickening. Here again many varieties are observed in individual instances, and in the same case the adhesions often differ in their characters over different parts of the pericardium. They may be in the form of fibrous threads or bands, more or less loose and long, and interfering but little with the free play of the heart ; or of short, close, firm, and strong attachments. In some cases the contiguous surfaces of the pericardium are agglutinated together throughout, the sac being entirely obliterated ; and when this condition is of old standing, separation of the two surfaces is impossible without tearing the heart substance. Occasionally, when comparatively recent, they may with care be drawn asunder ; or firm adhesions of old standing may exist side by side with those of recent origin, the result of a fatal intercurrent acute pericarditis, which can be easily broken down. The degree of thickening differs a good deal, but it may be very remarkable, as much as a quarter to half an inch or more ; it chiefly affects the visceral layer. The heart is then enclosed in a dense, strong, tight envelope or casing, which compresses and strangles the organ in its grip.

(*c*) There is a distinct class of cases in which the adhesions are entirely external or *exo-pericardial*, the outer surface of the pericardium being more or less extensively fixed to the front of the chest, and often to the pleurae, while the internal surfaces are quite free. They are usually chronic in their course, and secondary to neighbouring morbid conditions, being especially met with in association with very chronic pulmonary tuberculosis. These exo-pericardial adhesions may, however, extend from similar pleuritic changes ; or may possibly result from a mediastinitis occurring at the same time as the attack of pleurisy which led to the pleural lesions. The condition now under consideration is

really mediastinal, and has been named *chronic mediastinitis* (Vol. V. p. 612).

(*d*) In the most serious group of these cases the pericardial adhesions are both internal and external, there being a general matting of the sac to the heart, as well as to the chest-wall in front, to the adjacent pleurae, especially the left, to the diaphragm more extensively than in health, and occasionally to the structures in the posterior mediastinum and the spinal column. As a rule these conditions are accompanied with much fibrous thickening. When there is little or no general mediastinitis the term *pericarditis externa et interna* is applied; when there is a considerable increase of fibrous tissue in the mediastinum the condition is known as *indurative mediastino-pericarditis* (*vide* art. Vol. V. p. 612). The external adhesions vary considerably in area, but in extreme cases may extend from the second cartilage to the sixth; from the manubrium to the upper half of the ensiform cartilage; and from the right border of the sternum to the apex of the heart to the left of the nipple-line (Sibson).

(*e*) Exceptional instances are met with, of which I have seen striking examples, in which the prominent change is marked thickening of the pericardium, especially of its visceral portion, with little or no adhesion of the surfaces; and there may even be more or less fluid incarcerated between them. It is important to bear this variety in mind, for it may produce very serious effects upon the heart, with the consequent symptoms, without giving rise to any of the physical signs of pericardial adhesion.

Effects upon the Heart and Great Vessels.—There has been much controversy as to the effects of pericardial adhesions upon the heart; but they vary much, of course, in different circumstances. In a considerable proportion of cases the organ is unaffected, either functionally or structurally, and, provided it be free from valvular disease, remains of its normal size. The obvious tendency is to embarrass its action more or less; the embarrassment is greater in proportion to the extent and firmness of the adhesions, and greatest when they are both internal and external.

One of the most important structural changes affecting the heart which may be associated with adherent pericardium as the sole lesion is enlargement of the organ. The frequency with which this change occurs cannot be definitely stated, and different percentages have been given by different observers. Hope maintained that this morbid condition always gave rise to compensatory cardiac hypertrophy; but numerous observations have amply shewn that such a statement is not correct: even complete obliteration of the sac is not necessarily followed by enlargement. In 90 cases of uncomplicated adherent pericardium Dr. Kennedy found cardiac hypertrophy in 56 per cent, whereas Gairdner met with it in 36 per cent only. Sibson stated that the change occurs in about two-thirds of the cases; probably it supervenes in some degree in more than half. It is, however, when adhesion follows rheumatic pericarditis that hypertrophy of the heart chiefly occurs. Tuberculous pericarditis and pericarditis associated with pneumonia or septicaemia are rarely followed

by enlargement of the heart (Fisher). It is especially met with in children, in whom the organ may be as much as five times its normal
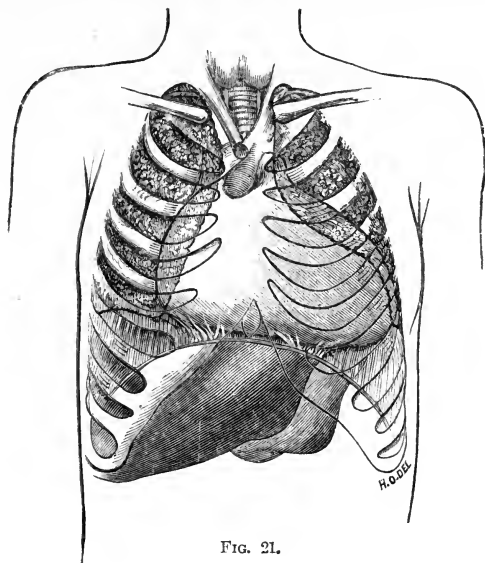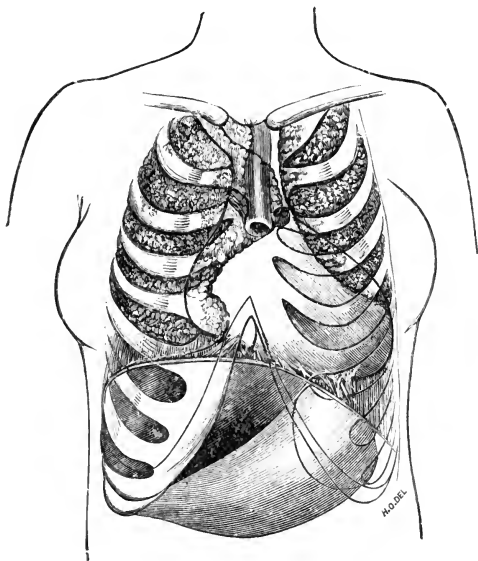

Fig. 21.

Fig. 22.

Figures shewing position of internal organs in cases of adherent pericardium. (Sibson.)

weight. Hypertrophy exceptionally develops with great rapidity, as in cases observed by Dr. Goodhart and Dr. Sequeira. Dr. Samuel West

believes that the cases in which the heart suffers are those in which
adhesions exist between the external surface of the pericardium and the
chest-walls, and no doubt these tend to make matters worse.   When
adherent pericardium is associated with valvular disease, it has been
questioned whether this condition has anything to do with the enlarge-
ment which follows.   From personal observations I am decidedly of
opinion that a generally adherent pericardium, when complicating valvular
lesions, does often materially contribute to the cardiac increase which
they induce ; at any rate it promotes and hastens its development.

With regard to the mode in which adherent pericardium may bring
about cardiac enlargement, the explanation usually given and accepted
is that it is mainly by the additional work imposed upon the heart, by
the hampering of its movements and the " constant struggle," aided by
the accompanying changes in the myocardium.   It has also been suggested
that the eccentric contraction of cicatricial tissue may in some instances
bring about dilatation of the ventricles, especially when the structures are
fastened to the spinal column or anterior chest-wall, but this is very
doubtful.   No doubt in not a few cases the dilatation of the heart
occurring during an attack of acute pericarditis, followed by rapid adhe-
sion, is the starting-point of cardiac enlargement, the organ being thus
prevented from returning to its normal size, more or less hypertrophy
subsequently supervening.   In accordance with his views, Dr. Sequeira
attaches most importance to the dilatation of the pericardium as the
primary cause of the cardiac changes.   Dr. T. Fisher is not satisfied with
these explanations, and suggests that " possibly the enlargement which
follows rheumatic pericarditis may be due to some deleterious influence
exerted by rheumatic toxins upon the nutrition of the growing heart."

As regards the nature, extent, and degree of the cardiac enlargement,
considerable differences are observed in different cases of simple peri-
cardial adhesion.   As a rule there is a combination of hypertrophy and
dilatation, the latter commonly preponderating ; and it may exist practi-
cally alone.   Both sides of the organ are usually involved more or less ;
but I fully accept Sir John Broadbent's statement that pericardial adhe-
sions in themselves are much more likely to affect seriously the right
ventricle than the left.   The auricles are much less affected ; indeed it
may happen that, while the right ventricle is much enlarged, the auricle
is compressed and may even be practically obliterated.   The left auricular
appendix has been found in a similar condition.   When the enlargement
of the heart is associated with valvular disease, it will necessarily be
influenced chiefly by the nature of such disease, but in particular instances
it may certainly be modified by the adhesions.   As a result of dilatation
associated with adherent pericardium, and involving the orifices, valvular
incompetence is prone to follow, especially at the tricuspid opening, which
may become greatly enlarged.

In exceptional cases the effects of pericardial adhesions upon the heart
are quite the opposite to those just considered.   In children the natural
growth and development of the organ may be prevented ; or it becomes

small and atrophied, its walls being grasped and compressed, and its cavities forcibly contracted in size by the dense, thick, tight envelope surrounding them. This may happen also from mere thickening of the visceral pericardium, without any adhesion. Dr. Kennedy found atrophy of the heart in 5 of his cases of adherent pericardium without valvular disease. It appears to occur only in wasting disease (Sequeira). Other important cardiac lesions which may be associated with pericardial adhesions are fibrosis, which may be well marked, and fatty or pigmentary change. They may result from direct pressure, or pressure on the coronary vessels ; or fibrosis may be due to a chronic interstitial myocarditis spreading from the pericardium. In not a few instances, no doubt, these myocardial changes are the outcome of myocarditis associated with an acute attack of pericarditis. It may be mentioned here that some have held that the hypertrophy of the heart following adherent pericardium is not true hypertrophy, but a thickening of the wall by changes mostly fibroid, secondary to interstitial myocarditis. Careful observations, however, have shewn that there is no foundation for this view.

When the pericardium is fixed externally, the great vessels at the base of the heart are often abnormally exposed ; and the arteries may be lifted up into an unusually high position. Occasionally one or both are compressed or constricted by pericardial adhesions ; or their walls undergo degenerative or fibroid changes. As the result of obstruction to the general venous circulation, produced indirectly by adherent or thickened pericardium, the large veins become more or less dilated, and such dilatation may ultimately be extreme. Moreover, notwithstanding statements to the contrary, contracting bands of organised exudation may in exceptional cases compress seriously the superior or inferior vena cava. The late Sir William Broadbent and Dr. Samuel West have brought forward instances in which the inferior vena cava had been completely occluded in this way. Adherent pericardium and its associated conditions tend to produce secondary changes in other organs and structures, mainly in consequence of interference with the circulation, but these will be sufficiently referred to later on.

Clinical History.—It is obviously impossible to give any definite clinical description that will apply even to the majority of cases of adherent pericardium ; all I can do will be to point out the symptoms and physical signs which may be associated with this condition, as well as the relations of these groups of phenomena to each other, upon which a diagnosis may reasonably be founded. They vary considerably in individual instances, depending not only upon the actual nature and degree of the changes affecting the pericardium, but also on the state of the heart, and their association with valvular affections, with vascular lesions, or with neighbouring morbid conditions.

As was stated in the introduction to this subject, a large number of cases of pericardial adhesion do not exhibit any symptoms or physical signs whatever ; and, unless there happen to be a well-known history of

acute pericarditis, the condition cannot even be suspected during life. This applies not only to partial and loose adhesions, which often do not disturb the heart in any way, but even to cases in which there is general agglutination of the internal surfaces, provided the organ itself be not materially damaged. Sudden death has occurred in my experience, as the immediate result of pericardial adhesion not detected or even suspected during life. It is well to bear in mind the possibility of the existence of this condition, if with acute pulmonary inflammatory affections the heart should exhibit signs of embarrassment quite out of proportion to their severity. My observations have led me to the conclusion that it may add seriously to the danger in a particular case in these circumstances, and even account for an unexpected death. The more pronounced the pericardial changes, the more prominent and definite are the clinical phenomena likely to be; and they are especially well marked when there is much thickening, and when the adhesions are both external and internal.

Symptoms.—Pericardial adhesions are undoubtedly not uncommonly the cause of pain, dragging, oppression and tightness, or other unpleasant sensations over the precordial region, and when in cases of chronic cardiac disease such sensations are a prominent feature, their existence may be reasonably suspected, and they should be carefully looked for; they are then often easily demonstrable on physical examination. The pain is sometimes felt over a very wide area beyond the limits of the precordia; and referred pains, associated with dilated heart, may radiate down the inner side of either arm, to the left shoulder, to the back, or even down towards the abdomen and loins. These referred pains are essentially transitory, and often last but a few hours (Sequeira). Cutaneous hyperaesthesia is often noticed; and, according to Dr. Head, both the pain and hyperaesthesia are essentially different from the sensations in acute pericarditis, being evoked by lightly picking up the skin, and diminished by firm pressure. Painful attacks of anginal character may possibly occur, and even present a grave aspect.

Adherent pericardium ought always to be thought of as a possible cause of *palpitation* or other cardiac disturbance, not obviously explained. The heart's action is in some instances irregular or unequal, and it may be so embarrassed as to lead to faintness or actual syncope. The persistence of rapid cardiac action, in spite of treatment, may be important evidence of the formation of pericardial adhesions in children and young persons. The patient is usually conscious of cardiac disorders associated with adherent pericardium, and is then likely to complain of palpitation, even at rest, but especially after exertion; this symptom is sometimes very pronounced; tachycardia has been noted in some cases. The pulse will present corresponding characters, but may also be modified in exceptional instances, as the result of direct interference with the aorta by pericardial adhesion and thickening.

Pericardial adhesions may themselves unquestionably cause *shortness of breath* on exertion, sometimes well marked; and a feeling of inability

to take a deep breath is sometimes a prominent symptom, especially when the external adhesions are extensive. Moreover, they often add materially to the dyspnoea associated with other cardiac affections; whilst their secondary effects, in the way of serous effusions, are likely to increase the difficulties of respiration, which may amount to orthopnoea. On the other hand, there may be an entire absence of dyspnoea or only rather hurried breathing, when other symptoms are very pronounced. No other respiratory symptoms can be definitely attributed to adherent pericardium alone; but when there is much thickening, with compression of the heart and changes in its walls, the pulmonary circulation is likely to be embarrassed, and cough, expectoration, or even haemoptysis may set in.

A very important and prominent group of symptoms in certain cases of pericardial adhesion are those indicating serious hampering or actual failure of the right ventricle, and consequent interference with the *general venous circulation*. In exceptional instances these may be aggravated by direct obstruction of one or both of the venae cavae. These phenomena as a rule develop gradually, becoming more and more pronounced; but occasionally they supervene with great rapidity, the ventricle appearing to break down and give way very speedily, or even suddenly. They occur not only in cases in which this cavity is obviously dilated, but also when the heart is strangled and compressed by dense fibrous thickening; and in such cases they may be extreme. No doubt they depend in great measure upon the associated changes in the myocardium. These symptoms are similar to those which arise in other forms of heart disease affecting the right side:—namely, general dropsy, involving the serous cavities as well as the subcutaneous tissue more or less extensively; overloading of the hepatic and portal system and its consequences; and congestion of the kidneys, nervous system, and other structures. The dropsy usually begins in the legs, but it may ultimately spread to the trunk, and even the arms. There is a remarkable class of cases met with exceptionally in which ascites precedes oedema of the legs, or may even exist alone, the abdomen becoming greatly distended, especially in children, the fluid returning after removal. This condition has been usually explained on mechanical grounds, and as being due to the direct influence of the pericardial adhesion upon the circulation; and such is the view of Wenckebach. Others, however, maintain that the ascites is due to chronic peritonitis, and Dr. T. Fisher has suggested that possibly a small patch of acute or subacute peritonitis, which has spread through the diaphragm from rheumatic pericarditis, paves the way for infection with micro-organisms, which produce later widespread chronic peritonitis and ascites. In this connexion it is interesting to note that Flesch and Schossberger performed experiments on dogs, producing adhesion of the parietal and visceral pericardium, and they found that the most striking effect was an isolated excessive ascites, which they attributed to pure portal stasis. There is another important group of cases in which adherent pericardium is accompanied with repeated effusion into the

peritoneal cavity and pleurae, the fluid having to be removed again and again to afford temporary relief. Probably these cases belong to the group of polyorromenitis (*vide* Vol. III. p. 948 and Vol. IV. Part I. p. 165). The appearance of the patient differs according to circumstances. Cyanosis with distended veins may be evident ; or there is a mitral flush on the cheeks, with enlarged venules. On the other hand, sometimes marked pallor is noted, with puffiness of the face. In prolonged cases the fingers and toes become clubbed. The liver becomes enlarged so that it can readily be felt below the ribs, and may be painful and tender. Occasionally it reaches even below the umbilicus, appearing to be very large ; but then it is usually displaced downwards as well. After a time the organ yields an abnormally firm sensation on palpation, and may become irregular ; in prolonged cases it may even pulsate. Symptoms connected with the alimentary canal are often prominent after a time ; and sickness may be troublesome. The spleen is sometimes perceptibly enlarged. The urine is more or less diminished in quantity, concentrated, and often albuminous. I have known the amount of albumin to be so large that the urine became almost solid on boiling. In bad cases the patient is very restless and sleepless ; and insomnia often gives much trouble, sleep being at last only possible in the propped-up posture. Terrifying dreams are not uncommon. Occasionally marked mental disturbance is noted, with hallucinations, especially of sight and hearing ; and nervous symptoms may be so pronounced in children as to simulate some cerebral trouble.

When acute pericarditis occurs early in life, growth and development are often much delayed and the patient presents a peculiarly youthful appearance. Marked wasting is a common symptom of cardiac disease in children, of which adherent pericardium is one of the most important conditions.

Physical Signs.—The existence of pericardial adhesions can often be recognised positively and demonstrated by careful and systematic physical examination ; and it is most desirable to have a clear and definite knowledge of the signs which, in different combinations, have to be looked for and studied in relation to this condition. At the same time it must be understood that they are frequently absent, or at any rate not at all characteristic ; and this may happen even when there are very pronounced symptoms directly due to an adherent pericardium. The physical signs are likely to be better marked in proportion to the extent and density of the adhesions, and especially when these are external as well as internal. They result not only from these lesions themselves, but also from their effects upon the heart and vessels, and upon the circulation. Cardiac enlargement is a most important factor contributing to the abnormal signs. They may be considered in the following order :—

(i.) *Change in Shape.*—In exceptional instances a distinct and permanent depression of more or less of the precordial region, with narrowing of the intercostal spaces, is observed ; the structures being drawn in by thick external adhesions. Far more commonly, however, there is

abnormal fulness or bulging, due to enlargement of the heart ; but this sign can hardly be regarded as an indication of adherent pericardium, except in particular circumstances.

(ii.) *Signs associated with Cardiac Movements.*—Certain visible and tactile signs coming under this head are of the utmost importance, and demand somewhat detailed consideration. Sometimes there are peculiarities in the cardiac movements which cannot well be described, but which are very suggestive of pericardial adhesions. The following are the more definite signs to be studied :—

(*a*) *Apex-beat.*—In cases of adherent pericardium, the ordinary so-called apex-beat presents many differences as regards its position, force, and characters ; but these depend mainly upon the effects of the particular valvular disease or diseases with which the condition happens to be associated. Thus it may be noticed far to the left, and presenting all the characters indicating a greatly hypertrophied left ventricle. One of the signs to be looked for is a displacement of the apex-beat, which is fixed in its abnormal position, and cannot be modified by any change of posture. As a rule it is carried somewhat outwards ; but the most suggestive displacement is elevation, it may be to the fourth space or even higher, while perhaps at the same time there may be marked evidence of hypertrophy. In many instances the apex-beat is very feeble, or even imperceptible, when other phenomena are prominent. This is attributed to small size and weak action of the heart, to restraint of the organ by adhesions, or to much thickening of the pericardium. When it is wholly due to feeble cardiac action, the beat may at times be perceptible, at other times not. There may, however, be a distinct impulse over the ensiform cartilage or in the epigastrium, associated with the right ventricle.

(*b*) *Impulse.*—Taking into account the entire impulse, it must be admitted that in different cases of adherent pericardium great variation is observed as regards its situation, extent, force, and characters ; but there are certain points deserving of attention. A remarkable extension of its area is often noticed, especially upwards over the precordial region ; and it may reach the second space or cartilage. At the same time the impulse is often strong and superficial, the heart pulsating in close contact with the chest-walls. In some instances the movement presents to the eye a decidedly undulatory, wave-like, or rippling character. In others it is peculiarly jarring, or has an abrupt jogging quality. The rhythm of the cardiac action is sometimes markedly disturbed, and irregularity may be a prominent feature in connexion with pericardial adhesions. In many cases it is obviously laboured. When the heart is at the same time enlarged, the extent of the impulse is correspondingly increased, often passing considerably beyond its normal limits, and probably tending more towards the right, in consequence of the greater enlargement of the right ventricle.

(*c*) *Systolic Recession or Retraction.*—A visible recession or retraction of certain parts of the chest-wall, associated with the ventricular systole,

has attracted much attention in relation to adherent pericardium. There can be no doubt that the signs coming under this head are of great importance in the diagnosis of this condition, and they deserve particular study in any suspected case. They come practically under three categories, namely :—

(a) Recession over the spot corresponding to the apex of the heart, occurring with or immediately after the systole. This phenomenon, when present, is usually associated with a definite apex-beat, but is sometimes noticed when there is no perceptible impulse at this point.

(β) Systolic depression of more or less of the precordial region, generally involving one or more of the intercostal spaces to the left of the sternum, especially the third, fourth, and fifth, along a variable extent of their length. The movement is sometimes distinctly wavy. In certain cases, in which the adhesions are extensive and strong, and the heart is acting powerfully, the cartilages are also involved, or indeed even the lower half of the sternum, the ensiform cartilage, and the epigastrium. When the recession occurs simultaneously with a pronounced apex-beat, the combination is very striking, but it may be indefinite or absent. Should the right ventricle be greatly enlarged, a similar movement may possibly be visible in the intercostal spaces to the right of and close to the sternum ; of this I believe I have seen examples. According to Friedreich, the recession is more marked at the height of inspiration.

(γ) Retraction of the posterior or lateral portions of the thoracic walls. This sign, when present, is regarded by Sir John Broadbent as most important in the diagnosis of adherent pericardium. He describes it in the following words :—" In cases of adherent pericardium, marked systolic retraction of some of the lower ribs on the lateral or posterior aspect of the thorax may sometimes be seen. This phenomenon is best seen when the patient is sitting up in a good light, and the movements of the chest are carefully observed from a short distance off, first from the front and then from the lateral aspect. When a pulsatile movement is seen over the lowest part of the left side posteriorly, it may at first sight appear to be expansile. On a more careful scrutiny it will be found that there is a tug on the false ribs during the cardiac systole, and a sharp rebound during diastole, which can be felt as well as seen when the hand is laid flat upon the chest-wall at the spot ; it is more marked when a deep inspiration is made ; it may be seen occasionally not only on the left side but also on the right, especially if the patient leans over to the left."

Space will not permit of any lengthy discussion of the association of the phenomena just indicated with conditions other than pericardial adhesions, or of their precise significance in any individual case of such adhesions. A few general observations on these points must suffice. Apical recession very rarely occurs except as the result of adherent pericardium, but it has been noticed in other circumstances. When it is associated with a definite beat, it probably indicates that the

heart is fixed at or near its apex to the chest-wall, and drags on it during the systole. When there is no palpable apex-beat, it is supposed that the heart is prevented by adhesion to the diaphragm or vertebral column from performing its normal forward and rotatory movement during systole ; or that the cardiac impulse is too feeble to be felt through the adhesion.

Skoda was of opinion that systolic recession of the intercostal spaces is pathognomonic of adherent pericardium, but numerous observations have proved that this is not the case, as the phenomenon may occur in cases of considerably enlarged heart, especially when associated with aortic regurgitation. Still it is an important sign of adhesion, and its presence should always have due weight in diagnosis. As a rule it indicates that the contiguous surfaces of the pericardium are adherent, and also that the sac is fixed in front to the chest-wall, and is firmly attached to some structure posteriorly, or, it is supposed, in some instances to the diaphragm ; according to Friedreich the latter is essential. I have met with this phenomenon in a pronounced form in cases of external peri-cardial adhesion with enlarged heart, in which the internal surfaces of the sac were quite free. As a result of diminution in the force of the cardiac action, systolic retraction may in course of time become less and less evident, and finally disappear.

The systolic retraction of the posterior or lateral portions of the thoracic walls is explained by Sir John Broadbent in the following way : —" The heart is, by means of the pericardium, adherent not only to the central tendon of the diaphragm, but probably also to a large area of the fleshy or muscular portion of the diaphragm, and, it may be, to the anterior thoracic wall as well ; as it contracts it drags upwards and inwards the less resistant fleshy part of the diaphragm towards the central tendon or anterior chest-wall ; hence the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and downwards. It will always be found in such cases that the retracted portions of the chest-wall correspond to the floating ribs or costal cartilages of the lower ribs at the points of attachment of the diaphragm."

(*d*) *Diastolic Shock or Concussion.*—This is a very exceptional sign, only occurring where the pericardium is firmly adherent to the anterior chest-wall, and when the heart is acting powerfully. It follows immed-iately after the systolic recession, and is in proportion to its force. The diastolic shock is felt by the hand as a " back stroke." It may be per-ceptible only at the apex-beat ; over one or more intercostal spaces ; over a more extensive surface—possibly the entire precordial area ; or even round the left side to the back. The phenomenon is attributed to the elastic recoil or rebound of the chest-wall, at the beginning of diastole, as soon as the systolic dragging force has ceased. In well-marked cases it may be felt as a distinct jerk or blow, which is occasionally so strong as to simulate the impulse of the heart. When present it is regarded as a pathognomonic sign of adherent pericardium.

Apart from the sign just considered, I feel sure that in some cases of

adherent pericardium, with exposure of the heart and great vessels, a diastolic impulse is felt, due to the closure of the aortic and pulmonary valves.   It is noticed over the base, and is quite independent of systolic retraction.

(e) *Posterior Systolic Impulse.*—I believe that this sign is sometimes of value in the diagnosis of adhesion of the pericardium to the structures posteriorly; especially when there are indications of probable agglutination of its two surfaces, and of anterior adhesions.   It is best recognised, not by the hand, but by the head, when placed over the back of the left side of the chest in the practice of direct auscultation.   The movement is directly due to the hypertrophied heart, and is often associated with more or less compression of the lung, which therefore conducts the sensation more readily; but I think that it is likely to be more pronounced when the structures are matted together by adhesions.

(iii.) *Cardiac Dulness.*—Pericardial adhesions or thickening do not in themselves appreciably affect the cardiac dulness, as a rule; but a mass of fibrous tissue about the vessels may certainly cause some increased dulness towards the base.   When, as a consequence of adhesion to the chest-wall, the heart and great vessels are abnormally exposed and superficial, the area of cardiac dulness will be proportionately enlarged, and may be of considerable extent, being often markedly increased in an upward direction, sometimes reaching the second rib.   Part of this altered percussion-sound may be due to adhesion and collapse of overlapping lung.   When enlargement of the heart is associated with the pericardial condition the dulness will be modified accordingly, and is not uncommonly very extensive.   In well-marked cases the dulness resulting directly or indirectly from pericardial adhesions and thickening is very pronounced or even absolute.   According to Dr. Sequeira the margin of the dulness is often particularly well marked, especially along the left upper border. When extensive calcification of the walls of the sac has taken place, the percussion-sound in rare instances has been described as presenting a peculiar osteal quality.

(iv.) *Auscultatory Signs.*—It cannot be said that there are any actually pathognomonic or trustworthy auscultatory signs of adherent pericardium; but one or other of the following points may be worthy of attention in particular cases :—

(a) Should the pericardium be fixed to the chest-wall the heart-sounds are likely to be remarkably superficial.   The first sound is certainly often abnormal in character.   In some cases it is peculiarly sharp and valvular in quality; in others it is markedly dull or muffled at the apex or over the mid-cardiac region; or again it may be prolonged and re-duplicated.   The second sound is frequently apparently reduplicated; but Friedreich maintains that this may be due to the rebound of the chest-wall, which not only causes the diastolic shock but produces a dull sound heard after the second sound of the heart.   Sir John Broadbent regards a weak pulmonary second sound, when there is evidence of hypertrophy of the right ventricle, as a very important indication that the hyper-

trophy was probably due to some intrinsic cause, perhaps adherent peri-cardium. Marked conduction of the heart-sounds towards the back of the left side of the chest, especially when associated with the posterior systolic impulse already referred to, is suggestive of pericardial adhesion behind.

(*b*) A rough pericardial friction-sound may remain over different points of the precordial region, especially towards the base, for a variable period after an attack of pericarditis; and, should it be associated with suspicious signs of adhesion, might be useful as corroborative evidence. Its eventual disappearance would probably indicate that adhesions had formed at the spots where it was previously audible. A double friction-sound at the base, associated with adherent pericardium and a much hypertrophied and dilated heart, may closely simulate aortic murmurs, for which they have actually been mistaken.

(*c*) With regard to endocardial murmurs, a kind of rumbling diastolic or presystolic murmur is sometimes heard at the apex, especially in children, which does not, however, indicate the presence of mitral stenosis. Attention was first drawn to this sign independently by Dr. Graham Steell and Dr. T. Fisher. The murmur may be prolonged and rough; is not followed by the sharp first sound usually present in mitral stenosis; and is unaffected by pressure or respiration. It is possible that a basic systolic murmur may result from the pressure of pericardial thickening upon one or both of the great arteries. The several valvular diseases will give rise to their corresponding murmurs, but I believe that these may be modified in their character by adherent pericardium. A tricuspid regurgitant murmur may ultimately result from dilatation of the right ventricle owing its origin to this condition.

(v.) *Signs connected with Respiratory Movements.*—When examining for pericardial adhesions, it is often highly advantageous to study the effects of deep inspiration and expiration. In the first place, the observation that the position of the apex-beat and the area of extended cardiac impulse are not thus affected may be of much importance; as well as that the area of precordial dulness is not altered. It implies the presence of external adhesions; and the want of any modification in the dulness is particularly marked when the pericardium is adherent to the margins of the lungs. As a result of extensive external pericardial adhesions, inspiratory expansion of the chest may be decidedly less on the left than on the right side. Another sign occasionally observed is impeded descent of the left half of the diaphragm in inspiration, as indicated by diminished movement of the upper part of the abdominal wall on that side. In some cases there is little or no forward movement of the thorax, the lower half of the sternum, and the abdominal wall during inspiration. Tracheal tugging might possibly result from adhesion of the pericardial sac to the bifurcation of the trachea, but I have never found this to be the case.

(vi.) *Arterial Signs.*—In some cases in which pericardial adhesions were proved after death to exist, I have observed a peculiar visible move-ment in connexion with the large arteries at the root of the neck, which

I believe may be of some significance.   It gives the impression that the
ventricle is making an effort to drive the blood into these vessels, but is
prevented from doing so effectually by the adhesions.   The movement
may be modified by the coexistence of aortic or mitral disease.   Arterial
blood-pressure is low ; and the pulse is usually small, and not uncom-
monly irregular, but this is generally associated with the period of failing
compensation.   I think that adherent pericardium tends to increase the
irregularity associated with mitral disease.   The arterial sign to which
Kussmaul and others have attached special importance is the presence of
a marked *pulsus paradoxus*, the pulse intermitting with inspiration, which
has been chiefly noticed in cases of indurative mediastino-pericarditis ;
this sign is, however, by no means trustworthy, as it is often absent and
occurs in other circumstances, and even in perfectly healthy subjects.

(vii.) *Venous Signs.*—The cervical veins are frequently dilated in cases
of adherent pericardium, and should tricuspid regurgitation supervene,
they pulsate.   Sudden collapse of the veins of the neck during the
ventricular diastole has been specially studied by Friedreich, who regards
it, when associated with systolic retraction of the intercostal spaces, as a
most valuable sign of adherent pericardium ; it is never present in any
striking degree without such retraction.   The veins, often tensely filled
during systole, disappear from view during diastole, the subsidence being
synchronous with the diastolic shock felt in connexion with the chest-wall.
Sometimes the supraclavicular fossae are deepened at the same time.   Sir
John Broadbent mentions exceptional cases, in one of which, observed by
François Franck, systolic emptying of the veins of the neck occurred ;
and in another systolic emptying of an enlarged vein on the front of the
chest, to the right of the sternum, which filled during diastole ; of this
I think I have met with another example.   Wenckebach has described
swelling of the veins of the neck during inspiration, concurrent with
pulsus paradoxus, due to impeded emptying of the systemic veins, as a
sign of adherent pericardium.

(viii.) *Skiagraphy.*—Dr. Sequeira has found skiagraphy of great assist-
ance in verifying the results of percussion in cases of enlarged heart with
pericardial adhesion.   It has appeared to him that the fixation of the
dilated cavities has led to a more definite margin, especially along the
left upper border.   The orthodiascope should be of similar assistance.

Course and Modes of Termination.—The course of events in cases
of adherent pericardium differs considerably according to circumstances.
Dr. Sequeira divides them into three great groups, namely : (1) Those in
which compensation is well established, the adhesions giving no trouble in
themselves, and the pericardium and heart being undilated.   (2) Those in
which compensation easily breaks down, presenting all gradations, from
the patient who has been in and out of hospital for years, to one in whom
compensation has been established but once.   The younger patients, and
especially the young females, form the bulk of the cases in this group.
From time to time acute exacerbations occur, the easy working of the
heart being again restored by rest, until at last compensation fails and

death follows. (3) Those in which compensation is never established, and failure of the heart is progressive from the first, and death occurs in from three to nine months after the acute attack of pericarditis. The fatal termination in cases of adherent pericardium may be sudden, even when the condition has not been diagnosed, as well as in those belonging to the second group, but in most recognised cases the failure is gradual, the symptoms becoming more and more pronounced. In exceptional instances death is ushered in with fits, not of uraemic origin.

Diagnosis.—It must be repeated that in a large proportion of cases where pericardial adhesions exist, there are no trustworthy data upon which a definite diagnosis can be based ; though nevertheless the possibility of their existence may suggest itself in explanation of pericardial sensations or cardiac disturbance of obscure origin. The rule is not to forget these lesions in any case, and to take some trouble in their clinical investigation. In not a few instances the diagnosis of adherent pericardium is quite obvious, and yet the condition is entirely overlooked. It is not enough to say that pericardial adhesions exist ; an endeavour must be made to determine their extent and nature ; whether they are external, internal, or both ; and their effects upon the heart and great vessels. Moreover, their association with valvular diseases of this organ, or other independent structural changes, must not be lost sight of, as they are often important factors in such combinations.

If the patient is known to have had one or more attacks of acute or subacute pericarditis, or of rheumatic fever, the existence of adhesions may be reasonably suspected. There may be a definite history of their having formed under observation during convalescence. In other instances an indefinite history merely points to cardiac inflammation of some kind. The frequent association of pericarditis and endocarditis in childhood has an important bearing on diagnosis ; and, when the origin of valvular disease can be traced to early life, pericardial adhesions should be particularly looked for. Unfortunately, in a large proportion of cases no history pointing to pericarditis can be obtained ; and it must not be forgotten that the formation of adhesions may be a chronic process throughout.

The positive diagnosis of adherent pericardium is founded upon careful and systematic investigation and study of the symptoms and physical signs already discussed, not only in themselves, but also in relation to each other. Individual cases differ much in their exact characters. Sometimes the diagnosis has to be made on physical signs alone, there being no prominent symptoms. On the other hand, progressive signs of general venous obstruction, following an attack of pericarditis, may alone indicate the presence of a thick, dense, adherent pericardium compressing the heart, there being no obvious physical signs of the condition. In other instances, again, enlargement of the heart, especially of the right ventricle, occurring without other adequate cause, or perhaps developing with unusual and inexplicable rapidity in connexion with valvular disease, suggests adherent pericardium as a possible cause. Moreover, I fully

agree with Sir John Broadbent that when symptoms of cardiac failure, more especially of right ventricle failure, occur of greater severity than the physical signs present seem to warrant, or where compensation breaks down unaccountably, adherent pericardium must be suspected ; more particularly when rest and suitable treatment fail to give relief. Sir Samuel Wilks has expressed the opinion that severe heart symptoms in young subjects without valvular murmurs point to pericardial adhesions. Dr. Theodore Fisher affirms "that wherever a very large heart is present in a child, in the absence of aortic valvular disease, there is almost certain to be an adherent pericardium."

Prognosis.—There has been much controversy with regard to prognosis in cases of adherent pericardium. Most of the older writers looked upon the condition as incompatible with long life. The late Sir William Gairdner took a more favourable view, and recognised that many patients, by the adoption of a "reduced scale of existence," might live in comfort for years. Modern observations have placed the question on a more rational and definite footing, and attention will now be directed to the more salient points. Often pericardial adhesions are of no consequence whatever, and in other instances they are merely a source of discomfort, and practically do not tend to shorten life. On the other hand, in not a few cases they are extremely serious in themselves, and then the outlook is often very grave, both as regards a fatal termination, and on account of the symptoms to which they give rise, these causing much distress, making life miserable, and being but little amenable to treatment.

The outlook is exceedingly unfavourable in early life. According to Dr. T. Fisher, "adherent pericardium is the most serious form of cardiac disease in children"; he also states that "after the age of twenty years pericarditis is rarely followed by serious consequences"; that "although up to the age of twenty-five or a little later cases of death may occasionally be met with when adherent pericardium—a sequel of pericarditis which occurred some years before—is the main, if not the only, cardiac lesion, after the age of thirty cases of death from adherent pericardium are decidedly rare." Dr. Sequeira's observations shew that in young subjects death occurs at different periods in the two sexes ; in females a pericardial lesion occurring in early childhood commonly leads to a fatal issue between the twelfth and fifteenth years ; in boys the failure occurs later—in the sixteenth and seventeenth years. In adult males the prognosis is good. In women the years of pregnancy and parturition are those in which failure of the heart is prone to occur. Cases are occasionally met with in which the patients live until the period of decay with pericardial adhesions which have probably existed from early life.

The prognosis in cases of adherent pericardium depends not only upon the nature and degree of the morbid conditions involving this structure itself, but also very materially upon the changes affecting the heart and vessels with which it may be associated, whether primary or secondary. If the organ is not enlarged, and works in an orderly manner, the prognosis is good, even when the pericardial adhesions are very extensive

or universal, and life may not be materially shortened, if at all. Dr. Sequeira, from this point of view, lays stress upon the absence or degree of dilatation of the pericardium as the underlying factor. The gravity of the prognosis in early life is generally recognised as due to the fact that in such subjects the effects of pericarditis are associated with those of myocarditis and dilated heart. That the coexistence of adherent pericardium with one or more valvular defects adds seriously to the gravity of a case cannot be doubted, and the pericardial condition often materially hastens the progress of events towards a fatal termination, whilst the combination increases the difficulties of compensation. This is well illustrated by cases of mitral stenosis originating from endocarditis in early life, with or without the effects of pericarditis. When a very thick pericardium grips and compresses the heart, the prognosis is very grave.

Another factor which influences the prognosis in cases of adherent pericardium is the social position of the patient and the conditions of existence. Among those who are exposed to the vicissitudes of life, and who have to work hard, especially during the period of youth and adolescence, soon after an acute attack, the outlook is much more serious. Indications as to prognosis are afforded by the progress of the symptoms, but it must be remembered that improvement may take place temporarily again and again when the end seems approaching, and no definite opinion may be possible as to duration, a case lingering on for a considerable time. Sudden death may take place unexpectedly. Adherent pericardium may materially add to the dangers of acute pulmonary affections, and account for a fatal result in certain cases. This condition adds considerably, in my opinion, to the risks of life insurance.

Treatment.—Chronic pericardial adhesions once formed cannot be got rid of by any kind of local or medicinal treatment. Rest, either prolonged or from time to time, good nourishment, and other suitable measures are of value in preventing or delaying their ill-effects, and in maintaining the nutrition of the myocardium. The patient must live as quiet and regular a life as possible, if the adhesions are obviously of a serious character, and especially when associated with other cardiac lesions, and anything like hard physical work is out of the question. Worry and other causes of disturbed action of the heart must be avoided ; and the digestive and other functions duly regulated. Possibly some of the various exercises now in vogue in the treatment of diseases of the heart may be of service in suitable cases, but where extensive and firm adhesions exist they certainly may do much mischief, if carried out thoughtlessly. Cardiac tonics may be useful in some cases ; but it must be remembered that pericardial adhesions may materially interfere with the action of digitalis and allied agents upon the heart, and then such agents may do much more harm than good. Symptoms must be dealt with on ordinary principles ; and dropsy often requires repeated removal by operation. An operation named "cardiolysis," proposed by Brauer (1902), is said to have proved decidedly beneficial in certain cases in which mediastinitis exists along with adherent pericardium, and the heart

is fixed to a rigid chest-wall. It consists essentially in the resection of some ribs and cartilages in the precordial region. The most striking improvements resulting from this operation are stated to be a better respiration, marked reduction in the size of the liver, and great diminution in the general anasarca. How far such a procedure is admissible or desirable must be left to individual judgment in any particular case, but it certainly does not seem to me attractive. More recently, however, Urban has discussed this operation, and states that it has been performed in 11 cases, with uniformly encouraging results. Most surgeons have removed the costal periosteum, portions of the sternum, and even the intercostal muscles with the ribs ; but in some instances subperiosteal resection of the latter is sufficient.

IV. Hydropericardium ; Dropsy of the Pericardium

Pathology and Etiology.—Hydropericardium, or hydrops pericardii, signifies a serous effusion into the pericardial sac, occurring during life, of a dropsical nature, as distinguished from one of inflammatory origin. As has been previously stated, a certain quantity of fluid, varying under different circumstances, is found in this sac at most necropsies ; this is merely due to transudation from the vessels and heart occurring during the act of dying, and for a time after death. It usually amounts to from half an ounce to an ounce, but under favourable conditions may reach three ounces or more. Definite hydropericardium may occur in the following circumstances :—(i.) As an acute or active effusion in connexion with certain cases of Bright's disease, and it may then follow scarlatina. (ii.) As a part of chronic dropsy, more or less general, usually in cases of cardiac or renal disease ; but occasionally associated with scurvy or purpuric conditions, grave forms of anaemia, tuberculosis, cancer, and other cachexias. In this group the pericardial dropsy almost always follows effusion into the pleurae, and the pericardium is much less frequently involved than other serous membranes. (iii.) Exceptionally from some mechanical difficulty interfering with the local circulation. It may thus occur in connexion with certain affections of the lungs, or even of the heart itself, impeding the return of blood from the cardiac and pericardial veins ; and with disease or thrombosis of these veins, atheroma of the coronary arteries, aneurysm, chronic mediastinitis, or a mediastinal tumour causing pressure upon the veins. Hydropericardium has been known to follow sudden extreme pneumothorax.

Dr. W. Ewart (32) has drawn special attention to cases of latent and transient pericardial effusions, which may occur, independently of acute pericarditis, under the influence of rheumatism, of cardiac affections, of Bright's disease, and so forth. He considers that they may be dependent upon a subacute inflammatory process, but that probably they are more often passive or mechanically induced. No doubt such cases are met with, and if the fluid be rapidly reabsorbed they may run their course entirely undetected.

Anatomical Characters.—The essential morbid condition in hydropericardium is the presence of a quantity of serous fluid in the sac, which has collected during life, but which is not accompanied by any indications of inflammation. The amount varies considerably in different cases. In the large majority of instances it is moderate, from six or eight to twelve ounces ; but it certainly may reach a pint to a pint and a half ; and as much as four pints have been reported, though it is very doubtful whether such large effusions are not really of inflammatory origin. The fluid is, as a rule, clear, and either colourless or of a yellowish or greenish tint. It is sometimes turbid from admixture of degenerated endothelium, or may be tinged with blood-pigment or bile. Haemoglobin may, however, have escaped after death. The effusion is alkaline ; and in composition resembles more or less the serum of the blood, with differences in the relative proportion of the albumin and other constituents. Even a dropsical accumulation in the pericardium may be spontaneously coagulable. In renal cases it may contain urea. When the fluid is abundant it tends to produce, in proportion to its amount, the physical effects already discussed under inflammatory effusion. In prolonged cases the pericardium may become sodden, its endothelium being also changed ; and it is said that the subserous tissue about the heart loses its fat and becomes oedematous.

Clinical History and Diagnosis.—The circumstances in which it occurs make it unlikely that there will be any definite symptoms of hydropericardium, especially if the fluid be but in small or moderate quantity. There is never any pain or other acute subjective sensation, such as is met with in pericarditis. Should the effusion attain a large amount, it may certainly cause a feeling of weight and oppression across the chest, with precordial anxiety ; and will either induce or aggravate previous dyspnoea, obstruction of the venous circulation, and low arterial pressure, with the usual symptoms arising therefrom. In the large majority of cases it merely intensifies pre-existing symptoms, and it is often very difficult to determine the share of pericardial dropsy in their manifestation, though sometimes its effects are obvious enough, especially should it come on rapidly. It does not give rise to any febrile symptoms ; and, as a rule, there is no particular disturbance of the heart's action.

It will thus be evident that by physical examination only can hydropericardium be positively recognised. The absence of friction-phenomena, such as are associated with acute pericarditis, is a most important point of distinction between the two conditions. The signs of the effusion are similar to those fully described under pericarditis, to which the reader is referred. As a rule they only indicate the presence of a moderate amount of fluid, and there may be so little that it cannot be detected at all. It is affirmed that the dulness is more readily altered by changes of posture than in cases of inflammatory effusion. Hydropericardium generally follows effusion into both pleurae ; and the physical signs of this latter condition will probably be well marked before those of pericardial dropsy are revealed. The combination may also cause a

difficulty in diagnosis. I have never met with a case in which acute pericarditis and hydropericardium could not be differentiated by due attention to the circumstances in which they severally occur, and to the points of distinction already indicated. Possibly in connexion with Bright's disease an effusion might collect which it would be difficult to classify definitely as inflammatory or dropsical. The chief danger in diagnosis is that hydropericardium is not thought of, and is consequently overlooked when physical examination would clearly have revealed its presence. The cases of latent and transient pericardial effusion referred to by Dr. Ewart must also be borne in mind, for it is probable that even when considerable it is likely to be overlooked, unless accurate and searching physical examination is made. Should the condition be associated with, and secondary to certain local affections within the chest, the diagnosis may be very obscure and difficult.

Prognosis in cases of pronounced dropsy of the pericardium is, for obvious reasons, usually very grave, and it generally indicates a speedily fatal termination. Temporary improvement or even recovery may, however, take place in some instances under favourable conditions.

Treatment.—As a rule treatment has to be directed to the cause of the hydropericardium, and the measures persisted in which have been previously carried out for the relief of the general dropsy which it usually complicates. It might be desirable in some instances to relieve the venous circulation by venesection or local removal of blood. Cardiac tonics are to be used when required. The application of blisters has been found advantageous in promoting the absorption of pericardial dropsy, but this treatment can only rarely be indicated. Whether tapping is permissible or desirable must be determined by a careful consideration of the circumstances of each individual case.

V. Haemopericardium ; Blood in the Pericardium

Etiology.—It is not uncommon to find a certain amount of blood mixed with inflammatory products in the pericardium; but the circumstances under which pericardial haemorrhage may occur as an independent condition are as follows :—(i.) As a consequence of traumatic injury from without, or by foreign bodies penetrating from the oesophagus. (ii.) Associated with scurvy, purpura, or, extremely rarely, leucocythaemia and allied conditions. (iii.) From rupture of the heart or of a cardiac aneurysm. (iv.) From lesions of the aorta. An aneurysm of the first part of the arch is very apt to open into the pericardium, not uncommonly by a pin-hole rupture. Rarely this event happens in the case of aneurysm of the descending aorta ; and in one reported by Dr. S. H. Habershon (42) the aneurysm was situated at the junction of the transverse and descending portions of the arch. A case has been reported by Charlewood Turner in which rupture of the inner coats of the aorta was followed by a dissecting aneurysm, which perforated into the pericardial sac. Transverse rupture

of the aorta above the valves may occur spontaneously as the result of syphilitic endarteritis and give rise to haemorrhage into the pericardium. Dr. Rolleston has described a very interesting condition in which the inner and middle coats of the commencement of the aorta ruptured transversely, and the blood leaked into the pericardium through a small hole the size of a pin's head in the external coat, but there was no dissecting aneurysm. (v.) From rupture of smaller vessels, namely, one of the coronary arteries, especially if it be the seat of aneurysm, or of vessels in a new growth.

Anatomical Characters.—The quantity of blood which collects in the pericardial sac varies in different circumstances. When there is a large opening and rapid extravasation takes place, it is much less than when it escapes gradually through a small aperture. When an aneurysm bursts freely into the pericardium, the quantity usually found is said to be about 7 ounces, whereas in the case recorded by Dr. Rolleston already referred to it amounted to over 24 ounces. In a traumatic case reported by Mr. Mansell Moullin over 6 pints of thin dark fluid blood were removed from the pericardium in the course of three hours. The patient recovered. In Dr. Habershon's case the pericardium contained about a pint and a half of dark fluid blood. The blood may appear as a soft red clot, jelly-like, or more or less decolorised; whilst a variable and sometimes considerable amount of serum will probably have separated from it. Haemorrhage in the pericardium may set up pericarditis. The sac is distended in a proportionate degree when there is a large collection of blood in its interior.

Clinical History.—There may be previous symptoms or physical signs of the morbid condition which causes the pericardial haemorrhage, but not uncommonly such is not the case, and the lesion is quite unexpected and sudden. Immediate or very rapid death usually occurs, but the event may be preceded by grave cardiac symptoms or collapse. In those cases where the accumulation takes place gradually, the patient may live some time, and may complain of pain, associated with serious cardiac disturbance, faintness or syncope, dyspnoea, and signs of loss of blood. The physical signs, if noted, will be those of an accumulation of fluid in the pericardial sac. Sir Clifford Allbutt has met with a case of a large collection of blood slowly effused into the pericardium from a ruptured coronary artery, in which the signs at the back of the chest which are occasionally associated with effusion were very definite and marked. The prognosis is hopeless as a rule.

Treatment can only be symptomatic. Stimulants and cardiac remedies may be of temporary service in the more prolonged cases. No operative interference is practicable in the great majority of cases, but it may possibly be successful in certain traumatic forms of haemopericardium, as in Mr. Mansell Moullin's case.

VI. Pneumopericardium and its Effects ; Gas in the Pericardium

Pneumopericardium is extremely rare, and it needs but brief consideration in this article.

Etiology.—Gas in the pericardium has been referred to the decomposition of fluid in the sac, especially if the fluid be of an ichorous nature; and it has even been said that this is its most frequent source. The probability is that such decomposition, in the large majority of cases if not always, is a post-mortem change. Its presence has also been attributed to secretion by the membrane, but on no adequate grounds. The two classes of cases in which it is clinically important are—(i.) *Traumatic*, from penetrating wounds, including paracentesis for pericardial effusion; fractured ribs; contusion or crushing of the chest; or injury from the side of the oesophagus. (ii.) *Perforative*, in which a communication is formed externally, or between the pericardium and a cavity or tube containing air. This kind of lesion has been already sufficiently described in relation to acute and suppurative pericarditis, and it will suffice to mention, as illustrations, perforation from the oesophagus, especially in connexion with cancer; rupture into the pericardium of a phthisical cavity or pyopneumothorax; and perforation of a gastric ulcer. A remarkable case is on record in which a hepatic abscess communicated with the stomach and the pericardium, and thus air gained access to the latter. The entrance of gas into the sac may be aided by pressure, by the elastic traction of the lungs upon the pericardium, or by diminution of the size of the heart during systole.

Anatomical Characters.—The gas in cases of pneumopericardium varies in its amount and composition, but is generally offensive. It may so distend the sac, that when this is punctured the gas escapes with a hissing noise. Blood or other materials often gain an entrance at the same time as the gas; or at any rate inflammation is so speedily set up that pneumopericardium has never been clinically observed alone, fluid being always present, rarely serum—*hydropneumopericardium*—usually pus —*pyopneumopericardium*; or the fluid may be ichorous and fetid, and of a dark brown colour. Whatever the position of the patient the gas will always be uppermost and the fluid below. The lungs will be pushed aside and compressed, and the diaphragm depressed, in proportion to the degree of distension of the pericardial sac.

Clinical History.—As might be anticipated, the symptoms of pneumopericardium and its consequences vary much in different cases, and are by no means characteristic. Sometimes there are none; or the patient is merely weak and apathetic. Should gas collect rapidly, there will probably be much precordial distress and sense of distension. The chief objective symptoms which have been observed in different cases are severe dyspnoea, cyanosis, fits of syncope, collapse, a feeble and irregular pulse, and rarely dysphagia. Sleep is necessarily disturbed; and delirium some-

times occurs. Occasionally pneumopericardium is accompanied with rigors, high fever, profuse sweats, and diarrhoea ; but such symptoms are probably due to other and more general causes.

Physical Signs.—It is upon the physical signs that the diagnosis of pneumopericardium and its consequences is practically founded. These are due to the presence of gas and fluid within the sac, and most of them are very striking and peculiar. They may be briefly described as follows :—

(i.) The precordial region is likely to present abnormal fulness or bulging, which may be very pronounced.

(ii.) The apex-beat is weak or absent, but is better felt when the patient bends forwards. Sometimes an impulse is observed over several intercostal spaces.

(iii.) The cardiac movements occasionally bring out a very peculiar crackling sensation, due to the bursting of air-bubbles. Possibly a succussion-splash might be felt on shaking the patient.

(iv.) Percussion-signs are usually very remarkable. Over the region corresponding to the distended pericardium there will be a tympanitic percussion-sound, often with a pronounced metallic quality. It is said that a variation in its height, owing to alterations of the shape of the body of gas in the pericardium by the rhythm of the heart, may be detected by rapidly-repeated percussion. It has also been affirmed that the note differs in its degree of resonance during the systole and diastole respectively, the organ being situated farther forward and downward during the former period, and thus pressing back the air. A distinct cracked-pot sound has been described in several cases, but only when there was an opening in the pericardium. In the recumbent posture the extent of tympanitic resonance is greatest in front. When fluid is present, if the patient be slowly raised to the sitting posture and made to lean forwards, this area diminishes progressively, and the clear sound is replaced below by the dulness of fluid. Lateral changes of position will modify the relations of gas and fluid in a similar way, and thus very rapid and striking changes in the situation and relative limits of the respective percussion sounds are produced. Metallic instruments have been used to bring out the peculiar characters of the percussion-sound.

(v.) Auscultation-signs are also very peculiar, and often remarkable for their loudness. They vary according to the relative amount of gas and fluid in the sac, and the consistence of the latter ; but as a rule different sounds are audible. If there be but little fluid the heart-sounds are abnormally loud, and are accompanied with a clear metallic ring, compared to a chime. Should there happen to be an endocardial murmur or friction-sound, it will probably assume a similar quality. The agitation of fluid and air within the pericardial sac by the action of the heart, and also by deep inspiration, produces adventitious sounds of the most extraordinary kind. They are all of metallic ringing quality, and have been described in different cases as splashing, spluttering, guggling, gurgling, rattling, large crepitating, and churning. They have been likened to the

sound of a water-wheel or mill-wheel (*bruit de roue hydraulique, bruit de moulin*); and in one case to the "shaking of shot in a shot-pouch." Occasionally metallic tinkling has been noticed, due to the dropping of fluid in the pericardial sac. Sounds of the character just described are said to have been produced by the presence of air and blood in this sac. In some instances the cardiac and adventitious sounds are so intense as to be heard, not only by the patient, interfering with sleep, but by those near him, or, it may be, even at a considerable distance off. Sometimes a splashing sound is brought out on succussion; or a bell-sound can be elicited by percussion with coins.

Diagnosis.—If the physical signs just indicated were always pronounced, the diagnosis of pneumopericardium and its accompaniments would be quite easy. Otherwise it would present much difficulty, or might be impossible. No reliance can be placed on symptoms. The only conditions with which it could possibly be confounded are a large cavity in the lung, in the vicinity of the pericardium; a localised pneumothorax; or a greatly distended stomach. Due consideration of the general circumstances and causation of each case, and of the clinical history and phenomena, should obviate any such mistake.

Prognosis.—This is obviously very grave, and the termination is almost always fatal, especially as the pneumopericardium is usually a complication of some grave disease or lesion. A few cases of supposed recovery have been reported, but these have been chiefly of traumatic origin.

Treatment.—But little can be said under this head. The patient must be kept as quiet as possible, and in the position which is found to be most comfortable. Stimulants, sedatives, or cardiac agents should be administered as circumstances require, but each case will dictate its own methods. The question of operation naturally presents itself, and in suitable cases it might be desirable to let out some of the gas by means of a fine trocar, the patient being in the recumbent posture; or to open up the pericardium freely, especially if it contain inflammatory or other products of a low type. This matter must be regarded and dealt with entirely from a surgical point of view.

VII. TUBERCULOSIS, MALIGNANT GROWTHS, AND HYDATIDS

In order to complete the account of diseases of the pericardium brief reference must be made to this group of morbid conditions.

Tuberculosis of the pericardium in its minor degrees is perhaps more common than is usually supposed. It is only in exceptional cases, however, that the membrane presents grey granulations in general acute miliary tuberculosis. In the large majority of instances tuberculosis implicating the pericardium is chronic, and secondary to tuberculous disease elsewhere, especially of the lungs, from which it spreads directly. It may, however, follow disease of the bronchial or mesenteric glands.

A simple pericarditis appears to be more common than tuberculous, even in cases of pronounced pulmonary tuberculosis ; and chronic inflammatory products in the pericardium may possibly become infected with the tubercle bacillus. Dr. Habershon (43) recorded an interesting case of general tuberculosis affecting unusual structures, in which there was extensive tuberculous pericarditis. In a case of phthisis which came under my observation, changes due to chronic pericarditis were pronounced, but careful examination failed to detect any tubercles or tubercle bacilli. In some cases grey and caseating tubercles are scattered over the serous coat, or in the midst of inflammatory products or bands of adhesion.

Malignant disease implicating the pericardium is of more pathological than clinical interest, and has attracted but little attention. The few remarks which I propose to offer are founded mainly on Dr. Sholto Douglas's observations. Rare examples of supposed primary sarcoma of the pericardium have been recorded by the late Sir William Broadbent and others, but it is possible the growth arose in the overlying thymus gland or its remains. Practically the condition is always secondary, and due either to metastatic invasion through the blood-stream or to extension from adjacent parts. Metastatic growths invading the visceral pericardium are not very rare as discrete nodules scattered over the surface of the heart. Usually they are sarcomatous, but in generalised carcinomatosis the pericardium may be invaded by secondary nodules in the heart-muscle. This mode of invasion is extremely rare in cases of primary malignant tumours in the thorax (2 in 92 cases collected by Douglas) ; and the structure is usually invaded by direct extension. This happened in 14 out of the 92 cases ; and the primary growth was situated either in the root of the lung, the glands about the bifurcation of the trachea, the lung, the thymus, or the mediastinum. Although the oesophagus is in an overwhelming preponderance of cases the most frequent site of primary intrathoracic growth, in no case was the pericardium invaded by extension, and in one only was there a secondary metastasis. There may be adherent pericardium or recent pericarditis, without any actual invasion by the growth. The route of extension in 7 of the 14 cases was along one or more of the great vessels, either after invading them, or by continuity along their outer aspects. In the other 7 cases the invasion took place by a route independent of the vessels. When the pericardium has become implicated, further extension seems always to take place by continuity in and under the visceral serous layer, sub-pericardial growth, and there is no record of secondary independent deposits.

All Dr. Douglas's pericardial growths were sarcomatous—round-celled, spindle-celled, or mixed-celled. The tumour appears as a diffuse infiltration, but it may project into the pericardial cavity in knobby elevations or fungating prominences. The wall of the pericardium may be partially destroyed.

Hydatids of the pericardium are so rare that out of 1897 cases collected by Davies Thomas of Adelaide, in only two was this structure affected. Moreover, in no instance had a hydatid cyst in the cardiac walls ruptured into the pericardial sac, probably because of adhesions between the two surfaces. This writer mentions one case, however, in which a cyst situated between the liver and the diaphragm ruptured into the pericardium.

The effect of any morbid growth in connexion with the pericardium would probably be to set up inflammatory changes. These changes are very seldom acute; they may be subacute; but by far most commonly are chronic in their development and results. The combinations in these chronic cases of adhesions, pericardial thickening, and localised collections of fluid, along with the morbid growths, may be very complicated. The effusion is commonly haemorrhagic; but in malignant cases it may be purulent or ichorous, and possibly also in those of a tuberculous nature.

Clinically implication of the pericardium by tuberculous or malignant disease could only be suspected or recognised by the appearance of symptoms and physical signs of pericarditis, especially chronic, in such cases as tuberculosis or old phthisis, or associated with an intrathoracic tumour. It certainly is desirable to watch the pericardium in cases of chronic phthisis, though, as already stated, the changes which may then arise are by no means always tuberculous. It is very likely that tubercle or malignant growth may produce a friction-sound, and this has been definitely asserted; but no positive diagnosis could be founded on this sign. The implication of the pericardium in these lesions, in cases in which the primary seat of mischief is away from the chest, could only be made out by the occurrence of pericarditis and its consequences, which would draw attention to this part.

Treatment is entirely symptomatic and constitutional, and no definite rules can be laid down. Operative interference might be indicated for the removal of pericardial effusion to give temporary relief, but nothing can be done for the morbid growths themselves. Obviously when the pericardium becomes involved in malignant disease the end cannot be far off.

<div style="text-align:right">FREDERICK T. ROBERTS.</div>

REFERENCES

1. ALLBUTT, Sir CLIFFORD. "On Paracentesis Pericardii," *Lancet*, London, 1869, i. 807.—2. *Idem. Brit. Med. Journ.*, 1870, ii. 31.—3. APORTI and FIGAROLI. "Experiments on Pericardial Effusion," *Boston Med. and Surg. Journ.*, 1901, cxliv. 236; also *Therapeutic Gaz.*, Detroit, 1901, 3rd ser. xvii. 418.—4. BACON. "A Procedure for Opening the Pericardium," *Amer. Journ. Med. Sc.*, Phila., 1905, cxxx. 652.—5. BALFOUR, G. W. "Diseases of the Pericardium," Quain's *Dictionary of Medicine*, London, 1894, 2nd edit., ii. 334.—6. BARNARD. "The Functions of the Pericardium," *Journ. Physiol.*, London, 1898, xxii.; *Proc. Physiol. Soc.*, p. xliii.—7. BAUER. "Diseases of the Pericardium," Ziemssen's *Cyclop. of Pract. Med.*, London, 1876, vi. 545.—8. BÄUMLER. "Cases of Partial and General Idiopathic Pericarditis," *Trans. Clin. Soc.*, London, 1872, v. 8.—9. BRAMWELL, B. *Diseases of the Heart and*

Aorta, London, 1884.—10. BRAUER. "Cardiolysis," *Medical Annual*, Bristol, 1908, 303 ; *Brit. Med. Journ.*, London, 1908, i. epitome, p. 73.—11. BRENTANO. " Zur chirur- gischen Behandlung der Pericarditis," *Deutsche med. Wchnschr.*, Leipzig, 1898, xxiv. 506 ; [abstr. in] *Medical Annual*, Bristol, 1902, 442.—12. BROADBENT, Sir JOHN. *Adherent Pericardium*, London, 1895.—13. *Idem*. "Discussion on Adherent Peri- cardium," *Trans. Med. Soc.*, London, 1898, xxi. 117.—14. BROADBENT, Sir W. H. " Malignant Disease (Sarcoma) of the Pericardium," *Trans. Path. Soc.*, London, 1882, xxxiii. 78.—15. *Idem* and Sir JOHN BROADBENT. *Heart Disease*, London, 1897.—16. CANTLIE. "Rational Treatment of Pericardial Adhesions," *Brit. Med. Journ.*, London, 1889, i. 833.—17. CHEADLE. " Acute Rheumatism," *Brit. Med. Journ.*, 1896, i. 65.—18. CHEVERS. "Observations on Diseases of the Orifice and Valves of the Aorta," *Guy's Hosp. Rep.*, London, 1842, vii. 387.—19. COLLINS. "Pyoperi- carditis," *New York Med. Journ.*, 1901, lxxiv. 919.—20. COOMBS, CAREY. "Rheu- matic Carditis in Childhood," *Bristol Med.-Chir. Journ.*, 1907, xxv. 193.—21. CORVISART. *Maladies du cœur*, Paris, 1818.—22. COUTTS and ROWLANDS. "Case of Purulent Pericarditis associated with Empyema in a Child aged two and a half Years," *Brit. Med. Journ.*, 1904, i. 9.—23. DEAN. "Surgery of the Pericardium," Quain's *Dictionary of Medicine*, London, 3rd edit., 1902, 1187.—24. DEGUY. (Pericardial Effusion, Surgical Treatment), *Journ. des praticiens*, Paris, 1902, 36 ; [abstr. in] *Medical Annual*, Bristol, 1903, 510.—25. DICKINSON, W. H. "Case of Purulent Effusion into the Pericardium treated successfully by Aspiration and Drainage," *Trans. Clin. Soc.*, London, 1889, xxii. 48.—26. DOCK. "Paracentesis of the Peri- cardium," *Brit. Med. Journ.*, London, 1906, ii. 1026.—27. DOEBERT. "Inflammation of the Pericardium and Paracentesis," *Brit. Med. Journ.*, 1904, ii. epitome, p. 13.—28. DOUGLAS, J. S. C. "The Invasion of the Pericardium by Malignant Tumours arising primarily within the Thorax," *Med. Chron.*, Manchester, 1906-7, 4th ser., xii. 207.— 29. DUROZIEZ. *Traité clinique des maladies du cœur*, Paris, 1891.—30. EDWARDS. "Diagnosis of Pericarditis," *Med. Rec. N.Y.*, 1902, lxi. 201.—31. EWART, W. "Practical Aids in the Diagnosis of Pericardial Effusion," *Brit. Med. Journ.*, 1896, i. 717.—32. *Idem*. " On Latent and Transient Pericardial Effusion " (plate), *Lancet*, London, 1896, ii. 1446.—33. *Idem*. "Discussion on Adherent Pericardium," *Trans. Med. Soc.*, London, 1898, xxi. 119.—34. FISHER, T. "Some Features of Adherent Pericardium," *Clin. Journ.*, London, 1907, xxx. 308.—35. FRANCK. "Maladies du péricarde," *Traité de Médecine* (Bouchard, Brissaud), Paris, 1905, v. 56.— 36. FRIED- REICH. "Zur Diagnose der Herzbeutelverwachsungen," *Virchows Arch.*, Berlin, 1864, xxix. 296.—37. GAIRDNER, Sir W. T. "On the Favourable Terminations of Peri- carditis, and especially on Adhesions of the Pericardium," *Month. Journ. Med. Sc.*, Edin., 1851, 3rd ser. iii. 103.—38. *Idem*. "On the Results of Adherent Peri- cardium," *Edin. Med. Journ.*, 1858, iii. 1119.—39. *Idem*, "Pericardial Lesions," *Brit. Med. Journ.*, 1898, ii. 758.—40. GEE. "The Tripod of Life," *St. Bart.'s Hosp. Rep.*, London, 1898, xxxiii. 1.—41. GIRAUDEAU. "De la paracentèse du péricarde," *Semaine méd.*, Paris, 1898, xviii. 377.—42. HABERSHON, S. H. "Aneurysm rup- turing into the Pericardium," *Trans. Path. Soc.*, London, 1889, xl. 62.—43. *Idem*. "Tuberculous Ulcer of Stomach associated with Tuberculous Disease of the Peri- cardium and Brain," *ibid.*, 1894, xlv. 73.—44. HARRIS, T. *Indurative Mediastino- pericarditis*, London, 1895 ; also, *Med. Chron.*, Manchester, 1894-95, N.S. ii. 1, 87, 178, 250.—45. HEAD. "On Disturbances of Sensation, etc.," *Brain*, London, 1896, xix. 153 (Pain in pericarditis), 158.—46. HOPE, J. *Diseases of the Heart*, London, 1839.—47. KELLY, A. O. J. "Multiple Serositis," *Am. Journ. Med. Sc.*, Phila., 1903, cxxv. 116.—48. KENNEDY. "On Adherent Pericardium," *Edin. Med. Journ.*, 1858, iii. 986.—49. KUSSMAUL. "Ueber schwielige Mediastino-Pericarditis und den paradoxen Puls," *Berl. klin. Wchnschr.*, 1873, x. 433.—50. *Idem*. Von Ziemssen's *Cyclop. Pract. Med.*, London, 1876, vi. 650.—51. LAENNEC. *Traité de l'auscultation*, Paris, 1826, tome ii. 651.—52. LEES. "Acute Dilatation of the Heart in Rheumatic Fever," *Med.-Chir. Trans.*, London, 1898, lxxxi. 401.—53. *Idem*. "Discussion on Adherent Pericardium," *Trans. Med. Soc.*, London, 1898, xxi. 120.—54. *Idem*. "The Harveian Lectures on the Treatment of some Acute Visceral Inflammations," *Brit. Med. Journ.*, London, 1903, ii. 1385.—55. *Idem*. "Treatment of Pericarditis," *Lancet*, London, 1893, ii. 188.—56. LEES and POYNTON. "Acute Dilatation of the Heart in the Rheumatism and Chorea of Childhood," *Med.-Chir. Trans.*, London, 1898, lxxxi. 419. —57. MONSARRAT. "Pericardial Effusion ; Surgical Treatment," *Medical Annual*, Bristol, 1903, p. 510.—58. MORISON, A. "On Thoracostomy in Heart Disease," *Lancet*,

London, 1908, ii. 7.—59. MOULLIN, C. MANSELL. "Case of Haemo-pericardium : Removal of Six Pints of Fluid," *Trans. Clin. Soc.*, London, 1897, xxx. 217.—60. OGLE and ALLINGHAM. "A Suggestion for a Method of Opening the Pericardial Sac, founded on a Case of Purulent Pericarditis," *Lancet*, 1900, i. 693.—61. PAGET, S. *Surgery of the Chest*, London, 1896, 384.—62. PARKER, R. W. "Extensive Pyo-pericardium associated with Osteomyelitis; Free Incision of the Sac : Irrigation : Death," *Trans. Clin. Soc.*, London, 1889, xxii. 60.—63. PEACOCK. "Congenital Misplacement of the Heart," Quain's *Dictionary of Medicine*, London, 1894, 2nd edit., i. 793.—64. PENDLEBURY. *Lancet*, London, 1903, i. 798 ; 1904, ii. 1145. —65. PLEASANTS. "Traumatic Pericarditis, Endocarditis, and Myocarditis," *Johns Hopkins Hosp. Bull.*, Balt., 1903, xiv. 124.—66. PORTER. "Suppurative Pericarditis and its Surgical Treatment," *Boston Med. and Surg. Journ.*, 1900, cxliii. 385.—67. POWELL, Sir R. D. "Discussion on Adherent Pericardium," *Trans. Med. Soc.*, London, 1898, xxi. 115.—68. POYNTON. "Case of Rheumatic Pericarditis and Extreme Dilatation of the Heart," *Med.-Chir. Trans.*, London, 1899, lxxxii. 355.—69. *Idem.* "Analysis of 100 Cases of Fatal Suppurative Pericarditis in Childhood," *Quart. Journ. Med.*, Oxford, 1908, i. 225.—70. PREBLE. "Etiology of Pericarditis," *Journ. Amer. Med. Assoc.*, Chicago, 1901, xxxvii. 1510 ; also, *New York Med. Journ.*, 1901, lxxiv. 1111. —71. REICHARDT. "Zur Kasuistik der Perikardiotomie," *Centralbl. f. Chir.*, Leipzig, 1900, xxvii. 1109 ; also, *Medical Annual*, Bristol, 1902, 443.—72. ROLLESTON, H. D. "Spontaneous Rupture of the Internal and Middle Coats of the Aorta ; Leakage into the Pericardium," *Trans. Path. Soc.*, London, 1893, xliv. 37.—73. ROTCH. "Absence of Resonance in the Fifth Right Intercostal Space diagnostic of Pericardial Effusion," *Boston Med. and Surg. Journ.*, 1878, xcix. 421.—74. SANSOM, A. E. *Diagnosis of Diseases of the Heart*, London, 1892.—75. SCOTT and LE CONTE. "Medical and Surgical Considerations in Pyopericarditis, with Report of Cases," *Am. Journ. Med. Sc.*, Phila., 1904, cxxviii. 447.—76. SEQUEIRA. "The Remote Prognosis of Pericarditis," *Med.-Chir. Trans.*, London, 1899, lxxxi. 401.—77. SIBSON, F. "Peri-carditis, Adherent Pericardium," Reynolds's *System of Med.*, London, 1877, iv. 186.— 78. *Idem. Works of*, edited by W. M. Ord, London, 1881.—79. SKODA, J. "Über die Erscheinungen, aus denen sich die Verwachsung des Herzens mit dem Herzbeutel an lebenden Menschen erkennen lässt," *Ztschr. d. Gesellsch. der Aerzte zu Wien*, 1852, Jahrg. viii. i. 306.—80. STEELL, GRAHAM. "Diastolic Murmur in Dilatation of the Heart," *Practitioner*, London, 1894, lii. 254.—81. *Idem. Diseases of the Heart*, Man-chester, 1906.—82. STEWART, Sir T. GRAINGER. "Case of Pericarditis," *Trans. Med.-Chir. Soc.*, Edin., 1884, N.S. iv. 53.—83. STOKES, W. *Diseases of the Heart*, Dublin, 1854.—84. STURGES. "Heart Inflammation in Children," Lumleian Lectures, *Lancet*, London, 1894, i. 583, 653, 723.—85. TAYLOR, F. "Combined Pleural and Pericardial Adhesion," *Brit. Med. Journ.*, 1898, i. 1336.—86. THAYER. "Two Cases of Tuberculous Pericarditis with Effusion," *Johns Hopkins Hosp. Bull.*, Balt., 1904, xv. 149.—87. THOMAS, J. D. *Hydatid Disease*, Sydney, 1894.—88. TRAUBE. *Gesammelte Beitr. z. Path. und Physiol.*, Berlin, 1878, iii.—89. TROUSSEAU. *Lectures on Clin. Med.*, London, New Syd. Soc., iii. 364.—90. TURNER, F. C. "Rupture of the Aorta, Dissecting Aneurysm, Perforation of the Pericardium," *Trans. Path. Soc.*, London, 1885, xxxvi. 152.—91. URBAN. "Über Cardiolyse bei pericardio-mediastinalen Verwachsungen," *Wien. med. Wchnschr.*, 1908, lviii. 395. — 92. WALSHAM, H., with G. H. ORTON. *Röntgen Rays in Diagnosis of Diseases of the Chest*, London, 1906.—93. WALSHE, W. H. *Diseases of the Heart*, 3rd edit., London, 1862. —94. WENCKEBACH. "Remarks on some Points in the Pathology and Treatment of Adherent Pericardium," *Brit. Med. Journ.*, 1907, i. 63.—95. WEST, S. "Case of Purulent Pericarditis treated by Paracentesis and by Free Incision, with Recovery," *Med.-Chir. Trans.*, London, 1883, lxvi. 235.—96. *Idem.* "Discussion on Adherent Pericardium," *Trans. Med. Soc.*, London, 1898, xxi. 118.—97. WHEELHOUSE. "Case of Distended Pericardium, threatening Death : relieved by Paracentesis," *Brit. Med. Journ.*, 1868, ii. 384.—98. WHITTAKER. "Diseases of the Heart and Pericardium," *Twentieth Cent. Pract. Med.*, London, 1896, iv. 1.—99. WILKS, Sir S. "Adherent Pericardium as a Cause of Cardiac Disease," *Guy's Hosp. Rep.*, London, 1871, 3rd ser. xvi. 196.

F. T. R.

DISEASES OF THE MYOCARDIUM

By Sir RICHARD DOUGLAS POWELL, Bart., K.C.V.O., M.D., F.R.C.P.

As with other muscular organs, the heart is liable to fatigue, to over-strain, to disturbed innervation, to impaired nutrition; and these conditions commonly depend upon either a defect in the nutritive qualities of the blood with which it is supplied, or on a temporary or permanent restriction in that supply through alteration of the vessels. Further, the heart-muscle may undergo degenerative changes, or may atrophy and shew fibrous substitution; and these changes may be general or localised. Yet, again, the heart-muscle may undergo hypertrophy in obedience to the demands of excessive labour; finally, the heart may be invaded or occupied by growths, parasitic or other, of various kinds. With the various diseases of the endocardium, pericardium, and valves of the heart I have here no immediate concern, although I shall have to refer to them incidentally in an endeavour to give a clear account of myocardial lesions.

The several lesions of the myocardium above mentioned will be found to group themselves naturally under the pathological headings of—

I. Impairment Secondary to General Blood Conditions.—(A) Anaemia; (B) toxic changes.

II. Impairment Secondary to Altered Blood-Supply.—(A) From paroxysmal affections of the coronary arteries: (B) from permanent changes in the coronary arteries; (i.) atheroma; (ii.) thrombosis or embolism; (iii.) aneurysm.

III. Impairment due to Senile Changes.—(a) Pigmentary degeneration; (b) atrophy.

IV. Impairment arising from Functional Strain.—(a) Hypertrophy; (b) acute dilatation; (c) textural damage.

V. Impairment of Inflammatory Origin—Myocarditis.—(a) Interstitial; (b) parenchymatous; (c) purulent; (d) syphilitic; (e) segmentation and fragmentation (?).

VI. Growths.—(a) Non-malignant; (b) malignant, primary, secondary; (c) syphilitic and tuberculous granulomas.

VII. Parasites. — (a) Hydatid; (b) *Cysticercus cellulosae*; actino-mycosis; (c) *Trichinella spiralis*.

I. IMPAIRMENT SECONDARY TO GENERAL BLOOD CONDITIONS.—A. **Anaemia.**—*Pathology.*—In cases of marked anaemia, as in chlorosis, the nutrition of the heart-muscle suffers; the organ is paler than natural, somewhat glistening and wet-looking on section, and gives less than the normal resistance to the pressure of the finger. On microscopic

examination in persons who have died from some intercurrent malady
no change may be noticed; but most commonly the fibres have under-
gone a certain degree of fatty change, and present a few refracting
granules. In some cases of extreme anaemia, however, a very notable
degree of fatty change may be found in the muscular fibres; the internal
surface of the organ, especially over the left ventricle and papillary
muscles, presents a streaked or flecked appearance, due to groups of small
opacities seen through the transparent endocardium, the degeneration
affecting the muscular fibres having a patchy distribution.

Clinically, in all cases of extreme simple anaemia of any considerable
duration, one may observe a certain degree of enlargement of the heart;
the apex - beat is a little to the left of the normal, and the area of
percussion dulness extends slightly upwards; frequently a soft murmur is
to be heard over the apex-beat, which is not merely conducted from the
pulmonary area, but has the characteristics of mitral regurgitation, and is
no doubt due to a dilatation of the left ventricle, so that the base of
attachment of the papillary muscles becomes displaced, and the mitral
valve slightly incompetent at the moment of greatest intra-ventricular
pressure. The heart's action is quickened, and is peculiarly irritable to
the calls of slight effort or to reflex or emotional stimuli. These symptoms,
which constitute the cardiac features of anaemia, are of course only in
part directly due to the state of the heart-muscle, they depend rather
upon the condition of the blood and the debilitated state of the nervous
system; and to both these latter causes, as well as to the cardiac enfeeble-
ment, is also attributable that degree of oedema of the extremities which
is so common in marked anaemia.

B. Toxaemia.—*Hyaline Degeneration.*—A peculiar hyaline swelling
of the muscle-fibres of the heart in diphtheria has been described by
Bouchut, Labadie-Lagrave, and Rosenbach. The last-named author looks
upon it as an inflammation. Similar changes are met with in the voluntary
muscles in enteric fever. Sir R. Boyce speaks of it as a hyaline de-
generation of connective tissue, consisting of hyaline material similar to
amyloid, but without the chemical reaction of the latter. Hyaline
degeneration identical with that in the myocardium is more commonly
observed around the arteries, sometimes permeating, and causing extensive
atrophy of the muscle-fibres of their middle coat. The change is often
limited to one part of the heart, and in sections sometimes only one,
sometimes several muscle-cells are found affected (Mollard and Regaut).

Cloudy Swelling—Granular Degeneration.—A condition in which the
fibres of the heart lose their striation and become finely granular; the
finely granular appearance is removed by the addition of acetic acid,
which would, on the other hand, define more clearly any fatty granules.
The granules are most numerous near the central parts of the muscle-
cell. The nucleus is normal. In a later stage the cells shrink in size,
the nuclei remaining relatively large. The nucleus contains compara-
tively little chromatin and stains faintly; striation becomes indistinct.
Granular degeneration is met with, especially in diphtheria, enteric

and typhus, and is indeed common to all febrile states of sufficient duration.

Fatty Degeneration.—In certain poisoned conditions of blood, as from lead, arsenic, and, in a most notable degree, from phosphorus, fatty change in the muscular fibres of the heart may be very extensive; and, in cases of phosphorus poisoning in which the patient has survived the more immediate gastro-intestinal symptoms, it is the principal source of danger. The mildest form of blood contamination—although very important from its being so common—is the absorption of toxins from the colon in neglected torpidity of the bowels, a source no doubt operative in the production of the fatty heart of anaemia. The most intense of the poisons of organic origin affecting the heart is that modification of the toxin of diphtheria which is formed in the later stages of this disease, and which appears to be responsible for the profound fatty change of the heart (in common with other organs) which is only equalled in cases produced by phosphorus. For the pathology of fatty degeneration of the heart the reader should refer to the article on the General Pathology of Nutrition (Vol. I. p. 576). It would appear that the fatty change in the muscle-fibre is at least not always due to degeneration of the albuminous protoplasm, but that the fat-granules may be imported into the cells and be there deposited unchanged.

Although the fatty heart is always somewhat increased in size, it may not be increased in weight; the specific gravity of muscle being reduced by fatty change. The pericardium and endocardium usually escape change, but the cavities of the heart are enlarged, especially the left ventricle; and slight incompetence of the mitral valve is often revealed when the valve is properly tested by a fluid pressure equal to that of the blood. I have often seen, in the post-mortem room, a heart inadequately tested in this respect. A degree of regurgitation, clinically observable, may be overlooked if the ventricle and valve are not subjected to sufficient fluid pressure.

On microscopic examination, groups of fibres are found in which the fibrillae are in part replaced by rows of refracting fatty granules, the change appearing first in the neighbourhood of the nuclei of the fibres. Besides the groups of more intensely fattily-changed fibres, the other fibres are more or less dotted with fatty granules (see p. 110).

Repair in Fatty Degeneration.—Clinical observations would lead us to suppose that repair of fattily degenerated hearts is possible, and even of frequent occurrence; Coats believed that it takes place by absorption of the fat and an actual new formation of the muscular tissue. That such new formation is abundantly possible is evident from the readiness with which healthy hypertrophy is established to compensate valvular defects, or in response to other unusual calls upon the muscular activity of the heart.

Under the heading of changes of the myocardium of toxic origin we should certainly include those consequent upon chronic gouty conditions and chronic uraemic poisoning; although, as in the less-defined changes

induced by alcoholism, nicotinism, and the like, the lesions have features in common with those induced by other causes, and will be described later.

There can be little doubt that the high-pressure pulse and consequent increased call upon the heart which are associated with chronic affections of the kidney are combined effects of central nervous induction, having for their purpose such an increase of blood-pressure as shall promote compensatory kidney function ; and it is very probable that some stimulation of the internal secretion from the suprarenals is the immediate cause of the central nervous stimulus.   In chronic gouty conditions the cardiovascular function is similarly modified.   Habitual high arterial blood-pressure from whatever cause produces the same changes, although varying in degree, in the myocardium.   These changes are, first, hypertrophy, and, secondly, fibro-fatty degeneration.

II. Impairment of the Myocardium Secondary to Altered Blood-Supply.—A. Paroxysmal Alterations of the Coronary Arteries. —Many authors have pointed out the occasional occurrence of angina pectoris in young people attributable to excess in tobacco-smoking ; and have observed the anginal paroxysm of like causation in older persons. Many such cases have come within my own experience.   Besides its other effects tending to disturbance of the cardiac innervation, Huchard holds the view that nicotine has a more direct action, by causing spasmodic contraction of the coronary vessels.   It is difficult to bring evidence sufficiently demonstrative to prove this opinion or to refute it. It is denied by Prof. Cushny.   Huchard relies chiefly upon the spasm of voluntary muscles and upon the pallor and arterial contraction observed in nicotine intoxication, upon the high arterial pressure often to be observed in smokers, and upon the experiments of Claude Bernard in 1857, and by himself and others since, shewing the local effect of nicotine in causing contraction of the vessels in the frog's foot.   There is every reason to believe that the coronary arteries, like other vessels of equal size and equally richly endowed with muscular tissue, are liable to spasmodic contraction ; and it is quite possible, as maintained by Huchard, that in some cases the abuse of nicotine may directly cause such constriction and produce temporary anaemia and disturbed function of the heart-muscle.   It has not been shewn, however, that any textural damage to the heart's substance has been caused by the vasomotor effects of nicotine upon its circulation.

The remoter effects of nicotine in causing arterial and muscular degeneration, if such there be, are not included in the present subject.

B. Permanent Changes in the Coronary Arteries.—(i.) Atheroma of the Coronaries.—This may arise : (a) From the natural effects of age leading to degeneration of the intima, with secondary thickening and softening, or calcareous deposition.

(b) These senile changes may be anticipated in constitutional dyscrasias, especially syphilis, alcoholism, and gout ; the sequence of

events being much the same, namely, degenerative impairment of elasticity, patchy thickening, fatty change, or calcareous deposition.

(*c*) Hereditary disposition plays an important part in determining premature decay of the arterial system.

The above conditions are general to the whole arterial system, but are most manifest at those portions of it at which the stress of normal arterial pressure is most heavy. The origin and arch of the aorta and the coronary arteries are the portions thus affected which concern us at the present moment; and it may be noted that atheromatous narrowing of the coronaries is generally most marked at their aortic origins, and is often limited to these parts, often being an aortic change involving the calibre of the coronary arteries rather than disease of those vessels themselves.

(*d*) The chronic arterial strain of laborious occupations has a very important influence in producing chronic patchy endarteritis of the aorta and the coronary arteries; and it operates very commonly in conjunction with the causes of arterial degeneration spoken of under headings (*b*) and (*c*).

There can be little doubt that the peculiar patchy distribution of endarteritic thickening is due to small rifts at points of least resistance of an intima rendered more brittle by degenerative changes, and to the secondary nuclear overgrowth and subsequent degenerative changes ensuing thereupon.

(*e*) Syphilis, apart from its general effect of disposing to arterial atheroma, may form granulomas in and about the arteries, thickening their inner coats and leading to narrowing or obliteration. [*Vide* art. "Disease of Arteries," p. 562.]

(*f*) Vessels of small calibre, such as the coronary arteries, when narrowed and with their intima changed by atheromatous or specific arteritis, are very apt to become abruptly and completely closed by thrombosis.

(*g*) The coronary arteries, like other vessels, are liable to embolic closure, although they are much less prone to this obstruction than are other vessels in the more direct current of the circulation. Such embolisms when they arise may be simple or infective.

Having now enumerated the possible causes of narrowing or obliteration of the coronary arteries, let us look to the consequences of such narrowing, which we shall find to embrace the most important lesions of the cardiac muscle.

(*a*) *Fatty Degeneration of the Heart.*—I have already spoken of fatty degeneration of the heart as a consequence of general anaemia, and in certain states of toxaemia; the degeneration arising from local anaemia, due to constriction of the supplying vessels, is of the same kind, but is much less acute, and is more patchy in its distribution. In hearts in which the coronary narrowing affects both vessels at their origins, the distribution of fatty change would be more uniform; but these cases are rare. Often only one coronary vessel is thus affected, and sometimes only

certain branches within the substance of the heart are much contracted by atheroma. Thus the change, at least in any serious degree, may be limited to one side of the heart, or to one or more portions of one or both ventricles or auricles.

The process of fatty degeneration of the cardiac muscle consists, as already stated, in the gradual replacement of the sarcous elements by fatty granules, the deposition of granules beginning about the nuclei and extending linearly towards the fibre-ends. The affected tissue is thus rendered more opaque in streaks of a tawny-yellow colour, is softer and more friable under the finger, and in well-marked patches gives a greasy section. In some cases, in which the degeneration is extreme over a restricted area corresponding with an occluded vessel, the fatty softening may be so great as to resemble abscess.

In combination with the fatty degeneration there is more or less atrophy of the muscular fibres, and in substitution for them an overgrowth of connective-tissue elements resulting in the formation of fibroid tissue (fibroid or false hypertrophy). In this respect fatty degeneration of the heart, induced by restricted blood-supply from narrowed vessels, differs from the same degeneration due to general anaemia or toxic causes. In the case of old people, in whom the degenerative changes are a part of general senile decay, the fatty change may be unattended with fibrosis.

Although the internal surface of the ventricles may be specked and streaked with opacities—much more irregularly disposed, however, than is the case with degenerations of general blood origin—the endocardium itself is rarely affected. The size and weight of the heart, and the thickness or thinness of its walls, depend chiefly upon the amount of fibroid substitution which is associated with the fatty change. The pericardium is not necessarily involved, although it may be more opaque and thicker than normal.

It was found, in speaking of the more acute fatty degeneration of the heart due to general blood conditions (p. 107), that partial or complete repair was possible by a renewal of the muscular fibres in the same way as an extra growth of such fibres can take place in healthy hypertrophy, whilst at the same time the fattily degenerated fibres became absorbed. In degeneration due to permanently narrowed blood-supply, however, no such repair can take place to any appreciable extent; for the anastomosis of the two coronary arteries, supposing only one to be affected, is not free enough to provide a sufficient circulation for the purpose. Nevertheless, we may see in the overgrowth of fibrous tissue, of a somewhat depraved sort it is true, an attempt to maintain the due resistance of the heart-walls to blood-pressure, without however any corresponding preservation of contractile power.

Symptoms and Signs.—The fatty heart is a weak heart, weak in its muscular power, and weak in its resistance to blood-pressure. It is either more or less arrhythmic in action, or readily becomes so under any extra demand upon it from excitement or effort. It is also (except in cases in which the degeneration of the heart goes hand in hand with general

atrophy of blood and tissues in old age) an enlarged heart, increased in size by the dilatation of the ventricles, and especially of the left ventricle, under the normal blood-pressure ; and often increased in size also by false (fibroid) hypertrophy. Hence, in a person, usually beyond middle life, with a feeble circulation and a tendency to blueness of the extremities, if we find the superficial dimensions of the heart increased, the apex more to the left than natural, the dulness extending an interspace higher, and perhaps a finger's-breadth more to the right than is proper, and if on auscultation we find a marked indistinctness of the first sound and an irregularity of beat both as regards time and force, we may be sure of degeneration of the heart, and that the degeneration is more or less fatty.

In advanced cases of fatty heart, cases in which more distinct anginal symptoms may not have occurred, an altered respiratory rhythm is not infrequently to be observed, which is especially apt to occur during sleep; namely, an increasing shallowness of breathing down to absolute cessation for 20, 30, 40 seconds, then renewal of breathing, rapidly deepening to profound and heaving respiratory movements, to subside again gradually to a complete pause (Cheyne-Stokes breathing). During the pause the patient, if sleeping, generally wakes up with a start, and his sleep is thus much interfered with and becomes reduced to a succession of short dozes. The peculiar breathing is to be observed during the waking hours also. The pulse, which is often irregular, continues practically unaltered during the arrhythmic breathing and pause ; it is to be noted, however, that in such cases during ordinary or deep breathing the pulse is distinctly weaker during the inspiratory wave. It must, lastly, be confessed that rare cases are met with in which, even with a marked degree of fatty heart, no signs are discovered up to the moment of fatal syncope or angina. I must state my belief, however, that if the opportunity presents itself for a careful examination of such cases, and the possible presence of emphysema be taken into account as masking an increase of the cardiac area, the clinical evidence of fatty or fibro-fatty degeneration is rarely to be missed.

The disease is most common at or beyond 50 years of age. Men suffer more frequently than women in the proportion of nearly two to one (Quain). All the functions of a person with fatty heart are performed in a languid manner. He is the subject of atonic dyspepsia, with a great tendency to flatulent distension of the stomach ; his bowel and liver functions are torpid ; the urinary excretion, very sensitive to external surface temperature, is of low range of specific gravity, and often contains a trace of albumin. The brain is easily fatigued, the temper irritable. Only gentle level exercise can be taken with comfort.

Treatment.—The treatment of fatty degeneration of the heart due to altered blood-supply is a matter of great importance, hence the necessity of recognising the lesions at the earliest possible stage.

In the earlier stages regular exercise short of fatigue, and adapted to a person in whom a weakness of the central organ of the circulation is recognised, is of importance ; quiet walking on the level, riding (not

hunting), cycling (avoiding hills), driving, motor driving, sailing, quiet rowing, may all be allowed ; and gentle incline walking, adapted to the case, may be taken as prescribed exercise.   Covert shooting may be allowed, but not rough walking or hill shooting.   Golf and croquet are games well adapted to such people.   For these early cases, a course of Nauheim baths and exercises may be taken with advantage from time to time, the exercises being especially valuable in aiding by tonic muscular contractions the return of blood to the heart, disgorging the venous circulation, and thus aiding the forward movement.   Cold bathing should be forbidden, and a warning given to avoid walking against cold winds.

A nutritious diet, rather nitrogenous than fatty or starchy, may be allowed, distributed in three regular meals daily, eaten slowly, and adapted in quantity to the diminished requirements of a less active life.   A moderate amount of wine is usually indicated.

Arsenic, iron, and strychnine are the tonics especially valuable ; but they should not be given in more than two, or at most three doses daily for short courses—the most careful regard being given to avoid digestive disturbances.   In all cases of lowered blood-pressure there is a tendency to passive congestion of the organs and especially of the liver, so that a mild dinner-pill and an occasional mercurial alterative are desirable.

In advanced cases of fatty degeneration the same general plan must be followed still more carefully, and with narrower restrictions as regards exercise, which should only be allowed on smooth level ground, all stair-climbing being strictly forbidden.   The diet must be closely watched, especial care being taken to avoid overloading of the stomach and acute dyspepsia, as many fatal seizures are attributable to gastro-intestinal disturbance.   Persons with fatty heart are extremely sensitive to external cold, and should be clothed very warmly.   A thorough rest, lying down, once or twice a day should be enjoined ; the best times to select are before meals ; a short rest being taken before luncheon, and a more prolonged rest, of one and a half to two hours, before the late dinner.   Such patients should only use warm water for bathing, and for them the Nauheim baths and exercises are not to be recommended.

To a strychnine and arsenic or iron tonic some digitalis, strophanthus, or convallaria may be added with great caution, in cases in which there is undue rapidity or arrhythmia.   An aromatic stimulant and carminative draught should always be at hand in case of syncopal attacks, and may often usefully contain a little nitroglycerin.

Finally, in cases of fatty heart which have advanced to the production of any decided symptoms, the employment of oxygen inhalations two or three times a day is valuable as a cardiac restorative ; it operates principally, no doubt, in stimulating cardiac nutrition and in facilitating the removal of waste tissues from the organ by flushing it with more highly oxygenated blood.   For the Cheyne-Stokes respiration, in advanced stages, there is no more powerful means of affording relief than oxygen inhalations in combination with strychnine.   It should not be employed with the naso-oral inhaler, but a current of oxygen should simply be

played over the mouth and nostrils of the patient for five or ten minutes without any extra respiratory effort on his part ; and in cold weather the gas should be warmed by passing through warm water or through a coil of tubing enclosed in a hot-water tin.

(β) *Fatty Infiltration of the Heart.*—Fatty infiltration of the heart is a condition in which deposition of fat takes place in the interstices of the muscular fibres, compressing them, impeding their action, and causing atrophy.

A certain amount of adipose tissue is naturally present on the heart, especially along the superficial course of the coronary vessels and in the sulci at the base ; under certain conditions this tissue develops in inordinate quantity and spreads over the cardiac surface, penetrating, chiefly with the arterial branches, into its muscular substance. This increase and extension of the adipose tissue is most marked over the right ventricle, and may constitute a layer of considerable thickness which by its encroachment upon and between the muscular fasciculi may cause their atrophy and replacement, and thus considerably weaken and embarrass the heart. The extension is always from the subpericardial surface and chiefly along the arterial lines. The atrophy of the muscular tissue which attends upon fatty infiltration is for the most part consequent and secondary ; but it is probable that in some cases a primary atrophy of the muscle leads to the secondary development of fat in the connective tissue, which is everywhere present and potentially fat-bearing.

Thus, clinically, we have two forms of fatty infiltration of the heart : the one in which the fat is rapidly stored and extends into and encroaches upon a higher tissue, the function of which it embarrasses, and the nutrition of which it mechanically interferes with ; the other in which the fatty tissue merely, as it were, fills up the interstices left by an atrophying muscular tissue.

Of these varieties the first is by far the more common and important. It is met with in persons of inactive and often indolent and self-indulgent lives, in men at middle age, in women towards the climacteric period or soon after it. People who have good appetites and good primary digestion with faulty assimilation and inadequate eliminative power are especially liable to this disease. Indulgence in alcohol, and especially in malt liquors and the sweeter wines, certainly favours its occurrence ; and there are certain maladies upon which it is peculiarly liable to ensue, especially those affections which involve a deprivation of respiratory surface, such as chronic emphysema, or fibroid disease of the lung in old-standing quiescent phthisis, or secondary to pleuritic effusion or unresolved pneumonia. It must be carefully remembered however, that no organic disease of any kind is necessary as the forerunner of this affection, which may arise solely from an excess of alimentary supply over demand, however this may be brought about.

Symptoms and Signs.—Persons thus affected are stout, increasing in weight, with a thickening layer of adipose tissue, full abdomens, and often tender livers. Their circulation is feeble and usually slightly

quicker than it was in former days. There is some excess of venosity
in their colouring, they are short-breathed on exertion, and sweat easily.
Later they manifest functional disturbances of the heart's action, readily
induced on exertion or coming on without it. The cardiac dulness is
increased by an interspace upwards, but the apex-beat is difficult to feel,
and the cardiac impulse is better felt towards the epigastrium. The
sounds are less clear than natural, otherwise unchanged. There is no
change to be felt in the arteries ; the pulse is usually soft, of low pressure,
and, if full, is compressible.   This condition of pulse may be varied by
other intervening states, such as gout, to which, however, these people
are not peculiarly liable.   The urine varies, but is habitually pale and
copious, and rather of low than of high range of specific gravity.

Whilst in a far less dangerous condition than is that attendant upon a
truly fattily degenerated heart, these patients are nevertheless very liable
to succumb to acute illness of any kind, and particularly to bronchitis,
pneumonia, enteric fever, or surgical injury.

The treatment is simple, rational, and, if loyally followed, very successful.
The dietary must be mainly nitrogenous, all superfluous starches, sugars,
and fats being discarded.   Only claret, moselle, or equivalent quantities of
spirit well diluted must be allowed, and in sparing quantity.   The meals
should be at regular times, slowly eaten and strictly moderate in quantity.
But little fluid should be taken with the meal, tissue change being duly
ensured and thirst satisfied by a moderate quantity of hot or cold fluid
slowly sipped about a quarter to half an hour after the meals, or, some-
times, better still, half-way between the meals.   A tumbler of hot water
with a little fresh lemon-juice may be taken at bedtime or in the early
morning.   Fresh lemon-juice instead of milk in the tea is useful.
Raw fruits, root vegetables, and bread must be avoided, or only
very sparingly taken.   Daily walking, riding, or cycling exercise must
be imperatively enjoined ; for the advantage of regulated exercise is
not merely to quicken muscular nutritive changes, and so to convert
the food taken into proper force-yielding material, but to deepen respira-
tion and to promote the respiratory and other eliminative functions.
Hence dumb-bell, fencing, or other home exercises carried on indoors,
although they may be useful supplementary aids, are not adequate to
replace open-air exercise.   Medicinal treatment is of quite minor import-
ance, and may be limited to promoting due elimination, and giving a
heart tonic if needed.   Turkish baths, or a course at Homburg, Carls-
bad, Marienbad, Harrogate, or Nauheim, may be suggested in appropriate
cases.

The other form of fatty infiltration attendant upon atrophy of the
heart is met with in an altogether different type of individual, one who
commonly is already the subject of some grave organic disease, such as
tuberculosis or cancer ; and its importance and treatment are both merged
in the more general malady.

(γ) *Fibroid Infiltration of the Heart* (Fibrous transformation, Coats ;
Fibroid degeneration of the myocardium, Orth ; Myocarditis productiva

or interstitial myocarditis).—This condition essentially consists in the separation and replacement of the muscular fibres of the heart by an imperfect fibrous tissue generated by overgrowth of the connective tissue of the organ. It is very closely analogous to fatty infiltration, and it cannot be rightly described as a degeneration of the myocardium. It would seem, therefore, that the term "fibroid infiltration" most fitly describes the morbid state present; interstitial myocarditis is also a fairly accurate term, although it conveys the impression of the disease being necessarily of an inflammatory origin.

Fibroid infiltration of the heart may be described as general and local, although even in general infiltration the disease is not uniformly distributed.

Causes.—Besides the coronary obstruction, general fibroid infiltration has another principal cause; namely, chronic congestion of the heart from mechanical impediment to the return of blood from the cardiac veins. This cause is chiefly met with in cases of old-standing emphysema, and in cases in which the whole or a large portion of one lung is the seat of cirrhotic change from old pleurisy, unresolved pneumonia, or fibroid phthisis. Extensive narrowing and destruction of pulmonary vessels and impairment of that inspiratory aid to the cardiac circulation which obtains in healthy respiration, result in a difficulty in the pulmonary circulation, at first overcome by greater diligence of the right heart, but gradually increasing until the venous return to the right auricle is seriously impeded. A chronic congestion of the walls of the heart ensues, most marked on the right side, but involving the left also; and, as a result of this chronic congestion, overgrowth of connective tissue and atrophy and degeneration of the cardiac muscle proper. In the more advanced stages of mitral stenosis and regurgitation the same conditions are to be observed, having similarly a mechanical origin. Bunting has collected some evidence as to the occurrence of chronic fibrous myocarditis in progressive muscular dystrophy, and inclines to the conclusion that the changes in the voluntary muscles and in the heart are allied.

Nevertheless the most important cause of general fibroid infiltration of the heart is the obstructive disease of the coronary arteries at or near their origin from the aorta, under which head we now consider it. A more marked degree of fatty degeneration of the muscular fibres is met with in association with fibroid infiltration arising from this cause, for a degree of blood irrigation which will suffice for an overgrowth of an inferior connective tissue, will not suffice for the nutritive maintenance of a tissue of such activity and reparative requirements as muscle.

It is thus to be remarked that hearts which are the seat of general fatty degeneration from coronary obstruction (except quite as a senile change) are always large hearts, the seat of so-called false hypertrophy; and this it is which furnishes us with an important clue to their clinical diagnosis. The increase in size is partly due to increased thickness of the cardiac walls, in part to dilatation of the cavities of the heart; for fibroid infiltration, although it increases the toughness of the cardiac wall,

diminishes its resilience and contractile power ; hence a gradual yielding to the blood-pressure, each stage of which is permanent.

Fibroid infiltration as a local affection of the heart arises from—1. Local obstruction to the circulation, due to local plaques of thickening, and degenerative constriction of the coronary branches. The heart's substance may be the seat of innumerable patches of greyish - white fibroid infiltration from this cause, or there may be one or two such patches of larger dimensions corresponding with the territory of a larger branch.

2. Corresponding with well-marked patches or "scars" in the heart's substance there will often be found a complete occlusion of a coronary branchlet from thrombosis or embolism, and in an earlier stage the more distinct signs of an infarct may be seen.

3. It is very possible that some of the heart "scars" which are found may be due to a fibrous repair of partially ruptured fibres. This view derives increased probability from the results of Pearce's experiments upon the heart with adrenalin (*vide* p. 124).

4. An extensive, although usually superficial fibrous infiltration of the heart may ensue upon pericarditis and adherent pericardium, the change beginning in the subpericardial tissue and extending more or less deeply into the muscular interstices of the heart. Such changes are started by direct inflammatory irritation, and are often accompanied with a certain degree of fatty infiltration.

5. Certain cases of acute myocarditis are, as will be presently seen, followed by chronic interstitial myocarditis.

Pathology.—The minute pathology of fibroid infiltration of the heart is the same, other things being equal, as that of the same process taking place in any other organ ; that is, it begins with a proliferation of the nuclei of the connective tissue, so that in the earliest stage, rarely observed except at the margins of extension, areas or groups of crowded nuclei are to be seen which are gradually transformed into fibres ; these again in their turn, losing their characters, form dense areas of wavy, glue-like interlacing processes, entangling a few nuclei. In the denser portions the muscular fibres of the heart are completely replaced or destroyed, or only appear as small islets of a few isolated fibres ; and towards the circumference of any local patch the muscular fibres are observed to present broken or atrophied terminations, and to be more or less widely separated by the intruding tissue. Here and there streaks of pigment granules may mark the site of destroyed muscular tissue.

The process of fibroid infiltration must by no means be regarded as in all cases a destructive lesion ; on the contrary, it is in most instances the result of an effort at repair. This is most distinctly the case in heart "scars," where the necrosed muscle, infiltrated with blood elements which constitute an infarct, is gradually removed by absorption and replaced in the only possible way by the growth of a living but inferior tissue, which serves the purpose at least of healing the breach and giving mechanical support to the heart-wall. And, rightly regarded, the fibroid

infiltration more generally dispersed through the heart substance in cases of retarded or restricted circulation is the means of maintaining the resistance of the ventricle walls to the blood-pressure, a conservative effort, although attended with but poor and temporary success.

In cases of local fibroid infiltration reparative of necrosing infarcts, the scars sometimes become infiltrated with lime salts, and grate under the knife on section.

Symptoms and Signs.—The symptoms of general or extensive fibroid infiltration of the heart are those of chronic heart-failure, and difficult to distinguish from those of fatty heart, with which, as already observed, the disease is often associated. The patient, usually fifty or upwards, and more commonly a man, has for some months been aware of scantness of breath, and of oppressed feelings about the heart on exertion ; but he has become accustomed to this, and the first symptoms compelling his attention, and leading him to seek advice, generally supervene quite suddenly. During some accustomed or slightly increased effort — the walk home from business or an extra round at golf, or a tramp with the gun over a turnip-field or up a sharper hill than usual—he is seized with severe breathlessness and oppression at the heart, which compel him to stop and rest for a time and to get home very quietly for fear of a further attack, of which he has some dread. The first attack may amount to a distinct anginal seizure (see " Angina Pectoris," Case 4, p. 167). After this experience his cardiac power is never on the same level as before, and often deteriorates rapidly. His breathing fails him on slight exertion, he becomes liable to dyspnoea on slight distension of the stomach, his face becomes somewhat puffy and dusky in colour, he is apt to be awakened at night with more or less urgent dyspnoea and wheezing, which he regards as asthmatic. The ankles and legs become puffy and oedematous, and finally he is confined to his room and chair on account of the constant and readily increased dyspnoea.

On physical investigation the fibroid heart is always found to be associated with other conditions in the same plane of degeneration, and which therefore help to point to the diagnosis. Thus in extreme emphysema, in the later stages of Bright's disease, as well as in the early manifestations of cardiovascular degenerations associated with gout, intemperance, and syphilis, we often find fibroid infiltration of the heart as a factor of importance in the illness of the patient.

Diagnosis. — Having indicated sufficiently, therefore, the general symptoms which may be attributed to this state of the heart, I may briefly add the salient points of physical diagnosis. In the majority of cases there is evidence of degenerative thickening of the vessels generally. The systemic vessels are wanting in elasticity, and more or less thickened ; the radial artery is more thick and palpable than natural : the pulse is not as a rule quick, it may be regular, but often it is irregular in force and rhythm ; the pressure varies, but is not high unless it be raised by some other disturbing condition. In cases in which the cardiac state is secondary to emphysema, mitral stenosis, or adherent pericardium,

there may be no arterial thickening; and the pulse is feeble, vacillating, or compressible. Indeed, it will often interest the clinical observer to contrast the big labouring heart, with no important valve lesion to waste its force, with the small output at the wrist vessel. The dimensions of the heart are increased in all directions, the apex-beat is extended beyond the line of the left nipple, the upper margin of dulness is raised to the third space or cartilage, the right margin of dulness extended to the median line or a finger-breadth beyond it. The size of the organ varies with the stage of the disease, but it is always increased considerably by the time the patient complains of symptoms. In cases having their origin in cardiac congestion from emphysema or mitral disease, the evidences of enlargement of the right side of the heart are most considerable, the extended impulse is most apparent towards the ensiform cartilage, and the dulness to the right of the sternum. The presence of emphysema tends to mask the percussion and palpation signs very considerably, and must therefore be taken into careful account. The cardiac impulse, although somewhat heaving, has notably less of the thrusting quality than would obtain over a heart of anything approaching to similar dimensions from pure muscular hypertrophy; it is also more generally diffused over the cardiac area. In cases of difficulty in defining the limits of the cardiac outline by palpation and percussion, auscultation, using a stethoscope with a small chest-piece, may be usefully employed. There is not necessarily any marked alteration in the sounds of the heart, but the first sound at the apex is always longer, duller, and less defined than normal, and it is often attended by a soft murmur; whilst the first sound at the base is barely audible, and the second sound there is dull, muffled, and prolonged. In mitral cases, however, the second sound over the pulmonary area may be strongly accentuated, although duller and less acute than in the earlier stages of the valve disease.

There is frequently some albumin in the urine, especially in the later stages; and other evidence of visceral congestion from retarded circulation, such as occasional congestion at the bases of the lungs, fulness of the liver, and the phenomena of slow digestion with flatulence and loaded urine. With increasing failure of cardiac force the urine falls in amount, and dropsical phenomena set in.

Treatment.—The treatment of fibroid degeneration of the heart is best considered under the diseases—emphysema, angina pectoris, and failing compensations in cardiac lesions, into the symptomatology of which it enters as an important factor.

(δ) *Aneurysm of the heart.*—Aneurysm of the heart is a rare condition, and one still more rarely clinically recognisable. It is questionable whether all the cases recorded by Heschl and Willigk are cases of true aneurysm. The left ventricle is almost exclusively affected and most commonly (in 59 per cent) at the apex; occasionally the septum between the ventricles is the seat of aneurysmal bulging. The pouch varies in size from that of a filbert to that of a large cocoa-nut; it is lined by stretched endocardium, and contains laminated clot and more recent coagulum.

Local destruction of the muscular fibre from any cause may lead to aneurysm. Local softening, consequent on disease or occlusion of a branch of a coronary artery, is commonly responsible for acute aneurysm. Circumscribed suppurative myocarditis is another cause of it. Chronic fibrous myocarditis disposes to aneurysm when the heart-wall is thin, not when it thickens.

Dr. Wickham Legg attributes such aneurysms to fibrous degeneration of the heart-muscle, and points out that while there is abundant evidence that this degeneration is commonly due to impaired coronary circulation, there are yet many cases of aneurysm of the heart which occur in people under forty years of age, in whom the coronary arteries shew no change, so that he doubts whether coronary obstruction is responsible for the myocarditis in all cases. Hilton Fagge regarded fibrous myocarditis as the cause of chronic aneurysm in almost all cases.

The tendency is for the sac to rupture into the pericardium, causing death. In other cases death occurs from mechanical interference by the sac with the movements of the heart. Spontaneous calcification and partial obliteration of the sac may result.

(ii.) **Thrombosis, Embolism,** and (iii.) **Aneurysm of the coronary arteries** require but brief notice. The symptomatology and diagnosis of thrombosis and aneurysm are for the most part included in the phenomena arising from atheroma of the vessels, whilst embolism is a rare affection, and difficult to recognise during life.

Thrombosis of the coronary arteries is a frequent result of previous atheromatous change and is also occasioned by specific arteritis. Any portion of the vessel already thickened and narrowed by atheromatous change may thus become more or less suddenly and completely occluded by coagulation. Thrombosis may occur at any portion of the coronary arteries, but is most frequently met with near their origins from the aorta for the reason that these portions are the most common seats of extensive atheroma. When it occurs deeper in the heart it is often associated with gummatous arteritis.

It is to be borne in mind that although it has been shewn by Wickham Legg and West, contrary to the opinion previously current amongst pathologists, that there is at least some intercommunication between the peripheral distribution of the two coronary vessels, yet this communication is very restricted, and the effect of a complete closure of one of the coronary arteries in any part of its course is to produce anaemia of the territory beyond. Fringing the anaemic area and encroaching upon it is a line of congestion or partial capillary stasis; but there is no filling up of the area with blood so as to form the damson cheese-like appearance of recent infarcts in more vascular tissues. The yellowish tinge of the area is that natural to anaemic muscle. A softening from fatty change and molecular necrosis of the area follows, and haemorrhages may occur into the softened area. Microscopically the muscular fibres are found broken up, their transverse striae are lost, and the remains of the fibres have assumed a hyaline or waxy appearance (Coats).

The area of congestion surrounding the infarct becomes the seat of more or less inflammatory reaction, attended with the usual proliferation of connective tissue and infiltration with leucocytes. The softened area wastes (falling below the surface on section), and gradually undergoes contraction by encroachment of fibroid growth extending from its periphery, the semi-liquefied tissues becoming slowly absorbed; the final result being a heart scar of dimensions varying with the size of the original infarct. In cases, however, in which the softened territory is of considerable dimensions, the branch occluded being large, it may yield before the blood-pressure to form an acute aneurysm of the heart.

The result of a partial occlusion of the coronary artery by thrombosis or atheroma has already been described, namely, a fibrous transformation of the corresponding territory; and, in cases in which the complete occlusion of the vessels is slowly effected, the same result is produced.

Embolism of the coronary arteries may occur under any of the conditions which occasion embolism of other systemic vessels; but the situation of the vessels at the commencement of the aorta, the wide angle at which they leave the vessel, and the bulk and impetuosity of the blood-current at this portion, are all conditions unfavourable to the passage of clot into these small side arteries.

The emboli may be of the ordinary fibrinous character, or septic as in cases of infective endocarditis. It is quite possible for débris from a softening atheroma of the main coronary trunks to be conveyed onwards to occlude some of their terminal branches.

Symptoms and Signs.—The symptoms of sudden occlusion of a considerable branch of the coronary artery generally begin with an anginal paroxysm which may be fatal at once. In cases in which the first seizure is survived, the subsequent phenomena are those of rapid heart-failure, dyspnoea with acute anginal paroxysms, rapid and more or less irregular heart's action, dilatation of the organ to the right or left according to the ventricle affected; systemic and pulmonary oedema are also correspondingly predominant. These acute phenomena almost invariably supervene upon chronic heart difficulties already ascribed to degenerative changes, and more or less quickly close the scene. Even in the rare cases of embolism of the coronaries there have generally been previous signs of acute or chronic endocarditis, usually of the aortic valves. W. T. Porter of New York conclusively shewed by some elaborate experiments in 1896 that ligation or occlusion of the coronary arteries causes feeble incoordinate or fibrillary contraction and arrest of the heart's action, and that, taking them singly, the largest vessel—the circumflex—if ligatured or blocked, produces arrest in 64 per cent, the descendens in 28 per cent, the right coronary in 14 per cent, and the arteria septi in no cases.

Aneurysm of the coronary arteries is a disease the secondary effects of which upon the cardiac muscle are of less importance. The coronary arteries may shew multiple aneurysms due to embolism. The coronary arteries are affected in nearly all the cases of the rare condition poly-

arteritis acuta nodosa, and then present the appearance that might be attributed to multiple aneurysms (9).

III. Impairment due to Senile Changes: Pigmentary Degeneration ; Atrophy.—(a) **Pigmentary Degeneration.**—This is a condition seen in nearly all people above the middle period of life, but the change is not met with in the voluntary muscles (Wilks and Moxon). The heart weighs less than normal ; it is hard and tough, and the muscle-fibres are a dark chocolate colour. The pigment itself consists of haematoidin granules of a reddish-yellow colour collected about the nuclei of the muscle-fibres. Atrophic changes usually accompany the pigmentation, though the striation of the fibres is not much altered. This condition is also met with in any general emaciation (Wilks and Moxon) ; it does not seem to impair the functions of the organ.

(b) **Atrophy of the Heart.**—Atrophy of the heart may be part of general wasting, as in old age or chronic disease. It may become reduced in weight—from 9 oz. in woman, or 10 or 11 in man, to 6 or 5 oz.,—drier in texture from loss of fat and fluid, and darker in colour from the accumulation of pigment granules about the nuclei of the muscular fibres. Local or general atrophy may result from impaired circulation in tortuous and diseased vessels ; but under these conditions, except perhaps in old people, the muscular atrophy is attended with the overgrowth of another tissue—the connective. Fatty and fibroid infiltration are both attended with more or less separation and atrophy of the muscle proper.

IV. Impairment of the Heart from Functional Strain requires little more than a reference here, since the forms it assumes are discussed elsewhere (p. 193).

Functional strain, resulting in hypertrophy, may be due to the prolonged endeavour of the heart to overcome some increased resistance to the circulation, or to compensate some defect in its valve mechanism. Undue rigidity of the vessels, generally from atheromatous changes, chronic heightening of the arterial blood-pressure, as in Bright's disease, obstructed circulation through the lungs, aneurysm of the main vessel, disease of the different valves of the heart, or congenital alteration of one of the orifices, are amongst the chief causes leading to hypertrophy. The hypertrophy thus occasioned cannot be strictly spoken of as disease of the heart ; it is rather an attempt—for a time successful—to compensate a pre-existing defect ; and the portion of the heart affected is dependent upon the seat of obstruction in the circulation.

Hypertrophy of the heart, secondary to obstructed coronary circulation or pericardial adhesions, is not real hypertrophy at all, but a thickening of the organ due to changes (mostly fibroid) secondary to chronic interstitial myocarditis.

Idiopathic or simple hypertrophy is a condition of muscular overdevelopment from excessive cardiac exercise. Sir Clifford Allbutt has

described changes in the heart ensuing upon prolonged muscular exertion, such as hill-climbing, athletic exercise, and the like. Perhaps the best-known example of alleged simple hypertrophy was that recorded by Professor Haughton in the case of the celebrated greyhound, "Master Magrath"; but veterinary surgeons are not yet agreed whether simple cardiac hypertrophy is found in horses and other labouring animals (*vide* p. 430).

Bollinger recorded forty-two cases of simple hypertrophy without valvular disease—thirty-eight men and four women—in which the hearts were one-third heavier than in health. The observations were made at Munich, and Bollinger considers the great consumption of beer in that city as the chief cause of the hypertrophy, producing its effects (*a*) through the toxic effects of the alcohol; (*b*) by the quantity of liquid taken into circulation; (*c*) by increased nutrition. A certain amount of dilatation accompanies or succeeds to the hypertrophy. Sometimes the right ventricle is especially affected. Jürgensen and Schroetter, whilst admitting that excessive beer-drinking may induce the changes in the heart referred to, yet point out that the habit is much associated with other causes of physical strain, such as duelling and fencing and other excesses involving want of sleep, and over-smoking. It is to be noted, however, that amongst wine-drinkers, given probably to the same excesses, these forms of heart disease are less frequent (*vide* also article on Mitral Incompetence, p. 413). The view that the heart (left ventricle) hypertrophies towards the end of pregnancy was first put forward by French accoucheurs. German obstetricians denied this. Macdonald upheld the view in this country, and Hamilton's observations confirm the French view. The probable cause is the increased work the heart has to do in driving blood through the enlarged uterus (Hamilton); it has also been attributed to a toxic state of the blood.

Acute strain of the heart may mean either acute over-distension or acute over-function.

In the first case, under sudden accession of the blood-pressure chiefly arising during great effort, especially when associated with some obstructive valve defect, such as aortic or mitral stenosis, the portions of the heart most concerned may become over-distended to the suppression of their function. Sudden death may ensue from complete cessation of the heart's action, or a grave embarrassment, threatening death, may only be averted by a timely bleeding. Again, an obstructed function, less in degree, may be to a certain point recovered from, but leaves the heart temporarily or permanently strained. What precisely does this mechanical strain of heart mean? With what changes in the myocardium is it associated?

In the St. George's Hospital Reports, 1870, and in a previous paper read before the British Medical Association in 1869, Sir Clifford Allbutt describes the effects of overwork and strain on the heart and great blood-vessels, especially to be observed amongst such hard labourers as forgemen, colliers, wharfingers, etc. He also relates some cases

illustrative of the earlier stages in which, after excessive exercise in mountain-climbing, hard gymnastics, and rowing respectively, signs of dilatation from acute overstrain are followed by those of hypertrophy of the heart. Sir Clifford Allbutt considers the sequence of events to be as follows :—(i.) Dilatation of right heart ; (ii.) dilatation of left heart ; (iii.) hypertrophy of one or both ventricles ; (iv.) chronic inflammatory endarteritis of the aorta ; (v.) dilatation of the aorta ; (vi.) incompetency of the aortic valves ; (vii.) further left ventricle hypertrophy compensating aortic defect ; (viii.) degenerative changes ensuing upon hypertrophy. (*Vide* art. " Over-stress of the Heart," p. 210.)

Mr. Myers in 1870, in a paper on " Diseases of the Heart among Soldiers," drew attention to the effects of prolonged exertion in tight-fitting uniforms, and especially whilst wearing the tight breast-strap, in producing cardiac and aortic diseases from overstrain.

Da Costa has described a condition of " irritable heart " as of very common occurrence in soldiers during the fatigues of a campaign, and observed by him especially amongst the soldiers in the American Civil War. A persistently quick action of the heart, with precordial and left shoulder pains, and bouts of severe palpitation under slight exertion, or digestive disorder, are the principal symptoms. At first these are unattended with any notable physical signs, and they may subside without such signs ; but in the cases of greater severity or longer duration there is obvious enlargement of the heart. The pathology of these cases would no doubt be for the most part the same as that described by Sir Clifford Allbutt, namely, a chronic myocarditis ensuing upon dilatation and mingled with muscular hypertrophy ; but probably there is also some direct damage to the cardiac nerves, originating at the terminals of the vagi and sympathetic. (*Vide* Soldier's Heart, p. 235.)

V. Impairment of Inflammatory Origin.—(*a*) **Interstitial Myocarditis.**—Myocarditis most generally consists of an irritative overgrowth of the interstitial connective tissues of the heart, which may extend from a pericarditis or an endocarditis. In its first stages an increased nuclear proliferation, permeating the muscular fibres, causes a " cloudy swelling " of the tissue, and a certain degree of increased softness to the touch, but the later result is more or less fibrous toughness of the part involved.

Disturbed circulation, general or local, will occasion cardiac fibrosis ; such as chronic congestion of the cardiac veins, or restricted or obstructed circulation through the coronary vessels. The cicatricial or tendinous patches of the heart are produced by interstitial myocarditis. An impairment even to destruction of the true muscular fibres of the heart necessarily ensues upon local or general fibrous myocarditis. Charlewood Turner pointed out that interstitial myocarditis may exist and extend apart from any affection of the pericardium or endocardium, and that in cases of dilatation of the heart or failing hypertrophy, from whatever cause, this morbid process is at work and responsible for further changes.

Experimental Myocarditis.—Light is thrown upon the pathology of some cases of acute and chronic myocarditis, and especially those cases which have their origin traceable to overstrain of the cardiac muscle, by the experimental observations of R. M. Pearce upon the myocardial changes that result from the intravenous injection of adrenalin. In a series of examinations of rabbits, 36 in number, after intravenous doses of adrenalin, he found degrees of myocardial change varying according to whether the animals died immediately (14 cases), or at various intervals after receiving seven to fifteen injections (22 cases). In those animals which died immediately the hearts were greatly dilated, the muscle of the left ventricle very pale, the auricular walls venously congested. The myocardium of the walls of the ventricles and the papillary muscles were oedematous, markedly so in areas some of which shewed ruptured bundles of fibres. The muscular fibres themselves appeared oedematous. In animals that survived seven to fifteen injections (usually given in doses of $\frac{1}{10}$ to $\frac{8}{10}$ c.c. of 1 in 1000 solution each second day) fibrous transformation was found, especially in the areas corresponding to those of acute oedema. The aorta in each series shewed focal necrotic lesions of the middle coat, in the more chronic cases becoming plaques of degeneration. Pearce attributes these effects to the mechanical strain upon the heart-muscle arising from the raised blood-pressure due to arterial constriction, and also in part to the contractile action of the drug upon the arterioles of the heart, causing for a time a direct interference with the nutrition of the areas affected. These purely experimental observations may have some bearing on human cardiac pathology in so far as they illustrate how a myocarditis may arise from cardiac muscular strain. They point also to the necessity of caution in the use of a drug which in excessive doses may bring about such serious myocardial changes. They have a bearing too, which may be speculative, on the interpretation of the chronic myocardial changes that ensue in chronic nephritis, which are brought about by the raised arterial pressure which is at first a compensatory or defensive condition induced and maintained by an enhanced adrenal function.

I have already fully described chronic interstitial myocarditis under Fibroid Infiltration, p. 115.

(b) **Parenchymatous Myocarditis,** which is met with in certain cases of septic poisoning, such as pyaemia and diphtheria, is probably, in its earliest stages, but a very active form of the preceding process. Leyden regards it as an acute myocarditis characterised by intermuscular nuclear proliferation and by secondary atrophic changes towards necrosis and deposition of pigment ; fatty degeneration of the muscular fibres accompanies it, possibly in consequence of the inflammatory changes. This form of myocarditis is always secondary to infective fevers, such as diphtheria, scarlet fever, and the like ; and has been met with in greater or less degree in all acute febrile diseases, rheumatism, cerebrospinal meningitis, variola, erysipelas, malaria, septicaemia, influenza, and so forth. In enteric fever and in gonorrhoea the respective specific bacilli and cocci have been found in the heart.

(*c*) **Purulent myocarditis** is in most cases secondary to infective embolism of the coronary vessels, as in pyaemia and infective endocarditis. In all cases microbes are conveyed to the cardiac muscle through the coronary arteries, and set up foci of virulent myocarditis resulting in minute or larger suppurations.

(*d*) **Syphilitic Myocarditis.**—Syphilitic myocarditis almost invariably occurs either in the immediate neighbourhood of a gumma or secondary to and in the territory commanded by a specific arteritis. Attention was first drawn to the occurrence of syphilitic lesions of the myocardium by Sir Samuel Wilks in 1856 ; and many isolated cases have been reported since at the Pathological Society of London and in various English and foreign medical journals. Our knowledge of the disease, however, is mainly derived from the post-mortem observation of cases in patients, by no means all of whom died with heart symptoms.

Syphilitic disease may affect the myocardium in one of the three following ways, and either singly or combined :—

(*α*) There may be syphilitic arteritis and secondary or combined chronic myocarditis. Thrombosis may ensue upon the arteritis resulting in anaemic necrotic change, softening and ultimate fibrosis of the muscular area involved ; provided an anginal attack do not carry off the patient.

(*β*) There may be gummatous formation in the heart-wall, around and extending from which chronic myocarditis takes place. Gummatous formations may occur in any part of the heart, most commonly in the ventricles or septum. They have the usual features and ill-defined microscopic characters of gummas elsewhere ; they may soften, or undergo fibroid change, and they are always surrounded by more or less spreading fibroid condensation of the heart-wall from associated chronic myocarditis.

(*γ*) There may be a diffused chronic myocarditis of specific nature affecting a considerable portion of the heart. It is doubtful, however, whether this latter form of diffused syphilitic myocarditis does not originate in the fusion of scattered gummatous depositions.

It cannot be said that any symptoms have yet been formulated which in their grouping or individual significance are characteristic of syphilitic disease ; and for the obvious reason that very different portions of the heart may be affected in different cases, and that the upshot of the morbid condition in each case is a spoiling of the cardiac muscle at the part affected, and more or less interruption or spreading disturbance of the cardiac mechanism therefrom.

(*e*) **Segmentation and Fragmentation of the Heart-Fibres.**—The *état segmentaire* first described by Renaut in 1877 as a form of myocarditis in which the heart-fibres separate by a process of softening along their cement lines has given rise to much research and discussion. Some observers, as Oestreich, regard the so-called segmentation as really for the most part fragmentation or rupture of the muscle-fibres and as occurring as a part of the death agony in many diseases. Von Recklinghausen also regarded the condition as one precedent to rigor

mortis and as especially associated with violent or sudden death.   Israel
likewise looked upon the condition as one of cadaveric change.   Renaut
and Aufrecht, however, maintain that segmentation is a definite disease
giving rise to symptoms of cardiac hypertrophy and dilatation and heart-
failure with dropsy.   Hektoen in an elaborate essay upon the subject,
including references to the literature up to 1897, agrees with Renaut in
regarding the segmentation of the heart-fibres as a condition distinct from
their breakage, although the two may be associated.   He fairly sums
up the subject by regarding both conditions as episodal in the course of
many diseases, including general infectious cardiac dystrophies, traumatic
and other fatal affections.   Finally, Krehl omits all mention of segmen-
tation and only alludes to fragmentation as possibly an agonal change.

In estimating the *clinical phenomena* dependent upon the changes
wrought by myocarditis we must not forget the hidden ancestry of the
cardiovascular system, and that the heart, although in the course of
evolutionary changes highly differentiated, has not altogether lost its
primordial intrinsic function of rhythmic contraction, a function which,
although in a great measure now subject to the guidance of the nervous
system, is by no means wholly dependent upon extrinsic nervous impulses.
Whilst in health this measure of automatic mechanism secures a great
saving of energy ; in disease, on the other hand, it is very conceivable
how myocardial changes of inflammatory or other source should bring
about disturbance of cardiac rhythm of a very grave kind and but little
amenable to outside nervous control.   This view furnishes us with a
better insight into the cardiac defects from myocarditis than is otherwise
attained.   The two cardiac phenomena significant of myocardial change,
apart from valvulitis, are dilatation which is a simple yielding of the
weakened muscular walls before the blood-pressure, and irregular action
which is an arrhythmia of contraction of the muscle from incoordinate
action of parts no longer possessing their original homogeneity, and
presenting positive interruptions or blockings of conductive paths or
areas.

If the myocarditis affect chiefly or solely some portion of the bundle
of His at its source or in its continuity, direct heart-block phenomena
may ensue.   If, again, different areas or segments of the ventricle
walls be involved in the inflammatory lesion, the rhythm of contractile
impulse may become broken up or disarranged.   It is in this regard
that the anatomical and physiological observations of Gaskell, Keith,
and Tawara upon the heart, and their clinical interpretations by Dr.
James Mackenzie, have been so invaluable in revealing the true signifi-
cance of those structural differences in the heart that were recognised
more than fifty years ago by Kölliker and others (see article on Stokes-
Adams Disease, p. 130).

Functional irregularity, anginal seizures, syncopal attacks, any of
which may prove fatal, are amongst the most common symptoms.   It is
remarkable that sudden death has terminated a large proportion of the
recorded cases of gumma of the heart, in most instances without any

previous recognition of the disease. The first case recorded by Sir Samuel Wilks ended in death this way, as did 14 out of 25 cases more recently collected by Dr. S. Phillips. Enlargement of the heart, or displacement of the apex-beat to the left, or more marked evidence of enlargement to the right, are amongst the later signs ; especially in cases of the more diffused form of syphilitic myocarditis.

The absence from the history of the case of rheumatism, of gout, of alcoholism, or strain ; and evidence—whether in the form of a distinct history or of collateral lesions of a specific kind—pointing to a syphilitic cachexia, are circumstances which, in the presence of such signs and symptoms of cardiac disease, may lead us to suspect its syphilitic nature, and direct our treatment to that probability. When the signs positive and negative with regard to such heart diseases above referred to are found in men, and before middle life (nicotine poisoning being also excluded), an additional argument in favour of syphilis will be found. The success of antisyphilitic measures of treatment, which, however, would be combined with cardiac tonics, strychnine, digitalis, iron, or arsenic appropriate to the case, would further help the diagnosis.

VI. TUMOURS OF THE MYOCARDIUM.—The heart is one of the organs least commonly affected by new growths. Hektoen in 1893 mentions about 110 cases of tumours of the heart, recorded up to date in the Index catalogue of the Surgeon's Office at Washington and the *Index Medicus*. Berthensen in the same year records 30 published cases of primary new growths of the heart, namely, sarcoma 9, myxoma 7, fibroma 6, carcinoma 3, lipoma 2, syphilitic 2, cyst 1. Karrenstein in 1908 quotes 39 cases other than myxoma, probably many of them the same as Berthensen's, the varieties being in much the same proportion. The ages of Karrenstein's cases are fairly evenly distributed between infancy and old age. As regards sex, the males greatly predominate, as 21 to 5. He also gives statistics of 38 cases of myxoma, the ages of which are likewise about evenly distributed. In these latter cases, however, the sexes are almost evenly represented—16 males to 17 females, and 5 not stated. The left side of the heart and the septum were the parts mainly affected, accounting for two-thirds of the cases. Wolbach has collected 12 authentic cases of congenital rhabdomyoma of the heart, all in infants or children under four years of age. Six of the cases also shewed diffuse cerebral sclerosis. These statistics may approximately represent the proportion in which the different kinds of growth occur in the heart, with the exception that syphilitic growths, rarely, it is true, amounting to tumours, are of far greater frequency and importance than would here appear. Tuberculosis, common in the pericardium, is rare in the heart substance, and then occurs almost exclusively as an accompaniment of generalised tuberculosis. Secondary growths are not common in the heart. Round-celled sarcoma is occasionally met with, extending apron-like over the pericardium, greatly thickening it, and embedding the great vessels yet not invading the heart itself (p. 101). Malignant

disease of the lung and mediastina frequently invades the parietal pericardium in cauliflower-like excrescences, and yet spares the heart. Sarcoma sometimes invades the heart from the mediastina along the sheaths of the coronary vessels and their ramifications, penetrating into the intermuscular tissue and separating the cardiac fibres, causing them to atrophy (Boyce).   Secondary malignant growths, both carcinoma and sarcoma, have been met with in the substance of the heart at post-mortem examinations ; secondary melanotic growths are not uncommon in cases of generalised melanosis ; in 1897 Drs. Calvert and Strangeways Pigg analysed the cases reported in the Transactions of the Pathological Society of London in order to determine the relative frequency of secondary melanotic growths in the various organs ; they found the liver most often invaded, in 19 cases, the heart next in 13 cases, then the lungs in 12, the kidneys in 9.

VII. PARASITES.—Hydatid is rarely met with in the heart of the human subject, although a good number of cases may be found in literature.   Peacock and Knaggs have each recorded a case of sudden death consequent upon a considerable hydatid tumour in the walls of the heart, which had not ruptured.   Peacock classified the recorded cases of hydatid of the heart into (1) those in which there had been no symptoms of heart disease during life ; (2) those in which symptoms of valvular disease were occasioned by the presence of the tumour ; (3) those in which impaction of the orifices causing fatal obstruction of the circulation had arisen from rupture of the hydatids.   Pericarditis or rupture into the pericardium may also be occasioned.   The cysticercus of *Taenia solium* is common in the heart of swine, and that of *Taenia saginata* in cattle ; but they are rare in man.

Actinomycosis may extend to the heart from the mediastina and lungs.

Trichinosis (*Trichinella spiralis*) is never, or extremely rarely, found in the heart (Wilks and Moxon).

<div align="right">R. DOUGLAS POWELL.</div>

REFERENCES

I. Impairment Secondary to General Blood Conditions. (a) **Anaemia** ; (b) **Toxic Changes** : 1. COATS, J.   *Manual of Pathology*, 3rd edit., London, 1895, 435.—2. DRESCHFELD.   *Brit. Med. Journ.*, 1905, ii. 1023.—3. HAMILTON, D. J.   *Textbook of Pathology*, London, 1889, i. 581, 588.—4. MOLLARD et REGAUT.   *Ann. de l'inst. Pasteur*, Paris, 1897, 97.—5. SCHROETTER.   "Diseases of the Heart Substance," Ziemssen's *Cyclopaedia of the Practice of Medicine*, London, 1876, vi. 246.—6. ZIEGLER.   *Lehrbuch der spez. patholog. Anatomie*, 11te Aufl. Bd. II., Jena, 1906, 1. **II. Impairment Secondary to Altered Blood-Supply :** 7. BUNTING, C. H. "Chronic Fibrous Myocarditis in Progressive Muscular Dystrophy," *Am. Journ. Med. Sc.*, Phila., 1908, cxxxv.—8. COATS, J.   *Manual of Pathology*, 3rd edit., London, 1895, 427.—8a. COWAN.   "The Fibroses of the Heart," *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii. 209.—9. DICKSON, W. E. C.   "Polyarteritis Acuta and Periarteritis Nodosa," *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii. 31.—10. LEGG, J.W. *Cardiac Aneurysms* (Bradshaw Lecture, 1883), London, 1884.—11. PORTER, W. T. *Journ. Exper. Med.*, N.Y., 1896, i. 296.—12. STEVEN, J. L.   "Fibroid Degeneration and Allied

Lesions of the Heart, and their Association with Disease of the Coronary Arteries," *Lancet*, London, 1887, ii.1153; also *Journ. Path. and Bacteriol.*, Edin. and London, 1894, ii. 190.—13. TURNER, F. C. "Fibroid Degeneration of the Heart," *International Med. Congress*, London (Abstracts), 1881, i. 427.—14. WEBER, Sir HERMANN. "Zur Lehre von der fettigen Entartung des Herzens," *Virchows Arch.*, 1857, xii. 326. —15. WILKS and MOXON. *Pathological Anatomy*, 3rd edit., London, 1889, 127. **III. Impairment due to Senile Change :** 16. BALFOUR, G. W. *The Senile Heart*, London, 1894.—17. OERTEL. "Therapie der Kreislaufs-Störungen," v. Ziemssen's *Handb. der allg. Therapie*, 1884, Bd. iv., Leipzig.—18. QUAIN, R. "Fatty Diseases of the Heart," *Med.-Chir. Trans.*, London, 1850, xxxiii. 121 ; and *Dictionary*, 1894.— 19. WILKS and MOXON. *Pathological Anatomy*, 3rd edit., London, 1889, 123. **IV. Impairment arising from Functional Overstrain :** 20. ALLBUTT, Sir CLIFFORD. "The Effect of Overwork and Strain on the Heart and Great Blood-Vessels," *St. George's Hosp. Rep.*, London, 1874, v. 23.—21. COATS, J. "Hypertrophy from Overstrain," *Manual of Pathology*, 3rd edit., London, 1895, 436, 440.—22. DA COSTA, J. M. "On Irritable Heart," *Amer. Journ. Med. Sc.*, Phila., 1871, lxi. 17.—23. DRESCHFELD. "Diagnosis and Treatment of Degeneration of the Heart apart from Valvular Disease," *Brit. Med. Journ.*, 1905, ii. 1023.—24. HAMILTON, D. J. *Textbook of Pathology*, London, 1889, i. 649.—25. JÜRGENSEN, SCHROETTER, and KREHL. "Diseases of Heart," in Nothnagel's *Encyclopedia*, edited by G. Dock, 1908, W. B. Saunders Co.—26. LEYDEN. "Ueber die Herzkrankheiten in Folge von Ueberanstrengung," *Ztschr. f. klin. Med.*, Berlin, 1886, xi. 105.—27. MYERS, A. B. R. *Diseases of the Heart among Soldiers* (Alexander Prize Essay), London, 1870.—28. PEACOCK, T. B. *Valvular Disease of the Heart* (Croonian Lectures, 1865), London, 1865.—29. POWELL, Sir R. D. "A Discussion on Functional Diseases of the Heart," *Brit. Med. Journ.*, 1894, ii. 1034. —30. SEITZ. "Zur Lehre von der Ueberanstrengung des Herzens," *Deutsches Arch. f. klin. Med.*, Leipzig, 1873, xi. 485 ; 1874, xii. 143, 297. **V. Impairment of Inflammatory Origin.** (a) **Simple or Secondary :** 31. COATS, J. *Manual of Pathology*, London, 1895, 441.—32. HAMILTON, D. J. *Textbook of Pathology*, London, 1889, i. 589.—33. HUCHARD, H. *Maladies du Cœur*, Paris, 1905, 194.—33a. MACKENZIE, J. *Diseases of the Heart*, 1908.—34. LEYDEN. "Ueber intermittirendes Fieber und Endocarditis," *Ztschr. f. klin. Med.*, Berlin, 1882, iv. 321. — 35. PEARCE, R. M. "Experimental Myocarditis, following Intravenous Injections of Adrenalin," *Journ. Exper. Med.*, N.Y., 1906, viii. 400. — 36. RECKLINGHAUSEN and others. "Ueber die Störungen des Myocardium," *Verhandlungen des X. internationalen med. Kongress*, Berlin, 1890, ii. Abth. 3, 67. (b) **Syphilitic :** 37. JACQUINET. "La syphilis du cœur," *Gaz. des hôp.*, Paris, 1895, lxviii. 917. — 38. PHILLIPS, S. "Syphilitic Disease of the Heart-Wall," *Lancet*, London, 1897, i. 223.— 39. ROBINSON, G. C. "Gumma of the Heart from a Case presenting the Symptoms of Adams-Stokes Disease," *Bull. Ayer Clin. Lab. Pennsylv. Hosp.*, 1907, No. 4.—40. WILKS, S. "On the Syphilitic Affections of Internal Organs," *Guy's Hosp. Rep.*, London, 1863, 3rd ser. ix. 41 ; *Trans. Path. Soc.*, 1856, viii. 24. (c) **Segmentation and Fragmentation :** 41. AUFRECHT. "Ueber einem Fall von primärer Fragmentation des linken Ventrikel," *Ztschr. f. klin. Med.*, 1894, xxiv. 205.—42. HEKTOEN. *Am. Journ. Med. Sc.*, Phila., 1897, cxiv. 555.—43. OESTREICH. "Die Fragmentatio Myocardii," *Virchows Arch.*, 1894, cxxxv. 79.—44. RECKLINGHAUSEN, *Verhandlungen des X. intern. med. Kongr.*, Berlin, 1890, ii. Abt. 3.—45. RENAUT. "Note sur les altérations du myocarde accompagnant l'inertie cardiaque," *Compt. rend. Soc. biol.*, 1877.— 46. *Idem.* "Note sur une nouvelle maladie organique du cœur ; la myocardite segmentaire essentielle chronique," *Bull. de l'Acad. de méd.*, Paris, 1890, xxiii. 3 sér. 245. **VI. Growths and Parasites :** 47. BERTHENSEN. *Virchows Arch.*, 1893, cxxxiii. 390.— 48. BOYCE, R. *Textbook of Morbid Histology*, London, 1892, 215.—49. CALVERT and PIGG. *Trans. Path. Soc.*, London, 1898, xlix. 297.—50. COATS. *Manual of Pathology*, 3rd edit., London, 1895, 464.—51. HAMILTON. *Textbook of Pathology*, London, 1889, i. 593.—52. HEKTOEN. *Med. News.*, Phila., 1893, lxiii. 511.—52a. KARRENSTEIN. *Virchows Arch.*, 1908, cxciv. 127.—53. KNAGGS. *Lancet*, 1896, i.—54. PAOLOWSKY. "Beitrag zum Studium der Symptomotologie der Neubildungen des Herzens. Polypöse Neubildungen des linken Vorhofs," *Berlin. klin. Wchnschr.*, 1895, xxxii. 393.—55. PEACOCK. *Trans. Path. Soc.*, London, 1873, xxiv. 38.—56. WILKS and MOXON. *Pathological Anatomy*, London, 1889, 123.—57. WOLBACH. *Journ. Med. Research*, Boston, 1907, xvi. 495.

R. D. P.

STOKES-ADAMS DISEASE

By Prof. William Osler, M.D., F.R.S.
Pathological Section by A. Keith, M.D.

Definition.—A condition of slow pulse with syncopal, apoplectiform, or epileptiform attacks associated either with (*a*) derangement of the junctional system of the heart, or (*b*) disease of the nerve-centres of the vagi or of the nerves themselves.

History.—Morgagni gave the first description of the disease. "I will just skim over . . . those many things which I have observed for a long time in my fellow-citizen, Anastasio Poggi, a grave and worthy priest. He was in his sixty-eighth year, of a habit moderately fat, and of a florid complexion, when he was first seized with the epilepsy, which left behind it the greatest slowness of pulse, and in like manner a coldness of the body. But this coldness of the body was overcome within seven hours, nor did it return any more, though the disorder often returned; but the slowness of the pulse still remained." Our modern knowledge dates from the work of two Irish physicians, Adams and Stokes, whose original descriptions may be summarised as follows:

Adams' patient, aged sixty-eight, was of full habit, and subject to oppression of breathing and cough. He was first seen when recovering from the effects of an apoplectic attack, which had come on suddenly three days before, but he was sufficiently well to be about the house and even to go out. What attracted Mr. Adams' attention was the character of the breathing and the remarkable slowness of the pulse, 30 to the minute. His regular attendant informed Mr. Adams that during seven years this patient had had not less than twenty apoplectic attacks; after a day or two of heaviness and lethargy he would fall down completely insensible, and on several occasions had hurt himself. The pulse would become slower than usual, and the breathing loudly stertorous. He never had any paralysis after the attacks. Death followed an attack, and the heart was found to be very fatty, the valves being sound. There was no statement about the coronary arteries.

In his much more important contribution, entitled *Observations on Some Cases of Permanently Slow Pulse*, Stokes describes the case of a man, aged sixty-eight, who had recurring fainting fits which, however, did not leave any unpleasant effects behind. In the course of three years he had had at least fifty seizures, which were induced by any circumstance tending to impede or oppress the heart's action, such as sudden exertion or a distended stomach. He was never convulsed and never had any paralysis. The duration of the attack was seldom more than four or five minutes, and during this time he was perfectly insensible. On admission his general health seemed very good. There was an apical systolic murmur,

and a pulse of 28 in the minute. The arteries appeared to be in a state of permanent distension, "the temporal arteries ramifying under the scalp just as they are seen in a well-injected subject." An interesting feature in this case was that the patient could ward off attacks by a peculiar manœuvre; "as soon as he perceives symptoms of the approaching attack he directly turns on his hands and knees, keeping his head low, and by this means, he says, he often averts what otherwise would end in an attack." It was noticed that, while his heart-beats were reduced to 28, there were occasional semi-beats between the regular contractions, 8 of them in the minute. On readmission an entirely new sign was observed, namely, a remarkable pulsation in the right jugular vein, the rate of which was more than double that of the manifest ventricular contractions. Subsequently cases were reported elsewhere from time to time; in France Charcot called attention to it in 1872, and in his *Traité des Maladies du Cœur* (1889), Huchard gave an admirable description of the condition, which he named Adams-Stokes disease. In this country very little attention was paid to it until the appearance of Dr. Webster's paper, in which the cases were carefully studied by the graphic methods. In 1903 I drew attention to the comparative frequency of the condition and to the various groups of cases. In Germany the studies of W. His, junior (1899), Jacquet (1902), and Luce (1902) stimulated a physiological interest in the subject, and brought the syndrome into relation with the phenomena of heart-block described twenty years before by Dr. Gaskell. In the United States the clinical features of slow pulse were very fully considered by Prentiss and by Edes (1901). Recently the brilliant anatomical demonstration by Tawara of the intracardiac junctional system of fibres, the physiological studies of Erlanger on heart-block, and the pathological investigation of the cases by Dr. Keith and others have combined to arouse the keenest interest in the subject. More than sixty separate papers have appeared within the four years 1905-8, references to which may be found in the *Index Medicus*, and in Pletnew's critical digest in Vol. I. of the *Klinische Ergebnisse* (1908). Bachmann has collected 177 cases of the Stokes-Adams syndrome.

Nomenclature.—Bradycardia, a very common condition, is due either (i.) to influences acting on the nerve-centres (or the vagi) or (ii.) to changes in the heart itself; and the cerebral features which characterise the Stokes-Adams syndrome may be associated with both these, neurogenous and cardiac, groups. The disturbance of rhythm which we know as heart-block is not an invariable accompaniment of bradycardia. I do not know that its existence has been determined in the cases due to organic disease of the medulla, the cases in which the heart simply slows down, without change in its rhythm. The syncope and epileptiform features are epiphenomena of a bradycardia however induced. Whilst in some ways it is a pity that the syndrome was ever labelled a disease, yet, as in the case of angina pectoris, the clinical picture is very definite however varied its pathology. Since it is too

late to attach Morgagni's name to it, we may continue to call the
syndrome or disease by the name Stokes-Adams ; and, remembering the
dictum of Socrates, that " it matters little what names you give to things
so long as you tell us just what the things are," I may repeat that under
this designation a description will be given of the cases with slow pulse
and syncopal and epileptiform attacks, and in two groups—cardiac and
neurogenous.

Incidence, Age, and Sex.—That the disease is not uncommon is
shewn by the number of cases reported in recent years.   In 1903 I
reported 13 cases, and since then I have seen 7 additional examples.
Men are more often affected than women—in my series all were men.
The age-incidence varies with the cause ; in a small group, due to acute
infections, young persons may be attacked ; Schuster indeed reported it
in a girl of four years of age.   In the group due to syphilis, men between
the ages of twenty and forty are affected ; but the great majority of the
victims are men who have reached the period of arterial degeneration.
Of my 20 cases, 2 were between thirty and forty, 6 between fifty and
sixty, 8 between sixty and seventy, and 4 above seventy.   My oldest
patient was a man of seventy-four.   W. O.

PATHOLOGY OF HEART-BLOCK AND ITS BEARING ON CASES MANI-
FESTING THE STOKES-ADAMS SYNDROME.—**Heart-block.**—Since 1906
it has become almost certain that the manifestations of the Stokes-Adams
disease depend on a lesion of the junctional system of fibres which unites
the musculature of the auricles to that of the ventricles.   The morbid
change may be so slight as to cause *partial heart-block*, or so severe as to
cause *complete* or *total heart-block*.   In the first condition the stimuli set up
by the contractions of the auricle traverse the junctional system at a
slower rate than normal, the contraction of the ventricle being thus
delayed, or some only of the stimuli excited by the auricular contractions
may succeed in reaching the ventricles, so that some of the ventricular
beats are missed.   In complete heart-block the junctional system is im-
permeable to all stimuli arising in the auricle ; if the ventricles continue
to beat, it is at a slow rate, and with no relation to the contraction of
the auricles.   The occurrence of complete heart-block in man is usually
marked by the appearance of the Stokes-Adams syndrome, but this is not
invariably the case, for Chauveau, Dr. J. Mackenzie, and others have
recognised a condition of complete heart-block without the manifestation
of syncope or epileptiform attacks.   Fits are more likely to occur in
incomplete than in complete heart-block (T. Lewis).   Bachmann has given
a very full clinical history of a case of complete block without syncopal
attacks.

The Nature of the Pathological Lesions in Cases of Heart-block.—
Such a short space of time has elapsed since the auriculo-ventricular
junctional system became a subject of inquiry and observation, that as
yet our knowledge of its pathology is very meagre.   I have access to

observations on the auriculo-ventricular system of twenty-one hearts from cases manifesting symptoms of heart-block during life. Sixteen of these are from published cases, five are from hearts examined by myself. The twenty-one hearts fall into five groups : (1) in which the system has been broken by a gumma (cases observed clinically by Chapman, Ashton, Luce (see Fahr), Vaquez, Robinson, Heineke, and Otto Grünbaum) ; (2) in which the system was partly fibrosed, evidently as a result of arteriosclerosis (Hay, James Barr, G. A. Gibson, C. H. Miller, and Turrell and Gibson) ; (3) in which the bundle was implicated in fibrous or cicatricial tissue, probably from rheumatic endocarditis (Schmoll, G. A. Gibson and W. T. Ritchie (22), Aschoff, Fahr, Dock, and Gerhardt) ; (4) in which the system was the seat of fatty infiltration (Butler, Aschoff) ; (5) in which the system was injured by an acute infection (Jellinek and Cooper).

(1) *Cases in which the Auriculo-ventricular Bundle has been invaded by a Gumma.* — During 1906 and 1907 I minutely examined the hearts of five patients in whom the Stokes-Adams syndrome was well marked. Figs. 23 and 24 represent, somewhat diagrammatically, the right and left chambers of the heart of one of these cases, which was originally described by Dr. C. W. Chapman, and afterwards by Dr. Miller and myself. On the right side of the heart (Fig. 23), at the junction of the septal wall of the right auricle with the corresponding wall of the right ventricle, there is a mass of cicatricial and gummatous tissue extending from the opening of the coronary sinus (between d and e) to the pars membranacea septi (between f and i) ; the region involved contains two essential parts of the junctional system—the auriculo-ventricular node ($a.$-$v.$ node)—which forms the auricular commencement of the system, and the upper part or main stem ($a.$-$v.$ bundle) of the system. In Fig. 24 the left side of the same heart is shewn. The gummatous mass is seen below the aortic orifice of the left ventricle, occupying the upper part of the interventricular septum and also the membranous part of that structure. As is shewn diagrammatically in Fig. 24 the mass involves the main bundle. The oblong area of the septal wall indicated in both figures was excised, prepared, and cut serially, the sections being made transversely to the long axis of the mass excised ; they commenced at the anterior or ventricular end, and were carried backwards to the auricular end. The sections were 10 to 12 μ thick ; each 30th section was kept, and stained by van Gieson's method. In the anterior or ventricular end of the mass, the right and left divisions of the main bundle were found intact (see Figs. 23, 24), normal in structure, and larger in size than usual—the left division forming a layer of 5 or 6 fibres deep beneath the endocardium of the septal wall of the left ventricle. On proceeding towards the auricular end of the block, the main bundle became involved in the amorphous material of the cicatricial mass, no trace of the upper part or of the auriculo-ventricular node being found. On the other hand the right and left divisions of the junctional system, and the final ramifications of these divisions in the sub-endocardial layer of the ventricles— these ramifications being most numerous and best developed at the bases

of the musculi papillares—were, as far as the microscope could shew, normal in structure and rather more than normal in amount.

The patient, a man aged fifty-six, died in 1905. In 1892 he first manifested symptoms of heart-block. No tracings were taken of his
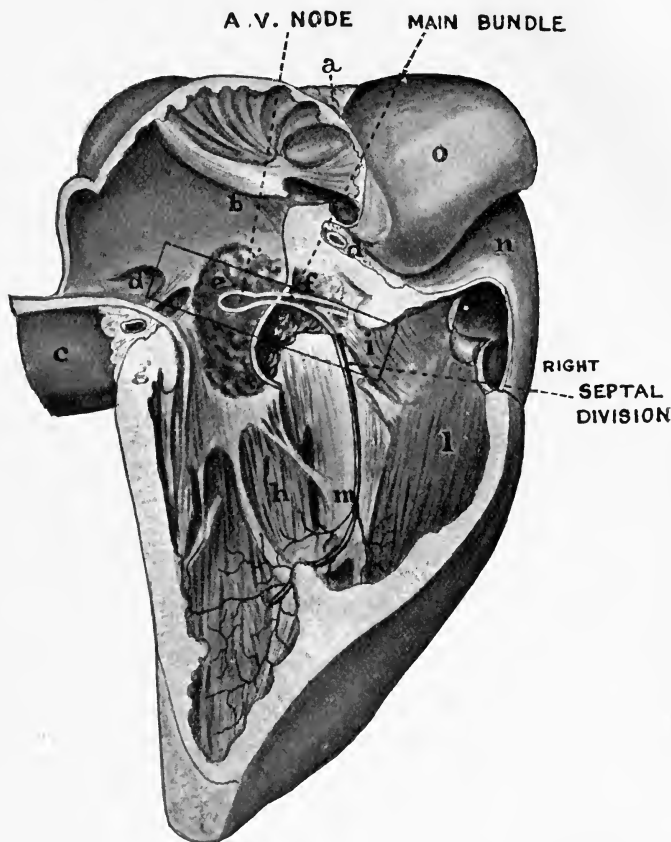

FIG. 23.—Right chambers of the heart, from a case of Stokes-Adams disease of the heart, described in the text, with position of *a.-v.* node, main bundle, and right septal division indicated. *a*, Remnant of superior vena cava. *b*, Position of orifice of superior vena cava, which is quite closed. *c*, Greatly dilated inferior vena cava. *d*, Fossa ovalis. *e*, Cicatricial tissue of interauricular septum (stippled). *f*, Pars membranacea septi and extension of cicatricial tissue into interventricular septum, in which the auriculo-ventricular bundle is involved. Between *d* and *e* is the opening of the coronary sinus. Oblong figure indicates the block cut out for examination. *g*, Base of right ventricle. *h*, Body of right ventricle. *l*, Infundibulum. *m*, Moderator band. *n*, Pulmonary artery. *o*, Aorta. For convenience of printing, the figure is placed base up; part of the septal cusp of the tricuspid is cut away to shew the gummatous mass and position of the *a.-v.* node and main bundle, which were destroyed by the disease. Two-thirds natural size.

jugular pulse. The terminal part of the superior vena cava and the musculature in which the heart-beat is believed to arise were completely destroyed by a gummatous infiltration (see Fig. 23). By itself this case does not prove that a lesion of the junctional system is the cause of the Stokes-

Adams syndrome, but since Humblet, Erlanger, Hering, and others have shewn that the main bundle of the junctional system is not merely the normal but the only path by which the auricular rhythm can be transmitted to the ventricles, it may be inferred, apart from the clinical symptoms, that this patient must have had a condition of complete
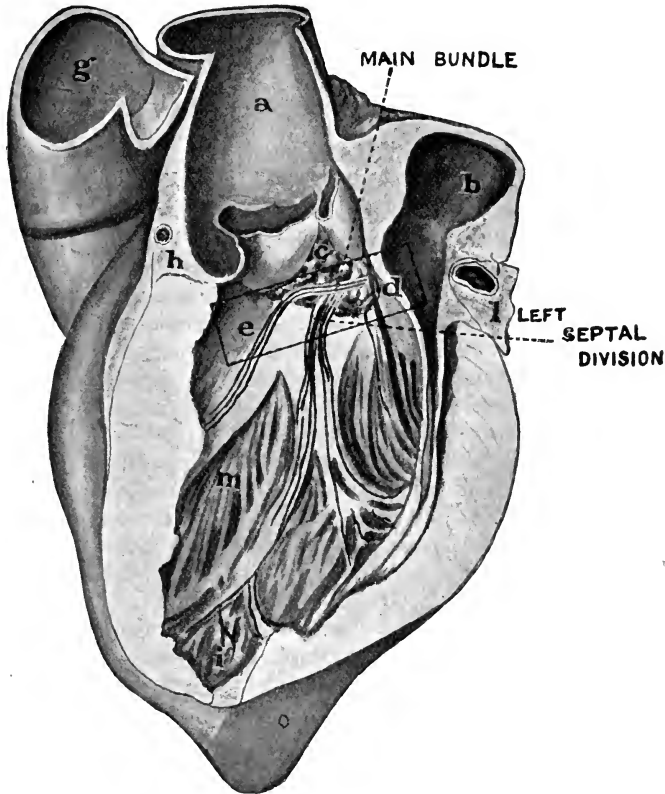
FIG. 24.—Left side of the same heart as is shewn in Fig. 23, and the position of main bundle with its left septal division indicated. *a*, Aorta. *b*, Contracted left auricle (stippling shews the extent of the cicatricial tissue). *c*, Pars membranacea septi ; the stippled area below shews the extension of the gummatous tissue into the interventricular septum, in which the auriculo-ventricular bundle is situated. *d*, *e*, The block cut out for examination. *f*, The left septal division of the bundle. *g*, Pulmonary artery. *h*, Cicatricial tissue at origin of aorta. *l*, Coronary sinus. *m*, Septal wall of the left ventricle. Two-thirds natural size.

heart-block ; this case has also an important bearing on the functional nature of the junctional system. If this were, as was at first supposed, merely a conducting system, it should, after being unused for over twelve years, have become atrophied. Since an opposite condition has resulted, we must suppose that it has the power of initiating as well as conducting contraction stimuli—a conclusion which has been supported by many, especially Mackenzie and Aschoff, and also by Lohmann from the result

of an experimental inquiry. Dr. Gaskell's experiments on the tortoise's heart, when considered in the light of comparative anatomy, also favour this supposition.

Other Cases of Gumma in the Heart invading the Auriculo-ventricular Bundle.—Another case, somewhat similar to the one just described as regards the situation and nature of the lesion, was examined by me for Dr. Otto Grünbaum, who has briefly recorded the clinical symptoms. The main bundle, for an extent of nearly a centimetre, was completely destroyed; the condition of the auriculo-ventricular node and the ramification in the ventricle were not examined, this being one of the earliest hearts examined to discover the pathological condition of the junctional system. Five excellently recorded cases of a similar kind are to be found in recent literature, one by Ashton, Norris, and Lavenson, another by Vaquez and Esmein, the third by Fahr, the fourth by Robinson, and the fifth by Heineke, Müller, and v. Hösslin. The first of these cases was that of a man of thirty, who manifested the Stokes-Adams syndrome for twenty-one days before death; the interventricular septum was infiltrated with a gummatous mass which had destroyed the main bundle at its bifurcation; in the second, that of a man aged forty-three who had manifested the symptoms of Stokes-Adams disease for ten months before death, there was a gummatous mass occupying an area of the septal wall of the heart similar to that found in the first case examined by me. Fahr's case resembled that described by Miller and myself. Robinson examined a museum specimen of a "gumma of the heart," and finding that it occupied the part of the septum through which the auriculo-ventricular bundle runs, referred to the clinical notes, which shewed that the symptoms of Stokes-Adams disease had existed before death. The cases recorded by Dr. Handford and by Dr. Phillips, although the condition of the junctional system was not examined, were similar as regards the nature of their lesion to the six cases just cited. In Sendler's case the bundle was apparently interrupted by a cartilaginous tumour.

(2) *Pathological Lesions of the Auriculo-ventricular Bundle in Cases of Arteriosclerosis.*—Cases of arteriosclerosis presenting the Stokes-Adams syndrome do not shew any well-marked pathological lesion. In the heart of a case recorded by Dr. John Hay—that of a man aged sixty-five, presenting all the symptoms of complete heart-block for eleven months before death, I found the following condition :—The auriculo - ventricular junctional system was nowhere broken; the central fibrous body, against the right aspect of which the auriculo-ventricular node is applied, was atheromatous with patches of calcification; the coronary arteries were markedly arteriosclerotic, their lumen being reduced to half in the larger vessels; this was also the condition of the artery to the bundle—a small branch that arises from the right coronary artery at the upper end of the posterior interventricular groove. Microscopical examination of the musculature of the auriculo-ventricular junctional system shewed that the muscle-fibres were larger and that some of them were more fibrous than usual; instead of the fibres of the main bundle forming an open

reticular arrangement they lay in parallel leashes ; there were scattered foci of slight inflammatory exudation—especially near the commencement of the bundle. But it could not have been said, from the morbid appearances, that this case should have presented a condition of complete heart-block ; hearts in which there has not been any heart-block may shew an equally fibrous condition of the bundle. It must be mentioned that occasionally in this case, even some days before death, the pulse-rate, instead of being from 24 to 36 a minute, ran up to 60, as if the block were then incomplete or removed. In Sir James Barr's case, a man aged sixty-four, a very similar pathological condition was found ; the bundle, though intact, was manifestly stretched and attenuated ; there was an unusual proportion of fibrous tissue in its main part, and the central fibrous body, especially round the point at which the main bundle perforates it, was atheromatous and infiltrated with lime salts ; here again it would have been impossible to say, on the pathological data alone, that a condition of heart-block had been present. In some twenty pathological hearts, submitted to me by Dr. James Mackenzie, all of them accurately recorded during life, none being cases of heart-block, some shewed a degree of fibrosis almost equal to that found in the cases of heart-block ; in another case, in which the nodal rhythm (Mackenzie) was present, a small endocardial ulcer had eaten right into the bundle, so that two-thirds of its diameter were destroyed or invaded by inflammatory material. Yet there was no heart-block. A third heart examined by me, belonging to the arteriosclerotic group of cases manifesting the Stokes-Adams syndrome, gave absolutely negative results. The case was examined by Dr. Charles Miller. The heart was that of a man aged fifty-five, with a pulse-rate of 22 to 36 per minute, who suffered for eight days before death from frequent fits, before each of which the rate of the pulse slowed ; the patient lost consciousness for about 20 seconds, the limbs became rigid, eyes open and staring. No record was taken of the auricular rhythm. Before cutting out the central part of the heart for microscopic section, the position of the auriculo-ventricular node was seen to be indicated by a dark-red patch. But in the examination of the sections, there was, in my opinion, no appearance that could be called pathological ; the bundle was particularly well developed and apparently healthy. Probably a case of heart-block recorded by Dr. G. A. Gibson, that of a man aged forty-four, should also be included in this group. The auriculo-ventricular bundle was intact, but there was a cellular infiltration amongst its fibres with some degree of fibrosis ; to the naked eye the bundle appeared paler than normal. Edes found that a condition of arteriosclerosis was present in 33 of 41 recorded cases of heart-block. The pathological condition of the case described by Drs. Turrell and Gibson was exactly similar to that found by me in the cases of Sir J. Barr and Dr. Hay ; but in Turrell and Gibson's case there is no evidence that the block was complete. Heineke's second case also belongs to this group. Recently, Drs. G. A. Gibson and Ritchie (22*a*) have published a full account of the case of the well-known physician, Sir

William Gairdner.   At the age of seventy-five he became subject to
syncopal attacks, which occurred frequently until he was seventy-nine
years of age.   Then the slow pulse (28-34 per minute, usually 32), at
first only temporary during the attacks, became permanent and the
syncopal attacks ceased.   He died when eighty-three years of age.   At
the necropsy, the coronary arteries were found to be atheromatous; the
node and upper part of the bundle fibrous and calcareous; the lower
part of the bundle partly fibrous, partly muscular.

(3) *Pathological Lesion of the Bundle in Hearts which may have been
affected by Rheumatic Endocarditis.*—In another group of recorded cases
with Stokes-Adams disease, in which the junctional system was examined
after death, the results of pathological investigation were indefinite,
as in the last group.   The morbid condition in this group was prob-
ably the result of endocarditis which, in some of the cases at least,
was rheumatic.   The best-marked lesion was found in Schmoll's case
of a woman, aged sixty-six.   The main bundle was large, more fibrous
than usual, and infiltrated by cicatricial tissue, but from the photomicro-
graphs published, it would appear that the main bundle was not seriously
damaged.   At the division of the main bundle into its right and left septal
divisions, the muscular tissue of the junctional system was interrupted
by a cicatrix.   There was also a certain degree of arteriosclerosis of the
artery to the bundle, but I have seen this artery much more severely
diseased in cases without any symptoms of heart-block.   The case
recorded by Fahr is very similar.   In a case examined by Aschoff the
continuity of the junctional system was not broken at any point, and
the arteries of the node and bundle were not markedly diseased.   There
was some fibrosis and infiltration of the bundle, but not to such an extent
as to suggest that its function was seriously impaired.   In two other cases
(Dock, and G. A. Gibson and W. T. Ritchie (22)) the junctional system
was intact, but certain fibrous patches were seen partly to invade the
main bundle or one of its limbs.   A moderate degree of endarteritis was
present in both cases.   In the case recorded by Stengel, the main bundle
was supposed to be involved by a patch of atheroma, but no microscopic
examination was recorded when the case was first published.   In Ger-
hardt's case there was a recent inflammatory exudate at the point where
the bundle perforates the central fibrous body of the heart.

(4) *Cases in which the Junctional System was the Seat of Fatty Infiltration.*
—Butler has recorded a case of heart-block in which the junctional
system, although not anywhere broken, was infiltrated with fat and
atrophied to one-fifth of its normal size.   The patient, a man of forty-
five, with the characteristic symptoms of Stokes-Adams disease for ten
days before death, had a temperature of 101° F.—not a sign likely to
result from fatty infiltration alone.   Aschoff has recorded a very similar
condition in the heart of a patient with symptoms of heart-block; and
I have seen two cases in which this system was infiltrated with fat,
especially its connective-tissue sheath, so that the whole of it, including
the right and left septal divisions with their ramifications, was plainly

seen as greyish-yellow strands, when the ventricles were laid open. In one of the cases—examined clinically by Dr. J. Mackenzie—there was no heart-block, but the nodal rhythm was present before death. In the other the clinical notes do not refer to any peculiarity of the cardiac rhythm.

(5) *Cases in which the Junctional System was damaged by an Acute Infection.*—The last group of cases to be described here is that in which the bundle is involved in an area of necrosis, due to an acute infection. The best—indeed the only—recorded case of this nature is that published by Jellinek and Cooper. It was that of a man aged thirty, with acute gonorrhoeal infection, who manifested symptoms of heart-block for fourteen days before death. The upper part of the interventricular septum, containing the main bundle, had undergone an acute necrosis, and the arteries in it were thrombosed. Foley's case was probably similar in nature, but there was no examination made of the bundle. Dr. Gossage has observed heart-block after influenza, and it is known to occur after diphtheria, but so far the pathological condition of the junctional system has not been examined in such cases.

Condition of the Central Nerve-centres.—The cerebral symptoms seen in cases of Stokes-Adams disease have frequently been ascribed to a lesion of the central nervous system. In two cases, Medea systematically examined the central nuclei of the vagus and spinal accessory, as well as the trunks of these nerves, but found no lesion. Dr. A. Webster shewed that the slowing of the heart preceded the cerebral manifestation, and it is now generally agreed that the cerebral symptoms are a direct result of a circulatory disturbance, following a momentary failure of the left ventricle. Amongst recorded cases shewing the Stokes-Adams syndrome, Edes found three in which disease of the bulbar or upper spinal centres was discovered after death.

Experimental Pathology of Heart-block.—Of the 21 cases of heart-block here recorded, 8 only shew a definite break in the continuity of the auriculo-ventricular junctional system, the interruption being in the main bundle itself or at its bifurcation into the septal divisions. The lesions caused by disease that do provide convincing evidence that heart-block and the Stokes-Adams syndrome are the result of a failure of the junctional system to transmit the auricular impulse to the ventricles ; the proof must be sought in the results obtained by direct experiment on this system. Dr. Gaskell's observations on the auriculo-ventricular junctional musculature of the tortoise's heart, published in 1883, laid the foundation of our knowledge of heart-block ; he demonstrated that this system transmitted the auricular impulse to the ventricles, and that in the absence of the auricular impulse it could give rise to a stimulus which brought about a ventricular systole. In 1893 Dr. Stanley Kent and W. His, junior, discovered independently that the junctional system of the tortoise was represented in the mammalian heart by a muscular strand, the auriculo-ventricular bundle. Tawara in 1906 shewed that the bundle was but one part of the junctional system ; that in the mammalian heart

this system commenced in the septal wall of the right auricle as a small node of reticulated muscle-tissue (*a.-v.* node), out of which issued the *main bundle* to divide at the upper margin of the interventricular septum into right and left septal *divisions*, one to each ventricle, these divisions ending in the ventricular musculature by widespread *sub-endocardial ramifications* (see Figs. 23, 24 ; also Keith and Flack).   Wenckebach proved in 1899 that disturbance in conduction at the auriculo-ventricular junction of the heart was the cause of certain arrhythmias ; but W. His, junior, was the first to try the effects of section of the bundle (1895), and in publishing a case shewing the Stokes-Adams syndrome in 1899 he definitely stated that he regarded the condition to be that of heart-block, and due probably to a lesion of the auriculo-ventricular bundle.   Humblet, Hering, and Erlanger proved that section of the bundle produced heart-block ; in 1906 Erlanger, from experiments on the hearts of dogs, had obtained ample proof that the Stokes-Adams syndrome was a result of a lesion of the junctional system.   He found that the symptoms which followed heart-block produced by clamping or cutting the main bundle depended on the readiness with which the ventricle assumed an automatic rhythm. The ventricles may respond at once, and this is more likely to be the case when the block is produced slowly, calling gradually into action the latent automatic power of the ventricles ; or they may not respond until the auricles contract sixty times or more.   In two of Erlanger's experiments the ventricles failed to respond sixty seconds after the block was established ; in these two cases respiratory convulsions appeared.   In cases such as those recorded by Chauveau, Mackenzie, and others, in which the auricles and ventricles contracted independently of each other, without any manifestation of cerebral symptoms, we must suppose that the automatic rhythm of the ventricles was speedily and well established. The less the inherent tendency of the ventricles to assume the power of automatic contraction—and clinical as well as experimental observation shews there is a considerable degree of individual variation in this respect —the more probable is it that cerebral symptoms or sudden death will appear.   At the present time there is not sufficient evidence to decide whether the ventricular response is a special quality of the junctional system or of the whole ventricular musculature.   I have now seen three cases in which the ramifications of the junctional system in the apical half of the left ventricle were destroyed by arteriosclerosis of the coronary vessels, and yet the only clinical symptoms were extra-systoles of the ventricles.

It is conceivable that heart-block might be produced by a lesion of any part of the junctional system : (1) of the fibres which unite the auricular musculature to the auriculo-ventricular node ; (2) of the auriculo-ventricular node itself ; (3) of the main bundle ; (4) of the septal divisions, and (5) of the sub-endocardial ramifications.   All the ramifications cannot possibly be diseased, as the whole ventricular musculature would then necessarily be implicated.   Biggs found that section of some of the ramifications in perfused hearts did not alter the contraction

of that part of the ventricle to which they were distributed, results in agreement with those obtained by Drs. L. Hill and Flack. I have seen such ramifications in a morbid condition in cases in which the symptoms of heart-block had been absent. The anastomosis between the ramifications is so free that anatomical considerations are quite in accord with the results obtained by Biggs. In 1899 W. His, junior, suggested that heart-block might be due to a refractory condition of the ventricular musculature or a failure of this muscle to respond to the stimuli transmitted to it by the junctional system, but of this there is as yet no evidence. The part of the junctional system at which it is possible for a gross single lesion to produce heart-block is limited to the auriculoventricular node and the main bundle ; this area is small and has a lineal extent of about 25 mm. in the human heart, being bounded at its auricular end by the orifice of the coronary sinus, and at its ventricular end by the lower margin of the pars membranacea septi. Experimental lesions must sever every strand of the bundle to give complete heart-block ; a few intact strands serve to convey the auricular impulse. If the auriculo-ventricular node possesses a superior degree of excitability—a proposition which has the support of comparative anatomy,—then a lesion between the auricle and the node, while giving a condition of heart-block, should at the same time give a fairly rapid ventricular rhythm (Mackenzie's nodal rhythm). We know that in complete section below the node the ventricular rhythm varies in most cases between 22 and 36 ; 28 may be regarded as the average manifestation of the inherent rhythm of the ventricles.

It must be kept in mind in connexion with the intact condition of the bundle found in some cases with the symptoms of Stokes-Adams syndrome, that heart-block may be produced by a functional derangement. Dr. J. Mackenzie and v. Tabora have shewn that digitalis has such an effect under certain conditions, and Prof. Cushny and Knoll have also been able to produce it by pharmacological means. As is well known, it may be brought on by stimulation of the vagus ; Erlanger demonstrated that after section of the main bundle, stimulation of the vagus had no longer any effect on the ventricle ; hence atropine does not affect the ventricular pulse in cases of heart-block. The vagus cannot exert a direct influence on the ventricles ; it can influence them only through the auriculo-ventricular junctional system. In a case of complete block, Bachmann found that the administration of 5 minims of the tincture of strophanthus three times daily increased the ventricular rate and diminished that of the auricles, so that an occasional ventricular beat resulted from the auricular contraction, the block becoming thus incomplete.

A. KEITH.

REFERENCES

1. ASCHOFF, A. "A Discussion on Some Aspects of Heart-Block," *Brit. Med. Journ.*, 1906, ii. 1103.—2. ASCHOFF und TAWARA. *Lehre von den pathologisch-anatomischen Grundlagen der Herzschwäche*, Jena, Fischer, 1906.—3. ASHTON, NORRIS, and LAVENSON. "Adams-Stokes Disease (Heart-Block) due to a Gumma in the Inter-

ventricular Septum," *Am. Journ. Med. Sc.*, Phila., 1907, cxxxiii. 28 ; also *Trans. Coll. Physicians, Philadelphia*, Phila., 1906, xxviii. 236.—3a. BACHMANN. *Amer. Journ. Med. Sc.*, Phila., 1909, cxxxvii. 342.—4. BARR, Sir J. "Case of Stokes-Adams Disease," *Brit. Med. Journ.*, 1906, ii. 1122.—5. BECK and STOKES. "A Clinical and Pathological Study of a Case of Adams-Stokes Disease," *Arch. Int. Med.*, Chicago, 1909, ii. 277.—6. BIGGS. "Investigation of the Bundle of His in Rabbits' Excised Hearts perfused with Locke's Fluid," *Brit. Med. Journ.*, 1908, i. 14-19.—7. BOINET et ROUSLACROIX. "Pouls lent permanent avec dissociation du rythme cardiaque chez un syphilitique," *Arch. gén. de méd.*, Paris, 1906, année lxxxiii., t. ii. 2497.—8. BUTLER. *Am. Journ. Med. Sc.*, Phila., 1907, cxxxiii. 715.—9. CHAPMAN, C. W. "A Case of Cardiac Syphiloma, with Bradycardia and Obstruction of the Inferior Vena Cava : The After-history and a Post-mortem Record," *Lancet*, London, 1906, ii. 219.—10. CHAUVEAU. *Rev. de méd.*, Paris, 1885, v. 161.—11. COWAN, J. "The Myogenic Theory," *Practitioner*, London, 1907, lxxviii. 453.—12. CULLEN and ERLANGER. "Experimental Heart-Block," *Johns Hopkins Hosp. Bull.*, Balt., 1906, xvii. 234.—13. EDES, R. T. "Slow Pulse, with special Reference to Stokes-Adams' Disease," *Philadelphia Med. Journ.*, Phila., 1901, viii. 264, 310, 367, 408.—14. ERLANGER, J. "An Instance of Complete Heart-Block in Man," *Amer. Journ. Physiol.*, Boston, 1905, xiii. xxvi.—15. *Idem.* "On the Physiology of Heart-Block in Mammals, with especial Reference to the Causation of Stokes-Adams Disease," *Journ. Exper. Med.*, New York, 1905, vii. 676.—16. FAHR. "Ueber die musculäre Verbindung zwischen Vorhof und Ventrikel (das His'sche Bündel) im normalen Herzen und beim Adams-Stokes'schen Symptomkomplex" (plate),¦*Virchows Arch.*, Berlin, 1907, clxxxviii. 562.—17. FOLEY. "Stokes-Adams Syndrome : a Report of Two Cases, with a Short Résumé of the Literature," *Boston Med. and Surg. Journ.*, 1905, cliii. 235.—18. GASKELL, W. H. *Journ. Physiol.*, Cambridge, 1881, iv. 43.—18a. GERHARDT. "Ueber Rückbildung des Adams-Stokes'schen Symptomkomplexes," *Deutsches Arch. f. klin. Med.*, Leipzig, 1908, xciii. 485-499.—19. GIBSON, A. G. "The Heart in a Case of Stokes-Adams Disease " (plate), *Quart. Journ. Med.*, Oxford, 1908, i. 182. —20. GIBSON, G. A. "The Electro-motive Changes in Heart-Block," *Brit. Med. Journ.*, 1906, ii. 22.—21. *Idem.* "Heart-Block," *Brit. Med. Journ.*, 1906, ii. 1113.—22. GIBSON, G. A., and RITCHIE, W. T. "Further Observations on Heart-Block," *Practitioner*, London, 1907, lxxviii. 589.—22a. GIBSON, G. A., and RITCHIE. "A Historical Case of Adams-Stokes Syndrome," *Lancet*, London, 1909, i. 533.—23. GOSSAGE, A. M. "Cases of Stokes-Adams' Disease," *Trans. Clin. Soc.*, London, 1905, xxxviii. 187.—24. HAND-FORD, H. "Remarks on a Case of Gummata of the Heart : Death from Heart-Block, Rhythmical Contraction of the Auricles during the Long Pauses," *Brit. Med. Journ.*, 1904, ii. 1745.—25. HAY, J. "Bradycardia and Cardiac Arrhythmia produced by Depression of Certain of the Functions of the Heart," *Lancet*, London, 1906, i. 139, 239.—26. *Idem.* "Stokes-Adams Disease : Report of a Case," *Liverpool Med.-Chir. Journ.*, 1906, xxvi. 66.—27. HAY, J., and MOORE, S. A. "Stokes-Adams Disease and Cardiac Arrhythmia," *Lancet*, London, 1906, ii. 1271.—27a. HEINEKE, MÜLLER, und v. HÖSSLIN. "Zur Kasuistik des Adams-Stokes'schen Symptomkomplexes," *Deutsches Arch. f. klin. Med.*, Leipzig, 1908, xciii. 459-484.—28. HEWLETT, A. W. "Heart-Block in the Ventricular Wall," *Arch. Int. Med.*, Chicago, 1909, ii. 139.—29. HIS, W., Jun. "Ein Fall von Adams-Stokes'scher Krankheit mit gleichzeitigem Schlagen der Vorhöfe und Herzkammern (Herzblock)" (plate), *Deutsches Arch. f. klin. Med.*, Leipzig, 1899, lxiv. 316.—30. HOFFMAN, A. "Zur Kenntniss der Adams-Stokes'schen Krankheit," *Ztschr. f. klin. Med.*, Berlin, 1900, xli. 357.—31. HUMBLET. "Allorythmie cardiaque par section du faisceau de His," *Arch. internat. de physiol.*, Liège, 1905-6, iii. 330.—32. JAQUET, A. "Ueber die Stokes-Adams'schen Krankheit," *Deutsches Arch. f. klin. Med.*, Leipzig, 1902, lxxii. 77.—33. JELLINEK and COOPER. "Report, with Comment of Six Cases of Heart-Block, with Tracings, and one Post-mortem Examination of the Heart," *Brit. Med. Journ.*, 1908, i. 796.—34. JELLINEK, COOPER, and W. OPHÜLS. "The Adams-Stokes Syndrome and the Bundle of His," *Journ. Amer. Med. Assoc.*, Chicago, 1906, xlvi. 955.—35. KEITH, A., and FLACK. "The Auriculo-ventricular Bundle of the Human Heart," *Lancet*, London, 1906, ii. 359.—36. *Idem.* "The Form and Nature of the Muscular Connections between the Primary Divisions of the Vertebrate Heart," *Journ. Anat. and Physiol.*, London, 1907, xli. 172.—37. KEITH, A., and MILLER. "Description of a Heart, shewing gummatous Infiltration of the Auriculo-ventricular Bundle," *Lancet*, London, 1906, ii. 1429.—38. KENT, A. F. S. "Researches on the Structure and Function of the Mammalian

Heart" (plate), *Journ. Physiol.*, Cambridge, 1893, xiv. 233.—39. KNOLL. "Graphische Versuche an den vier Abtheilungen des Säugethierherzens," *Sitzungsb. d. kaiserlichen Akad. der Wissensch.*, Wien, 1894, ciii., Abt. iii. 298. — 39a. KRUMBHAAR. "On the Growth of our Knowledge of Stokes-Adams Disease," *Univ. Penna. Med. Bull.*, Phila., 1908, xxi. 278.—39b. LEWIS, T. "Occurrence of Heart - Block in Man and its Causation," *Brit. Med. Journ.*, 1908, ii. 1798.—40. LOHMANN, A. "Zur Automatie der Brückenfasern und der Ventrikel des Herzens," *Arch. f. Anat. u. Physiol.*, Leipzig, 1904, Physiol. Abth. 431.—41. LUCE, H. "Zur Klinik und pathologischen Anatomie des Adams-Stokes'schen Symptomencomplexes," *Deutsches Arch. f. klin. Med.*, Leipzig, 1902, lxxiv. 370.—42. MACKENZIE, J. "Definition of the Term Heart - Block," *Brit. Med. Journ.*, 1906, ii. 1107.—43. *Idem. The Study of the Pulse*, etc., 8vo, Edinburgh, 1902.—44. MEDEA. "La pathogénèse de la maladie de Stokes-Adams," *Progrès méd.*, Paris, 1905, 3me sér. xxi. 65.—45. PHILLIPS, S. "Syphilitic Disease of the Heart-Wall," *Lancet*, London, 1897, i. 223.—46. RENTOUL. "A Case of Heart-Block," *Brit. Med. Journ.*, 1907, ii. 85.—47. ROBINSON, G. C. "Gumma of the Heart from a Case presenting the Symptoms of Adams-Stokes Disease," *Bull. Ayer Clin. Lab.*, Phila., 1908, No. 4, p. 1, 2 figs.—48. SCHMOLL, E. "Adams-Stokes Disease," *Journ. Amer. Med. Assoc.*, Chicago, 1906, xlvi. 361.—49. *Idem.* "Zwei Fälle von Adams-Stokes'schen Krankheit mit Dissoziation von Vorhof und Kammerrhythmus und Läsion des His'schen Bündels," *Deutsches Arch. f. klin. Med.*, Leipzig, 1906, lxxxvii. 554.—50. SENDLER. "Beitrag zur Frage über Bradycardie," *Centralbl. f. klin. Med.*, Leipzig, 1892, xiii. 642.—51. SHORT, A. R. "A Case of the Stokes-Adams Syndrome, with Necropsy," *Lancet*, London, 1906, i. 30.— 52. SIEVERS. "Un cas de maladie de Stokes-Adams," *Arch. gén. de méd.*, Paris, 1906, année lxxxiii., t. i. 51.—53. STENGEL, A. "A Fatal Case of Stokes-Adams Disease with Autopsy, shewing Involvement of the Auriculo-ventricular Bundle of His : A Preliminary Communication," *Am. Journ. Med. Sc.*, Phila., 1905, cxxx. 1083.—54. TABORA, VON. "Ueber die experimentelle Erzeugung von Kammersystolenausfall und Dissociation durch Digitalis" (plate), *Ztschr. f. exper. Path. u. Therap.*, Berlin, 1906, iii. 499.—55. TAWARA. *Das Reizleitungssystem des Säugetierherzens*, Jena, 1906.—56. TURRELL, and GIBSON, A. G. "A Case of Adams-Stokes Syndrome observed for more than Eight Years," *Brit. Med. Journ.*, 1908, ii. 1486.—57. VAQUEZ et ESMEIN. "Maladie de Stokes-Adams par lésion sclérogommeuse du faisceau de His (Herzblock)," *Presse méd.*, Paris, 1907, xv. 57.—58. *Idem.* "Pouls lent d'origine myocarditique (Herzblock)," *Bull. et mém. Soc. méd. des hôp. de Paris*, 1907, 3me sér. xxiv. 78.—59. WENCKEBACH. "Arrhythmia of the Heart," translated by Snowball, 1904.

A. K.

Pathological Physiology.—As already stated, the bradycardia may be due to many causes, grouped into the two divisions neurogenous and cardiac. It may be a true bradycardia involving both auricles and ventricles, and there may then be complete or partial heart-block. In a majority of all cases of the Stokes-Adams syndrome there is dissociation of the auricular and ventricular contractions, as shewn in the tracings, and the ventricular rate becomes permanently slow. This condition may persist for many years without symptoms, or there may be transient attacks of giddiness. The explanation of the remarkable cerebral phenomena of Stokes-Adams disease is to be found in a study of the cases of disease of the cervical vertebrae, and in the well-known Kussmaul and Tenner experiment. In a patient with destruction of the check ligaments of the odontoid process, a movement which caused pressure on the medulla was immediately followed, instantaneously indeed, by reduction in the pulse-rate and by loss of consciousness (*vide* also p. 145). It was remarkable with what rapidity recovery took place when his head was straightened. He had had a score or more of such attacks. The irritation of the vagus centres (or of the nerves) slowed the heart and a

relative ischaemia of the brain with loss of consciousness at once resulted. Kussmaul's observation is still more striking, and the description of an experiment on a friend may be given in his own words :—" His carotids were most favourably placed. Scarcely had I compressed them with my fingers when he turned pale and collapsed off the stool. I had just time to catch him. He recovered consciousness immediately and said, 'Where am I?'" It is interesting to note that Kussmaul attributes a knowledge of this result to Galen. The transient loss of consciousness may be followed by convulsions if the pressure be maintained. In the cardiac group of cases it may not be altogether ventricular in origin, for there is often widespread arteriosclerosis, and, as Huchard suggests, the arteries of the medulla may play a part. If calcified and stiff they may not so readily adapt themselves to changes in the action of the ventricle, and we know that in a certain stage of sclerosis arteries are very prone to spasm : the cerebral features of Raynaud's disease, and the transient aphasia and monoplegia seen in the subjects of arteriosclerosis, indicate what an important part is played by spasm and relative ischaemia. But for most of the cases of Stokes-Adams disease the Kussmaul experiment satisfactorily explains the cerebral symptoms, and an appeal to angiospasm and reflex action is unnecessary.

CLINICAL PICTURE.—A. *Cardiac Group.*—A man of sixty or seventy, healthy and strong, or a younger man who has had syphilis, is attacked by vertigo, faints, or has a slight epileptic seizure and is found by his physician to have a pulse of 40 or 50 per minute. Weeks or months may pass before there is a second attack, and meanwhile the patient feels and looks well, and goes about his business as usual. There may be no subjective sensations about the heart, but the pulse remains permanently slow, and may sink to 20 or 25 and remain at that rate for years. Examination of the heart shews nothing abnormal, the sounds being clear and the beats strong and natural though slow. In a few cases they may be very feeble. Careful inspection usually shews the venous pulse in the neck to be 2, 3, or 4 times the rate of the carotid. Sometimes in the intervals between the cerebral attacks the pulse-rate is normal, but as a rule it is permanently slow, and may not vary more than a beat or two in rate per minute for years. Angina pectoris may complicate the cardiac condition, or there may be signs of myocarditis with oedema of the lungs, dyspnoea, and Cheyne-Stokes breathing ; but, once established, the even tenor of the cardiac rhythm is not often disturbed. In several cases the cerebral attacks are pseudo-apoplectic, as Stokes called them, and they may recur with great frequency, and, though they are apparently serious, the patient recovers quickly and may return to work as if nothing had happened.

The cerebral symptoms vary in different cases. Attacks resembling petit mal are perhaps the most common, with twitchings of the limbs and face. The epileptic fit with its orderly sequence of events is rare. A slight aura may precede an attack, and the patient may be able to ward it off ; after recurring for a year or more the attacks may cease ;

in other cases they become extraordinarily frequent, 30, 50, or even 150 in a day, and consist of brief periods of loss of consciousness with twitchings of the muscles. During these paroxysms the pulse-rate may fall to 6 or 8, and there may be prolonged intervals between the ventricular beats. After presenting the symptoms for a period of two to ten or more years the patient dies in an attack, drops dead suddenly, or dies in an attack of angina pectoris; more rarely he is carried off by heart failure or by an intercurrent disease. In the syphilitic cases complete recovery may follow proper treatment. This is the clinical picture of the average case, and anatomically there have been found lesions in the heart implicating the junctional system—either sclerosis of the Kent-His bundle or fatty degeneration, or a gumma or widespread changes in the system itself (*vide* p. 132).

B. *Neurogenous Group.*—In a second group of cases a lesion of the medulla or of the vagi causes slow pulse and convulsions. The following categories will be briefly mentioned :—

(1) In a fracture of the cervical spine or dislocation due to injury or disease, slow pulse is more commonly present alone, but the pressure may also cause transient loss of consciousness. A man in the Montreal General Hospital with tuberculous disease of the first and second cervical vertebrae had on several occasions attacks of syncope and slow pulse. I well remember when helping to put on a "jacket" to support his head that as the result of a sudden movement he became unconscious and the pulse dropped to 10 or 12 per minute, so that we were afraid he would die before the head could be so placed as to relieve the pressure. He died subsequently in an attack, and the necropsy shewed ulceration of the check ligaments.

(2) Of a very similar nature are the cases with narrowing by disease of the lumen of the vertebral canal. This was the condition in Holbertin's patient who had had a fall five years before, and was found to have enlargement of the odontoid process. In Lépine's case narrowing of the canal caused pressure on the left side of the medulla, and in another instance there was narrowing of the occipital foramen (Boffard).

(3) Tumours of the medulla, such as aneurysm, sarcoma, and gumma, and tumours in its neighbourhood, for example in the cerebellum, may cause slow pulse and syncopal attacks. Edes gives a long series of cases collected from the literature, a majority with slow pulse alone. In a sarcoma of the medulla which I reported the young man had had vertiginous attacks, but at the end he had syncope and a pulse-rate of from six to eight per minute. Neuburger and Edinger report the case of a man aged forty-six with vertigo and fainting fits, who for nine days before his death had deviation of the head and eyes and a pulse of 18 per minute ; the necropsy revealed absence of the right lobe of the cerebellum and a varix so situated that it might have irritated the vagus. In Brissaud's case there was a gumma in one cerebellar peduncle.

(4) In Triboulet and Gougerot's case marked atheroma of the vertebral and basilar arteries had produced a sclerosis of the medulla and pons

with atrophy of the cells, which shewed chromatolysis and a rounded outline.

(5) The vagi may be involved in a neuritis. The case of Zurhelle's (quoted by Pletnew) is possibly of this nature. In the second week of a febrile attack a man began to have pains on the left side of the neck and later on the right side increased by pressure. Swallowing was difficult. The heart became irregular, the pulse - rate gradually sank to 36 in the minute, and there were attacks of fainting with clonic contractions of the muscles. Then he had inflammation of the lungs, and a bilateral paralysis of the recurrent laryngeal nerves with huskiness of the voice. Recovery took place gradually.

(6) In the absence of careful investigation by modern methods of the medulla and heart we must for the present rule out the functional group into which such a case as the following, reported by Edes, has been placed. An excessively neurotic woman, aged fifty, had for seven or eight months recurring attacks of loss of consciousness, in which the pulse fell to 20 per minute. No morbid changes were found in the heart or brain ; but as a microscopical examination was not made, there may have been degenerative changes in the junctional system. The cases in this group require further study by modern methods. We do not know whether heart-block exists or not ; and I am not aware of any cases in which careful tracings have been taken and analysed.

Symptoms complained of by the Patient.—As a rule, it is a painless affection, and the cerebral features first call attention to its existence. Transient vertigo, a "die-away" feeling, an attack of fainting, or a more complete loss of consciousness with or without convulsions alarm the patient and make him seek medical advice. Morgagni's patient complained of pain in the right hypochondriac region ; in other cases headache and dyspnoea or pain on exertion may be present. Palpitation, a sense of cardiac oppression, and in a few cases attacks of angina have been recorded, but as a rule there are no unpleasant sensations about the heart. There may be evidence of cardiac failure in cyanosis, dyspnoea, and dropsy ; but these are rare, being present in two only of my series. The patients may become highly nervous and apprehensive, or even present a picture of aggravated neurasthenia.

The Cerebral Attacks. — The pulse may be slow for years without causing any discomfort, but so soon as giddiness or fainting or an epileptic fit occurs the patient becomes alarmed and seeks advice. Vertigo is one of the earliest and most common symptoms. On getting out of bed or in the street the patient experiences a sudden sensation as if he were about to fall, but he recovers himself easily. One of my patients (No. 2) had as many as twenty-five of these spells in the day, but never lost consciousness. The condition may be mistaken for the ordinary arteriosclerotic vertigo, so common in elderly people. In the neurasthenic group of cases of bradycardia, giddiness may be a very prominent feature. In one case (No. 14 of my series) a man aged twenty-six, with a pulse of 42, had attacks in which he felt "giddy-headed," and

everything in the room seemed to turn round ; when he tried to get up he was very unsteady, and was obliged to support himself by a chair. Transient mental confusion may be associated with the vertigo. In one case (No. 2) transient aphasia (a common symptom in cerebral arterio-sclerosis) not infrequently followed an attack.

Fainting.—Syncope is one of the most common features of the cases. Without warning, while about his work or in the street, or when speaking in public, the patient falls unconscious, usually with pallor or an ashen-grey hue of the face. The suddenness of onset, without the slightest premonition, is an interesting feature of the attack, and gives to it a cerebral rather than a cardiac character. On this point the late Sir William Gairdner wrote to me about himself, October 1903, "The sensations, so far as I could judge, were not those of swooning, and still less of any apoplectiform seizure, but rather, I would say, epilepti-form." The loss of consciousness may be for a few seconds only, so that the patient falls, but gets up immediately, or it may last a minute or two. The longest simple syncopal attack in any of my cases was ten minutes. There are remarkable attacks with all the associated pheno-mena of a fainting fit, but no loss of consciousness. One patient would turn pale, the hands and feet got cold, he sweated, and the heart became slower and more feeble, but in constantly recurring attacks of this sort he only twice actually fainted. As the late Sir William Gairdner insisted these are really epileptiform, that is cerebral, not syncopal in character.

Apoplectiform Attacks.—Stokes described these as pseudo-apoplecti-form, as the patient had all the appearance of having had a stroke, but in a little while recovered without any trace of paralysis. This is not a common form, and occurred in one only (No. 6) of my cases. A large full-blooded man had for ten years a slow pulse, vertigo, and occasional attacks in which he dropped unconscious, had stertorous breathing, and the pulse fell to 20 per minute. After lasting from five to fifteen minutes consciousness returned, and after a short interval he was able to go about his business. This patient had hundreds of these attacks, some-times two or three a week. Later he had transient losses of conscious-ness like petit mal, in which he did not fall, followed by motor aphasia of short duration.

Petit Mal.—The vertiginous attacks are sometimes of the nature of petit mal, and in some cases there are recurring periods of momentary dazing or a sensation of "dying-away." In others true petit mal occurs, transient loss of consciousness with pallor of the face, but without loss of control of the voluntary muscles so that the patient does not fall. Mental confusion may follow, but as a rule the individual knows when he has had an attack, which is not always the case in petit mal. "The absolute instantaneousness of the attacks, the absence of premonition and of all permanent results seemed to me more in harmony with a minor epilepsy than with either syncope or apoplexy . . . in some of the attacks in which the loss of consciousness was not absolute, and I retained

sufficient self-possession to watch carefully for the access (occurring as it did in some nights between twelve and twenty times), I seemed to realise in my consciousness something like a faint trace of an aura" (Gairdner).

Convulsive Attacks.—These may be mild or severe, either localised twitchings of the muscles of the face and arms, or general convulsions which have all the characters of a true epileptic attack. The mild seizures are the more common. In my series fourteen had attacks of unconsciousness only; four had slight movements of the muscles of the face or arms or both, and two had more severe attacks. Aurae may precede the attacks, but they are not so common as in true epilepsy—peculiar feelings in the head, buzzing in the ears, precordial distress, or a sensation starting from the heart, tingling or a sensation as of a wave of heat passing from the periphery, or a sensation of a lump in the stomach rising through the right side to the head, where it burst with a thunderclap (Stokes), are among the sensations which I find described. The following are descriptions of attacks, the onset of which I have seen:—
"The eyes were turned to the left, became fixed, and consciousness was lost, the muscles of the face twitched, and those of the hands worked in slight clonic movement. The face became pale. The heart-beats which had been 18 per minute sank to 12, but remained regular and forcible; the apex-beat could be seen. In about half a minute he regained consciousness and seemed quite himself." In another case, "The patient shook hands with me and spoke quite naturally; the pulse was 20 per minute. After I had finished the examination the pulse suddenly stopped at the wrist, and I could not feel the heart's impulse, the features became fixed, the face slightly cyanotic, the breath was held, there was a general tremble of the muscles with 'rigidity,' the eyes twitched, the eyeballs rolled up, and the hands moved slightly. Within half a minute he was conscious again, the pulse began its regular rhythm at about 20. The intervals in which no heart-beat could be felt or heard ranged from twenty to thirty-five seconds. The patient had scores of such attacks in the day—150 by actual count!"

The rate and vigour of the heart do not necessarily change in these attacks, but there may be a complete cessation of the heart's action for from half a minute to two minutes and ten seconds in Stengel's case. The loss of consciousness is rarely more than a few minutes, but it may last half an hour, and Strayesko's patient remained unconscious for thirty-six hours. The breathing may be impeded for a few seconds, sometimes it is quickened; Cheyne-Stokes rhythm is not infrequent, and may begin during a prolonged attack. In some cases the slight epileptiform seizures occur daily with great regularity, one or two in the day. A patient seen with Dr. Turrell had them most often just as he was taking his breakfast. In other cases the attacks occur at long intervals. After persisting for years they may cease entirely. Mental excitement, bodily exertion, and flatulence are liable to bring on the attacks. One patient never had them when resting quietly, but when up and about or if he tried to do a little gardening the attacks recurred. The attacks

may sometimes be averted. When Stokes' patient felt the symptoms approaching he turned on his hands and knees, keeping the head low, and in this way prevented a seizure. Dr. Turrell's patient could sometimes stop the attack if he could in time grasp firmly the bar of the bedstead, just above his head. Case 12 of my series could sometimes keep himself from fainting by rubbing the wrist forcibly.

Cardiovascular Features.—A majority of the cases in my series had arteriosclerosis, usually the senile form. As Stokes remarked, the permanently slow arterial pulse is the special characteristic of the condition. It is usually under 40 per minute and regular. In a few cases it is between 40 and 60, whilst some patients have a pulse-rate between 20 and 30 for long periods.

Dr. Turrell's patient had the pulse taken by a nurse three times a day for eight years—a unique record which is worth quoting (22). The records begin on July 25, 1900, some few days after a series of convulsions ; the rate was then 38 to 40. On July 26, 1900, it became quicker, about 53 to 55. From that date on it ranged about 60, never being below 50 and never above 72 till September 21 of the same year, when a rate of 40 was recorded. On October 1 a pulse-rate of 32 was recorded, and from that date the rate was for the most part between 30 and 40, except for intervals of two or three weeks, when it was 50 to 60. From October 31, 1901, it was below 40, except for an isolated observation on July 22, 1904, when it was 68 after an attack of convulsions. During this period up to the patient's death the rate was never observed below 16—that is, apart from the convulsive attacks—and seldom above 37.

In Case 20 of my series the slow pulse only came on at the time of the attacks, and in the intervals the rate was normal. He passed an entire year without an attack, and the pulse was always above 70 ; then for two months it was between 25 and 28, and the syncopal attacks recurred.

In the senile form the pulse becomes permanently slow, and the patient may be perfectly comfortable for years with a rate of from 25 to 30 per minute. The rhythm is nearly always regular, but a few cases shew extra-systoles. The arterial pulse-rate is not easily influenced when once permanent bradycardia is established ; thus emotion and exercise may not raise the rate more than a few beats. After the cerebral attack, however, the rate may be quickened, and a bout of indigestion or a febrile attack may have the same effect. The pulse-rate may remain the same in the recumbent and standing postures, and before and after exercise. As a rule atropine does not produce any quickening of the ventricular rate in the cardiac cases, whereas in bradycardia of vago-medullary origin the pulse is accelerated.

The Pulse in Relation to the Cerebral Attacks.—As Dr. A. Webster pointed out, a slower pulse usually accompanies the attack, for example, a rate of 20 or 25 per minute may fall to 10 or 12. In a severe attack, particularly in cases in which many occur in the day, the radial pulse and the heart-beats may be imperceptible for some seconds, or even for more than two minutes, as in Stengel's case. In some cases the attacks follow

a drop of 8 or 10 beats per minute; on the other hand, when a permanently slow rate is established, say at 25, attacks may be more likely to come on, as in Dr. Turrell's patient, when the pulse rises to 30.   In this case irregularity had a marked influence; an occasional drop made no difference, but if the pulse missed two beats in succession the patient had to make a great effort to retain consciousness, and often an attack followed.   The very slow beats may be extraordinarily vigorous and convey a sense of fulness, which corresponds with the powerful action of the heart.

The Venous Pulse.—Stokes noted in his case the remarkable pulsations of the jugular vein which were more than double the ventricular contractions.   When the patient is thin this peculiarity is constantly present, and its investigation by the modern graphic methods has thrown much
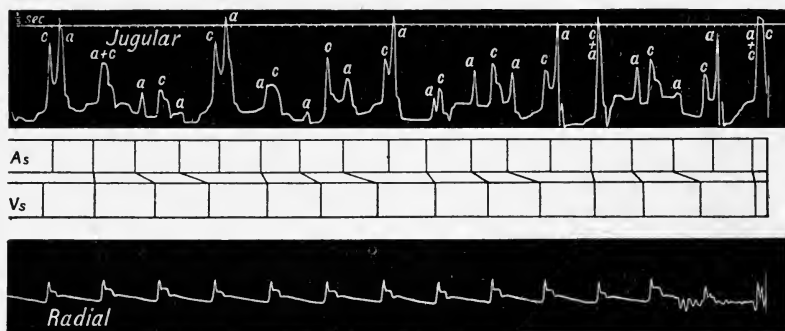
Fig. 25.—The upper tracing is from the jugular pulsation, the lower from the radial artery; the interpolated diagram between represents graphically the beats of the auricle and ventricle.   *a* and *c* in the jugular pulse represent the waves due to the auricle and the carotid artery respectively. As.=auricular systole; Vs.=ventricular systole.   The diagram shews an increasing difficulty during three beats in the transmission of the auricular contraction to the ventricle, followed by an auricular beat which fails to reach the ventricle.   From a case of Stokes-Adams disease with partial heart-block.   (A. G. Gibson.)

light on the physiology of the condition.   Sometimes an undulatory impulse only is seen, or a series of beats following each other so rapidly that it is hard to distinguish them; whereas in other cases the beats are readily counted; but the graphic method enables the rhythm to be studied accurately.   The accompanying tracings with analyses will give a clear conception of the relation of the venous and arterial pulses.

The heart may not shew anything abnormal on inspection.   In 11 of my cases the apex-beat was visible; in 9 it was not seen; in 5 the impulse was forcible and outside the nipple line.   Sometimes, as in Case 5 of my series, although there was no cardiac hypertrophy the impulse was very strong, and shook the chest.   A wavy impulse has been seen in the diastolic period over the precordia.   An ordinary normal impulse may be felt, only the beats are slow; in other cases there is no palpable pulsation, even after exertion; in other instances again a gallop rhythm may be both seen and felt.   The shock of the first sound may be very intense and a

sharp diastolic snap at the base may be felt; with valvular disease or with senile sclerosis a thrill may be present, usually at the base (Case 2). The area of cardiac dulness may be normal, or in the aged diminished by emphysema; when increased it is usually in association with valvular disease. In only 6 of my cases was the heart evidently hypertrophied.

Auscultation.—The sounds may be normal in tone and in relative intensity; this applies to a majority of the patients, particularly in the intervals between the cerebral attacks. The sounds may be muffled or quite inaudible. In Case 4 of my series it was impossible at times to hear the sounds; after exertion a feeble first could be heard with a soft murmur, but I repeatedly examined him when it was impossible to hear

FIG. 26.—The respirations and the jugular and radial pulses, from the same case as Fig. 25, about three months later when complete heart-block had been established. The auricle is beating regularly at a normal rhythm; the ventricle pursues its own rhythm, unaffected by the auricle as is shewn by the dropping out of one ventricular systole. a=auricular systole; c=the carotid wave. Time-marker=⅕ sec. (A. G. Gibson.)

anything at the apex or base. On the other hand the sounds may be exaggerated, the first either clear or valvular, like the ringing, flapping sound in mitral stenosis; or a loud booming cannon-tone of extraordinary intensity. The second sound at the base may be greatly accentuated, and of a bell-like amphoric quality. Disease of the myocardium and valves may be accompanied by the usual phenomena, such as gallop rhythm, which was present in several of my cases; in 8 a mitral systolic murmur, in 2 an aortic systolic, and in Cases 10 and 15 the signs of aortic insufficiency were present. A terminal pericarditis occurred in Case 8.

In several cases soft sounds were heard in the interval between the ordinary heart-beats. In Case 5 a faint systolic sound was heard after the loud first; when this patient was having many fainting and epileptiform attacks with a radial pulse of 12, very loud booming heart-sounds, and a fluttering jugular pulse of about 120 per minute, there were seen in the 4th and 5th interspaces "small regular systolic impulses exactly 100 to the minute, and corresponding to these would be heard faint systolic

sounds at the same rate." In Case 6 feeble tones were heard in the long diastolic interval, but I could never determine that these soft intervening sounds corresponded accurately with the auricular beats, and they are not heard in a majority of the cases, even when the pulse in the neck is well marked.

During the attacks the pulse-rate may not alter, but as a rule the heart beats more slowly, and there may be prolonged intervals in which no impulse or sounds are perceptible. Thirty-five seconds was the longest interval I have noted, but in Stengel's case neither sound nor impulse could be determined for more than two minutes. The impulse may be much more forcible during the attack ; in one case of my series (No. 5) the heart-beats, at 12 or 15 per minute, shook the front of chest with each impulse, and the first sound had a booming cannon-like quality. By means of skiagraphy Batjer was able to see clearly the auricles beating without the ventricles in one of my cases (No. 17). In one of Dr. G. A. Gibson's cases the auricular beats were seen on the electro-cardiogram.

Pulmonary Features.—Cases with chronic myocarditis present the usual symptoms of this condition, attacks of cardiac dyspnoea, cough, or angina pectoris. In several cases I have seen the acute emphysema which Dr. Goodhart describes in angina pectoris, a state in which the lungs are full of wheezing and bubbling rales, and the area of pulmonary percussion is increased—the *Lungenschwellung* of von Basch. Cheyne-Stokes breathing is not uncommon, and was present in 3 of my series. In the pseudo-apoplectic attacks the stertor, with deep laboured respiration and expiratory puffing of the cheeks, may have all the intensity of the genuine stroke. Before the epileptiform attacks there may be transient arrest of respiration with flushing of the face.

Clinical Course.—(i.) *The Cardiac Group.*—The cases associated with post-febrile myocarditis form a very definite and remarkable category. Cases have been reported in connexion with diphtheria, pneumonia, gonorrhoeal infection, and streptococcic pharyngitis complicated with nephritis. It has long been known that bradycardia may follow any of the acute infections. Usually regarded as an indication of myocarditis, it is quite possible that the nerve-centres are implicated, but in the reports with the Stokes-Adams syndrome there were no symptoms to suggest a lesion of the medulla. Next to the syphilitic this form is the most hopeful. Schuster's patient, a child aged four years, recovered after very severe attacks. The slow pulse may last for a few days only, as in Case 1 of my series in which the condition followed pharyngitis and nephritis. In some of the post-febrile cases the bundle of His is involved in an acute mural endocarditis.

The syphilitic category is also well-defined; the lesion, whether arterial or a coarse gumma, may invade the bundle. In my series 2 cases only had a history of syphilis. The picture may be very typical. A man (No. 17), aged thirty-four, was seen, November 17, 1904, with attacks of loss of consciousness and slow pulse. He had had syphilis in 1897, and a gumma of one rib in 1901. The heart-block, at first complete, was care-

fully studied by Erlanger, in whose paper the case is given in full. While under observation he had five or six severe syncopal attacks. He recovered rapidly on iodide of potassium, gaining twenty pounds in three months. Both cerebral and cardiac symptoms disappeared and he has remained well, his heart being normal when I examined him in January 1906.

By far the largest group is made up of the cases in which there are degenerative changes, fibrous, fatty, or pigmentary, at the auriculoventricular node, in the bundle of His, or in the ramifications of the junctional system. A diagnosis of the nature of the lesion is not possible. The cases occur in older individuals than in the other groups, and in a large proportion the anatomical change is an atrophy due to arteriosclerosis. In Case 19 (Turrell and Gibson) of my series, the coronary arteries were sclerosed and partially calcified. The bundle of His and its main ramifications were implicated in fibrous tissue, the bundle itself being represented only by a few indolent strands of muscular tissue. A majority of the senile cases will probably be found to present this kind of lesion. In a group of cases (Schmoll, Gibson, Stengel, and Dock), the bundle of His was implicated in a patch of chronic endocarditis. Degenerative changes of a fatty nature were present in the case of Adams, and of recent cases those of Butler and of Aschoff. In Case 20 of my series Dr. A. G. Gibson found a widespread brown atrophy in the fibres of the system.

The cases of the arteriosclerotic group present certain special features :—

Extraordinary Chronicity.—Although from five to six years may be taken as the average duration, in some cases the symptoms have persisted for twenty years or more. Heineke's first case was a women, aged sixty-four years, who had suffered from her thirtieth year, and in Case 7 of my series the attacks had lasted for more than ten years.

A Remarkable Variability in the Frequency and Character of the Cerebral Attacks.—After causing a great deal of trouble and worry the syncopal and epileptiform seizures may disappear, and for years, as with the late Sir William Gairdner, the patient may be very comfortable. Isolated epileptic attacks may occur over a long period of years. One patient had 9 attacks only in twelve years, and in the intervals enjoyed good health. Possibly Napoleon may have had this disease, as he is said to have had a slow pulse, and he is known to have had epilepsy.

When the attacks recur with great frequency, 50, 100, or more times in a day, the condition is serious. The prolonged syncope, with slowing of the pulse to 8 or 10, and the attacks with protracted intervals of asystole are always dangerous. The pseudo-apoplectic attack, which looks so serious, is one of the least ominous ; Adams' patient had them for more than seven years, and Case 7 of my series for more than ten. As already mentioned, certain circumstances are apt to bring on the attacks, but we are quite at a loss to say why, with an apparently unchanged cardiovascular condition, the cerebral symptoms should disappear.

The Liability to Sudden Death.—As in all myocardial affections this accident is very likely to happen, either in an attack of syncope or while the patient is up and about and feeling very well. This occurred in 10 cases in my series.

Ordinary Myocardial Symptoms.—Dyspnoea on exertion, cough, signs of oedema of the bases of the lungs occur in a few cases, but it is remarkable for how many years the efficiency of the heart may be maintained with a ventricular rate under 40, the cerebral attacks alone indicating any disturbance of the cardiovascular function. Angina pectoris has been present in a few recorded cases; the attacks *sine dolore* have much in common with the syncopal seizures in Stokes-Adams disease, and the pulse may fall to 20 or 30, or there may be the same prolonged period of asystole.

The duration depends upon the cause. The acute myocardial forms are of short duration, but the arteriosclerotic and degenerative cases last for from 8 to 10 or 12 years, and in the intervals between the cerebral attacks the patients may be very well, and able to work, and enjoy life.

(ii.) *The Neurogenous Group.*—After injury to the cervical spine or caries of the 1st and 2nd cervical vertebrae bradycardia is common, but cerebral features are exceptional, and there are only a few well-marked cases with the Stokes-Adams syndrome. With gradual narrowing of the upper part of the spinal canal, as in the cases of Holbertin, Lépine, and Boffard, the condition may be more chronic, and recurring attacks of syncope give a very characteristic picture. The cases of meningitis (chronic and gummatous), tumour, and abscess usually present the localising symptoms due to pressure on other parts. The cases are very rare; Edes pointed out in his collected series that bradycardia is common, but the combination of slow pulse with characteristic syncopal and epileptiform seizures is exceptional.

Diagnosis.—There is rarely any difficulty in recognising the cases. In a patient seen for the first time in an attack of syncope or of pseudo-apoplexy the slow pulse should suggest the condition, as neither in true apoplexy nor epilepsy is bradycardia a prominent feature. The prompt recovery and the slight after-effects are peculiarities which distinguish the attacks from true epilepsy and apoplexy. There is a large group of borderland cases of bradycardia with occasional vertiginous attacks—*formes frustes* of the French. Some of these are very remarkable, as heart-block may be present for years without cerebral symptoms. In a man of sixty-eight, after an attack of pain beneath the sternum and slight vertigo, the pulse was noted to be slow, and for four years it remained at about 32, the auricular pulse as counted in the jugular vein being about double this rate. In senile bradycardia, with the pulse below 60, transient vertigo or syncope is not uncommon. In arteriosclerosis at any age vertigo may be an early symptom, but it is not necessarily associated with a slow pulse. The question of the existence of true epilepsy may arise, as in Burnett's case, a naval officer who sixteen years previously had had an epileptic attack and then at the age of forty-six

had bradycardia with paroxysms of an epileptiform character. In Case 15 of my series a man of sixty had, at the age of forty-eight, a convulsion, and in twelve years had nine attacks. In the attack in September 1902 the pulse was noted to be very slow. When I saw him in October 1908 he had a pulse of 40, which had been the usual rate for a year. He had moderate arteriosclerosis, slight hypertrophy of the heart, and a soft diastolic murmur at the base. It is quite possible that the attacks at rare intervals during the twelve years were of the Stokes-Adams character, and his physican did not think there had been any change in their nature, though he had never been able to see one.

Slow pulse may be associated with neurasthenia, and there is a group of cases with certain of the features of Stokes-Adams disease. As already mentioned (p. 146), it is doubtful if Edes' well-known case should come in the group. I have reported 2 cases, both in typical neurasthenics, with pulses under 50 ; one patient, aged forty-four, had peculiar swaying attacks with pains in the head ; the other had vertigo of a severe character. Both patients recovered. A third patient, a man aged thirty, very healthy and strong but excessively neurotic, felt faint while in church, so that he had to sit down. The attacks recurred for three months, one or two in a week. The pulse was 44 ; there was no evidence of arteriosclerosis, and the heart was normal ; no jugular pulse could be seen as he was very stout. For more than a year the pulse-rate was under 50. He gradually improved, and when last heard of, on June 6, 1906, three years after my first note on his case, he was quite well.

Treatment.—In the post-febrile form the patient should be kept in bed. Small doses of iodide of potassium may be of use, but it is very doubtful if any medicine has an influence on acute myocarditis. With rest a majority of the cases recover. The syphilitic cases require thorough treatment ; Case 17 in my series recovered rapidly on moderate doses of iodide of potassium. To any young man who admits exposure, it is well to give the benefit of the doubt and a thorough antisyphilitic treatment, inasmuch as gummas and arteritis may clear away and leave no damage. In the large group of arteriosclerotic cases in middle-aged men the life should be carefully regulated, moderation in food and drink and exercise enjoined, and, if the blood-pressure is high, nitroglycerin or sodium nitrite may be used. Iodide of potassium may retard the progress of the sclerosis.

There is no satisfactory method of accelerating the pulse-rate. The rhythm may return to normal for months without any special treatment. Atropine may be tried hypodermically, beginning with doses of $\frac{1}{100}$ of a grain, and, if this be well borne, the amount may be increased. Dehio, Gibson, and others have reported good results, but when once the heart-block is established in a man over seventy, nothing seems to influence it. Strychnine may be tried ; a patient whom I saw in Oxford had great faith in it, and attributed the long intervals of freedom from attacks to its use. Digitalis is rarely called for, though in Case 7 of my series, a

patient with an extremely feeble heart action and signs of oedema of the bases of the lung, it gave relief.

During an attack the usual restorative measure may be employed. The transient syncopes are usually over before anything can be done. A bottle with smelling salts may help to avert an attack. In the prolonged syncope with reduction of the heart-beats hypodermic injections of ether or of strychnine, strong electrical currents over the heart, and inhalations of oxygen may be tried. Nitrite of amyl does not appear to be of much service, but it may be used in the presence of pallor and marked vaso-motor disturbances.

Patients should be encouraged to watch for the earliest symptoms of the cerebral attacks, and attempt to ward them off by a sudden movement or a sharp stimulus to the skin. Blisters over the heart may be used.

<div align="right">W. Osler.</div>

REFERENCES

1. Adams. *Dublin Hosp. Rep.*, 1827, iv. 396.—1a. Bachmann. "Complete Auriculo-ventricular Dissociation without Syncopal or Epileptiform Fits," *Amer. Journ. Med. Sc.*, Phila., 1909, cxxxvii. 342.—2. Boffard. *Arch. de méd. expér. et d'anat. path.*, Paris, 1890, ii. 90. — 3. Brissaud. *Leçons sur les maladies nerveuses*, 1899.—4. Charcot. *Leçons sur les maladies du système nerveux*, 1872, t. ii. 135.—5. Edes, R. T. *Trans. Assoc. Am. Physicians*, 1901, xvi. 521.—5a. Gerhardt. "Ueber Rückbildung des Adams-Stokes'schen Symptomenkomplexes," *Deutsches Arch. f. klin. Med.*, 1908, xciii. 485.—6. Gibson, G. A. *Brit. Med. Journ.*, 1906, ii. 22.—6a. Heineke, Müller, v. Hösslin. "Zur Kasuistik des Adams-Stokes'schen Symptomkomplexes und der Ueberleitungsstörungen," *Deutsches Arch. f. klin. Med.*, Leipzig, 1908, xciii. 459.—7. His, W., junior. *Ibid.*, 1899, lxiv. 316.—8. Holberton. *Med.-Chir. Trans.*, London, 1841, xxiv. 76.—9. Huchard. *Traité des maladies du cœur*, 1889.—10. Jacquet. *Deutsch. Arch. f. klin. Med.*, Leipzig, 1902, lxxii. 177.—11. Kussmaul. *Aus meiner Docentenzeit in Heidelberg*, 1893, 28.—12. Lépine. *Lyon méd.*, 1889, xliii. 315.—13. Luce. *Deutsch. Arch. f. klin. Med.*, Leipzig, 1902, lxxiv. 370.—14. Morgagni. *De Sedibus et Causis Morborum*, Alexander's Transl., London, 1769, i. 192.—15. Neuburger und Edinger. *Berlin. klin. Wchnschr.*, 1898.—16. Osler. *Lancet*, London, 1903, ii. 516.—16a. Pletnew. Lubarsch u. Ostertag's *Ergebnisse*, 1908.—17. Prentiss. *Trans. Assoc. Am. Physicians*, 1889, iv. 120; 1890, v. 185; 1891, vi. 258.—18. Schuster. *Deutsch. med. Wchnschr.*, 1896, xxii. 484.—19. Stokes. *Dublin Quart. Journ. Med. Sc.*, 1846, ii. 73. — 20. Tanhoffer. *Centralbl. f. med. Wissensch.*, Berlin, 1875, xxiii. 403.—21. Triboulet et Gougerot. *Arch. gén. de méd.*, Paris, 1906, ii. 2579.—22. Turrell and Gibson, A. G. *Brit. Med. Journ.*, 1908, ii. 1486.—23. Webster, A. *Glasgow Hosp. Rep.*, 1900, iii. 413.

<div align="right">W. O.</div>

ANGINA PECTORIS

By Sir R. Douglas Powell, Bart., K.C.V.O., M.D., F.R.C.P.

Definition.—A sudden paroxysmal disturbance of the heart's function accompanied by severe pain, distressed breathing, and a vague or instant apprehension of death.

Angina pectoris is a disease that has probably existed longer than there is any historical record of it, and has ever claimed its victims amongst those who have relinquished the simple life for the more complex conditions of civilisation. It is a malady of fairly frequent occurrence amongst those classes who work with their brains at high pressure, and who employ their so-called leisure in social duties which are often even more exacting than the labour proper to the day. And, with other diseases of nervous tension and arterial degeneration, it is likely to become of increasing prevalence, as through improved sanitation more persons live to beyond middle life, and the stress of commercial and professional work becomes more severe.

It is perhaps because of its tendency to prevail amongst the more prominent official and commercial classes that the disease has attracted so much attention, and that the appalling symptoms of its graver and fatal examples have been recorded in such picturesque detail. But although comparatively rarely recognised in hospital practice, it is nevertheless to be met with amongst those who toil with their hands and have not the gift of fluent language to describe their sufferings. Their cases are included amongst the more general group of heart failures.

The first record of the symptoms of angina pectoris is probably that in which the Earl of Clarendon in his memoirs describes the seizures and sudden death of his father in 1632. The classical description of the disease by Heberden in 1772 remains to us to-day in all the strength of portraiture by an old master, and the pathology insisted upon as essential by Edward Jenner and Parry remains true for the grave and fatal cases which were alone considered in Heberden's description. But the advancement of our knowledge within the past fifty years of the physiology and pathology of the cardiovascular system, its mechanism, innervation and degenerations, has rendered a wider canvas necessary to depict angina pectoris in its larger aspects and its true relationships. It is in this regard unfortunate that angina pectoris should, from the notoriety of its illustrations, have become in the public mind almost synonymous with an imminent and sudden death, for no one who has carefully observed any considerable number of cases can fail to recognise that a most exquisite counterpart of the symptoms of the gravest form of the malady may be presented by one who yet lacks what has been regarded as the essential factor in its pathology, namely, obstructive disease of the coronary

arteries of the heart.   The bravest man has an organic apprehension of death, and he who is sick unto death endures a higher degree of anguish than he who may suffer more unto recovery.   Hence to be able in some cases to remove the dread of impending death from the mere physical suffering of angina is an achievement that more than justifies the most careful pathological classification and study.   Reeder, in 1821, seems to have been the first to draw a distinction between so-called true and false angina, but it could not be until physiological knowledge, especially of cardiovascular innervation, was much farther advanced that Nothnagel and Eulenburg, and particularly the former, were in a position to establish vasomotor angina as a definite group of symptoms.

The observations of Landois (1865) and Ross (1885), and particularly the researches of Dr. Gaskell on cardiac and vascular innervation have rendered it possible to give a better explanation of the phenomena of angina, and more recently the anatomical and clinical observations of Dr. A. Keith, Dr. James Mackenzie, and Tawara upon the heart have shewn that those specialised, or perhaps speaking more accurately those more primitive, fibres which constitute the bundle of His and its ramifications are intimately concerned in its healthy rhythmic function and with those disturbances of it which are found in some forms of angina.

There are some elemental considerations about the circulation to which I would draw the attention of the reader.   They may be enumerated in a few brief propositions.   (1) The circulatory system should always be regarded clinically as a whole, of which the heart is but a specialised part—the most active part ; the other—arterio-venous—portions of the system are, however, by no means passive.   (2) The elastic contractile arteries and arterioles conserve and control the output of cardiac force, direct here or there the blood-current, flushing or rendering pallid various organs or parts in accordance with functional requirements or excitations messaged through the vasomotor nervous centres. The old saying *ubi irritatio ibi fluxus* expressed the latter part of this proposition in terms of strict but then undefined accuracy.   (3) The blood may be said to be held in the grasp of the cardiovascular system on the arterial side between the aortic valves and the terminal arterioles ; and as in death we see the arteries empty entirely into the veins, so in the vasomotor relaxation of some diseased conditions and under the influence of some drugs they may bleed into the great abdominal veins even to the production of syncope.   (4) The cardiovascular system is thus but half full and the blood is in truth held in the somewhat *tightened* grasp of the circulation on the arterial side, with a degree of tension varying from time to time, realising an average blood-pressure in the arteries of from 130 to 180 mm. of Hg.   (5) The veins, however, are not altogether passive.   They have valves and are in touch with compressing muscles, and, under the support and pressure of muscular action, the venous blood is moved on towards the heart with varying swiftness.   Regulated exercise is thus an aid, not an embarrassment to the circulation.   (6) Through the negative pressure that obtains within the pleurae and mediastinum in

obedience to the reacting tractions of lung and parietes, there is a constant aspiration of venous blood towards the cavities of the heart, increased during inspiration, lessened during expiration, momentarily overcome during ventricular contraction, but helping to fill the auricles and ventricles during the diastolic pause. The coronary circulation of the heart itself is, it must be remembered, aided by this aspiratory force which is, *pro tanto*, persistent so long as the lung elasticity is not impaired, by age, permanent disease, or temporary disablement. (7) The heart retains amid its complexity some primitive features and perhaps some islets of primordial tissue-structure. Its rhythmic contractility, derived from its original plasmic construction and retained through its tubal and later developments, although now so largely regulated and interfered with by an extrinsic nervous mechanism, still dwells as a primal function in certain portions of the auricles and in the septum with its conducting strands extending therefrom. The great importance of this fact has been disclosed to us by the labours of Gaskell, His, Kölliker, and more recently, by the interesting clinical, histological, and pathological researches of Drs. Mackenzie, Keith and Flack, and Tawara (see p. 139). (8) I need only further allude to the effect of the very large extra-vascular secretion and absorption of tissue and digestive fluids upon the mechanism of the blood-circulation.

Angina pectoris manifests itself in three forms :—(i.) In the first group are to be found those cases in which the disease is a pure neurosis of the cardiovascular system, a disturbance of the innervation of the systemic vessels, sometimes including as alleged by some authors the vessels of the heart itself, causing their spasmodic contraction and thus increasing the resistance to circulation. The sudden excessive demand upon the propelling power of the heart thus occasioned produces a more or less painful embarrassment of its action. We speak of this variety as *angina pectoris vasomotoria*, and we have to inquire into the conditions which lead up to such disturbed innervation and its consequences. (ii.) Another and graver class of cases presents precisely the same mechanism of disturbed cardiovascular innervation, but associated with it a diseased heart, either a texturally damaged heart-muscle or a valvular defect, or both combined. This form may be designated *angina pectoris vasomotoria gravior*, or *secondary cardiac angina*. (iii.) A third group of cases contains well-defined forms of disease in which the heart itself is the primary seat of the painful and often fatal symptoms. In these cases also the cardiac lesion may be valvular or textural. We may distinguish this third group by the designation *primary cardiac angina*. It includes certain cases of obstructive cardiac disease, especially cases of aortic or mitral narrowing, cases of textural degeneration and ischaemia of the heart, generally dependent on coronary narrowing ; and, lastly, cases of fatal syncope dependent upon degenerated heart, which are unattended with other anginal phenomena.

We have thus two fairly definite kinds of angina pectoris :—(i.) Angina pectoris vasomotoria. (ii.) Angina pectoris gravior ; which latter is divisible into (*a*) secondary cardiac angina ; (*b*) primary cardiac angina.

I have endeavoured to indicate in the following table-diagram the relations of these three principal forms of angina, and to shew that whilst they are very distinct, as illustrated by their characteristic cases, they merge into one another in their marginal examples.   The first group consists of cases of pure angina pectoris vasomotoria in which the heart is sound.   The second group consists of cases in which vasomotor spasm of vessels is still a very prominent feature in the clinical picture, but the heart is unsound from valvular disease or textural defect.   In the third
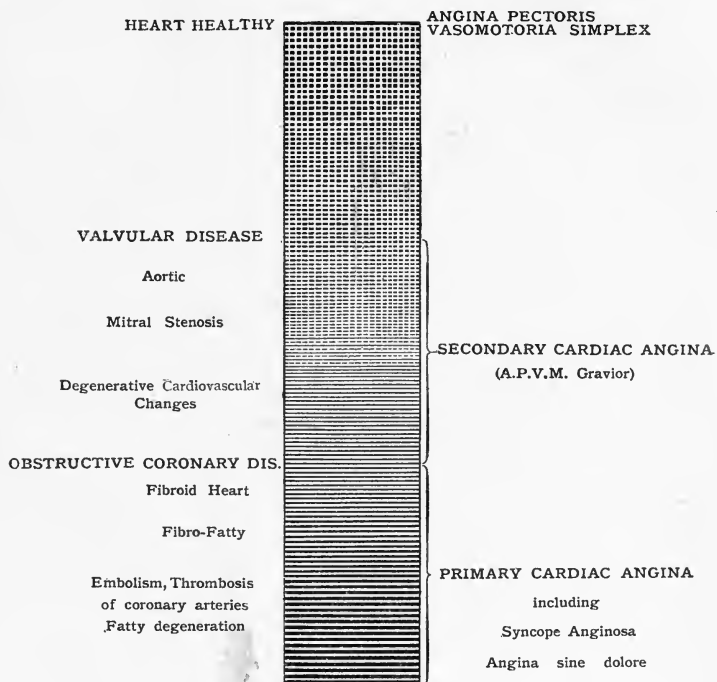
Fig. 27.

group there is textural degeneration of the heart arising from coronary obstruction, but there are no attendant vasomotor phenomena to be observed.

My case books of the ten years 1898-1908 contain 96 cases of angina pectoris ; of these 26 belong to the first group, 36 to the second, and 34 to the third.   Whilst I have not had much difficulty in making a well-defined group of the first, it is impossible to separate the second and third groups with any sharpness of definition ; on the border lines it is a mere matter of account in which group individual cases should be placed, whilst the typical cases of each group are clearly to be differentiated.

I. Angina Pectoris Vasomotoria

Syn. :—*Pseudangina.*

Etiology.—*Age.*—Vasomotor angina may occur at any age, but it is rare before twenty ; the cases are about evenly scattered between the ages of twenty-five and fifty, but some are met with in advanced life.

Sex.—This form of the disease is much more prevalent in the female sex ; taking my experience of cases in the ten years 1898-1908, the proportion has been 20 females to 6 males. The cases seen in advanced life are almost exclusively in the female sex.

Hereditary Tendency.—A very distinct hereditary tendency may be observed in this as in the graver form of the disease. Eulenburg speaks of vasomotor angina as alternating in persons and in families with a history of insanity or epilepsy ; and certainly an instability of the nervous system and the occurrence of such diseases as asthma and the neuralgias may be frequently observed to be associated with it.

Climate ; Temperature.—Although it has not been shewn that climate has any important influence upon the prevalence of angina pectoris, it is quite certain that cold, in the sense of getting chilled, is a most important cause, whether immediate or remote, of attacks of angina in all varieties, but especially in the vasomotor forms. Riding or walking against a cold wind, sleeping in cold rooms, getting the extremities chilled, are causes frequently assigned. Sudden immersion in cold water, or remaining too long in water, will cause an attack in the predisposed ; and there can be little doubt that attacks of " cramp " in the water are sometimes anginal in nature.

Occupation.—Probably all practising physicians will accept Huchard's statement that those occupations and professions—such as finance, politics, medicine—in which, through nervous strain, arterial pressure ranges high, are favourable to arterial disease. Prior to any organic alteration they favour the occurrence of vasomotor disturbance tending to angina.

Emotion. — Emotional disturbance, fright, pain, sudden shocks of sorrow will produce attacks, and are thus sufficient causes.

Dyspepsia—Constipation.—Dyspepsia is a frequent exciting cause of anginal attacks, and particularly those forms of it which are attended with flatulent distension of the stomach or colon. It must be remembered, however, that flatulent distension is also a frequent concomitant of anginal paroxysms, no doubt as a reflected or an associated nervous phenomenon. Constipation is a very frequent cause of high arterial blood-pressure and favours attacks in this way.

Gout enters into the etiology of angina by engendering a high arterial pressure. It is, however, commonly associated with some permanent cardiovascular changes, which render the patient liable to anginal distress from slight accessory causes.

M

Uraemia.—The " heart asthma " attacks of chronic uraemia are also generally associated with permanent cardiovascular changes.

Toxaemia.—Various drugs which raise arterial pressure by exciting the vasomotor mechanism tend to favour the occurrence of this form of angina.   Nicotine is of importance amongst them (*vide* p. 163).   Strychnine and the digitalis class must be used with caution in this regard in anginal cases.

Influenza.—Amongst the pure neuroses of influenzal epidemics angina pectoris has been frequently met with.   In my experience it has been generally vasomotor, and associated with a remarkable excess of urea in the urine.

Pathology.—Angina pectoris vasomotoria cannot be said to have any morbid anatomy.   It is a disordered innervation of the vessels, peripheral or visceral, resulting in their contraction, causing an increased pressure of blood in the cavities of the heart, and a consequently embarrassed action with pain and dyspnoea.   The texture and valves of the heart are sound, although in cases which have extended over years there may be some secondary changes in the heart and vessels attributable to long-continued high blood-pressure ; just as in asthma we find, after a time, secondary textural changes in the lung consequent upon the bronchial spasm which was originally a pure neurosis.   The nerves concerned are the sympathetic, the vagus, the depressor nerve, the vaso-constrictor and vaso-dilator nerves of the blood-vessels, and any nerve which conducts afferent impulses to the central nervous system.

The writings and clinical insistence of George Johnson pioneered the recognition of the influence of the arterioles upon the general circulation, whilst the works of Broadbent, Nothnagel, Eulenburg, and especially the researches of Dr. Gaskell, have made us familiar with vasomotor mechanism.

Those conditions, such as toxic influences and the effects of certain drugs which increase general arterial pressure, render the patient liable to the occurrence of that accession of contraction of peripheral vessels which shall cause acute cardiac embarrassment.   Thus a man with an arterial pressure ranging about 180 or even 200 mm. Hg may at any time acquire the condition of angina.   Many neurotic states keep the vessel innervation in the same sensitive condition.   The first effect of sudden increase of intracardiac pressure is to stimulate the cardiac muscle to rapid and often disorderly efforts of the heart to overcome the resistance ; efforts, however, attended with but imperfect success.   As the intracardiac pressure rises the depressor nerve becomes in turn excited, and inhibits sympathetic activity ; the vessels are relaxed, the pressure is relieved, and the heart's action is restored to regularity.

It has been shewn by Dr. Gaskell that fatigue of cardiac muscle, as of other muscles, causes liability to cramp ; and that accumulation of products of waste in the cardiac muscle renders its tissues unduly acid, and thereby liable to paralysis.   Whilst these considerations are of no great practical importance in reference to attacks of pure vasomotor angina,

unaccompanied by any organic disablement of the heart, beyond explaining the sense of fatigue and often of prostration that follows such seizures, they are of the utmost importance in the graver cases of the next category, and explain the fatal termination of many of them.

Whilst angina pectoris has distinct pathological relations with ordinary high arterial blood-pressure and with such extreme degrees of vessel constriction as are observed in Raynaud's disease, there are, on the other hand, many cases in which secondary cardiovascular changes have resulted from long-continued high arterial pressure, and in which the final symptoms are those of cardiac distress with anginal paroxysms due to accessions of arterial spasm or to failure of cardiac power. Such cases are witnessed in connexion with chronic interstitial nephritis when the malady is not cut short by acute uraemic phenomena.

The effect of nicotine upon the small vessels and heart seems to be a matter of considerable uncertainty; Claude Bernard finding contraction of the vessels, whilst other experimental observers find dilatation to follow its use. This discrepancy seems to be very much a matter of dose; the physiological action of nicotine is, however, currently regarded as contracting the blood-vessels (Brunton). Clinically, one finds varied arrhythmic disturbances of the cardiac function in those who are indulging too freely in tobacco (and much the same in coffee), which suggest that the pneumogastric nerve is most affected, and thus control of cardiac and vasomotor innervation is practically lost. And when Huchard himself speaks of the cardiac phenomena arising from the abuse of tobacco as "accélération ou ralentissement du pouls, intermittences et arythmie du cœur, lipothymies et syncopes, angoisse et anxiété précordiale, palpitations, battements tumultueux, instabilité extrême des functions circulatoires, état particulier méritant la désignation 'cœur irritable' des fumeurs" (p. 178), we cannot doubt that loss of pneumogastric control is the chief neurosis present. Under this loss of pneumogastric control it is easy for slight causes to bring about vasomotor anginal seizures. Huchard speaks of three forms of tobacco angina. One is purely functional, in which the heart is sound, but the coronary arteries are spasmodically narrowed, producing temporary anaemia of the heart-muscle (l'angine spasmo-tabacique); secondly, there is organic narrowing of the vessels from arterial sclerosis due to chronic tobacco poisoning, which he calls "l'angine scléro-tabacique"—a more grave condition to be considered in our next category; thirdly, the arterial spasm is attendant upon dyspeptic causes of tobacco origin, "l'angine gastrique tabacique." These would also come under the head of functional or vasomotor angina. Huchard (p. 191) quotes one fatal case in which no other reason for death could be found than a functional failure of the heart from excess of tobacco; and two other cases in which both patients, aged respectively 38 and 50, died of tobacco angina, but in which, as no necropsy was made, the absence of coronary sclerosis could not be verified. One of the most severe cases of angina pectoris I ever witnessed, some ten years ago, ending in complete recovery, was attributable to excessive cigarette-smoking.

There has been no recurrence, and the patient is now well, having entirely relinquished his habit.

Symptoms.—Angina pectoris vasomotoria, false or spurious angina as it has been called, is not in its marked degrees a very common affection; it merges on the one side into the attacks of mere perturbed heart's action so common in young people, and on the other gravitates into those cases of the second and much more serious category of angina in which vasomotor phenomena still play a very important part. The subjects of this form of angina are liable to disordered peripheral circulation; their extremities are habitually chilly; they get "dead" hands or fingers, local sweatings, and they suffer much from cold feet at night. Not infrequently they suffer also from migraine attacks or sometimes from asthma. The attack is apt to occur under the influence of some emotion; or it may be induced by walking or riding against cold winds; or, again, it may occur at night, and is then often attributable to coldness of the extremities. There are, however, generally some premonitory symptoms of defective surface-circulation,—"creeps," "pins and needles," "deadness of hands,"—and these premonitory symptoms often affect particular sides or parts of the body in different people. Headache, chiefly frontal or supra-orbital, is frequently complained of about these times; and constipation is often present. The patients are commonly but not necessarily anaemic. Sometimes, in cases approaching the menopause, they are unduly plethoric. If they be under medical observation, it will be found that the pulse is habitually quick, and that, although the arterial pressure is very variable, the range of it is decidedly high. Marey's law that the high-pressure pulse is slow does not always apply in clinical medicine, the reason no doubt being that with instability of vessels more or less irritability of heart is associated.

The heart presents nothing abnormal to percussion or auscultation, except that usually the second sound is accentuated. The cardiac impulse is normal in position, and, at least in the earlier years of the malady, normal in character, except for a certain excitement of action which is commonly to be observed. The patients are usually of spare build; one finds amongst them a relatively large number of instances of mobility of the right kidney; and undue pulsation of the abdominal aorta is also another occasionally associated symptom.

The attack begins quite suddenly, with acute pain and a sense of distension or oppression in the region of the heart. Severe palpitation ensues, accompanied by more or less dyspnoea and sense of air-hunger. Sometimes the heart appears to stop altogether, and the patient may fall down in a semi-faint with a cry of pain; this is rare, but it was a marked symptom in a young married lady seen in consultation with the late Dr. Sansom and Dr. F. J. Smith (*vide* p. 167). If the pulse be observed at this stage it will be found small, perhaps very irregular or intermitting, and generally quick. The attack may last a few seconds or a few minutes only, during which time the surface is pale and cold, and the countenance pinched; there may be clammy sweats, and the patient has

a sense of extreme illness ; the patient pants more or less convulsively, and tends to toss about or move to the window for air. This restlessness and tendency to seek fresh air by personal effort is very different from the still attitude of the subject of the graver angina. It is particularly noticeable in cases in which the severity of attack is not sufficient actually to stop the patient, who in such cases will often continue in exercise throughout an attack ; a feature never observed in grave angina, and therefore of some value in diagnosis in cases otherwise doubtful. After a few seconds or minutes the pulse becomes relaxed, if not already rendered so by treatment. A copious flow of pale urine is a usual sequel in cases of angina presenting the vaso-constrictive element. The attack passes by and leaves the patient fatigued and alarmed, and there is a decided tendency to a serial recurrence of the attacks during the ensuing few hours or days, when they will cease for perhaps a considerable interval. The degrees of severity of symptoms are, as already said, very variable, and the diagnosis is, as a rule, not difficult.

I will now relate three cases to give a more clinical character to the above statement of symptoms :—

CASE 1.—A lady of forty-five, of active habits and neurotic temperament, complained that for the past four or five years she had been conscious of her heart's action ; that it had been occasionally disturbed, palpitating, and irregular, sometimes intermitting. She had had rheumatic fever fifteen years before, and was subject to occasional rheumatic pains. Her mother, without previous illness, had died almost suddenly of heart-failure. As long as ten years ago she would occasionally, when in the midst of a sharp run out hunting, experience a severe pain at the heart, lasting for two or three minutes, and interfering with breathing. She would not stop for this pain, although it was severe, and it would pass away while still riding fast. She now complained of having similar attacks of pain ; sometimes as sharp as before, at other times more dull and of longer duration. Occasionally she felt very giddy and ill, but has never actually fainted. She suffered at times from numbness of the arms and a great sense of chilliness, requiring extra clothing even during sharp exercise. Sometimes quick walking would bring on an attack of the heart pain, but not always. This lady presented no symptoms of approaching menopause, such as flushings or perspiration, or irregularity of the catamenia. Her digestion and nutrition were good ; her pulse was quick, small, and of high tension, that is, when stopped by the strong pressure of one finger, the vessel beyond was still filled and pulsated from the peripheral side. The heart's apex was only just within the nipple line, the impulse was rather increased in force, the first sound muffled, but unattended with murmur ; its action intermitted about twice in each minute. Urine, sp. gr. 1020, did not contain either albumin, excess of phosphates, or sugar.

It will be observed in this case that some noticeable cardiac signs were present, indicative of slight dilatation and hypertrophy. These conditions, however, were not due to the anginal seizures, or in any way responsible for them, but were attributable rather to the chronic high arterial pressure of which the seizures were incidental accessions.

The following is a fair example of several cases of vasomotor anginal attacks which I have observed, always, in my experience, in women in advanced life :—

CASE 2.—A widow lady, aged seventy-two; seen with Dr. Dickinson, of Sloane Street; has had four children. Father died suddenly of bursting of an abdominal aneurysm ; mother of diabetes ; one brother of dropsy, probably renal ; one sister of consumption ; one sister of asthma, and a sister living has diabetes. The patient has passed uric acid calculi, and has had two attacks of bronchopneumonia. She is a thin, bright-eyed woman, of vivacious manners and nervous temperament. For twelve years she has suffered from attacks of pain at the heart, which are brought on by physical exertion, by passing into a colder atmosphere, or by the excitement of seeing visitors, and frequently on beginning a meal. This pain is situated behind the sternum, and spreads upward to the throat and jaw, under the tongue and ears, thence to the shoulders and down the arms ; it is also felt to some extent in the back, but leaves the cranial vertex free, and never goes below the waist. The bowels are habitually confined, but are kept regulated by cascara. The urine contains neither albumin nor sugar ; the pulse is hard, tense, and has been observed to become more so during the attacks ; rate 76. The heart-sounds are normal, the action somewhat forcible. Since the beginning of 1894, the attacks, under trinitrin treatment, have to some extent become less frequent ; but lately they have been replaced by attacks of palpitation, during which the pulse is soft and the heart's action very irregular ; these attacks are remedied by ether and strophanthus, and have taken place since the trinitrin was left off, and are, therefore, not immediately caused by that drug. I may add that this lady has no sign of disease of the heart, but on several occasions on which I have seen her, usually soon after an attack, the pulse has always been thready and incompressible ; on one occasion, when a mild attack came on during my visit, it perceptibly hardened.

CASE 3.—Mrs. F., aged fifty-two, a married lady with five children, one of whom is epileptic, had recently suffered shock from the sudden death of a son under operation when she first consulted me in December 1906, on account of having during the past six weeks suffered from pain of a cramp-like and paroxysmal character in the right sternal region. She had previously had an attack of " neuritis " in the left arm, and suffered occasional neuralgic pains in that arm and shoulder. Mrs. F. had been long subject to mild asthma. The attacks of late years would come on quite suddenly and unexpectedly, and were occasionally attended with palpitations and lasted for four or five hours. Her complaint was that with the oppressed breathing, not attended with much wheezing, she sometimes felt severe clutching pain in the centre of the chest and down the arm ; this pain may be quite independent of the asthma, or may come on towards the end of an attack. It lasted for an hour or less. The catamenia had been irregular of late, occurring at intervals of from two to eight months, and she had had the usual flushings and perspirations ; she stated that the menopausal flushing gave relief both to the cardiac pain and also to the asthmatic dyspnoea, if either should be present at the time.

The pulse was thready. There was a slight whiff in the aortic region, but the cardiac dimensions were normal. Trinitrin relieved both the asthma and

the cardiac attacks, and she has improved in the past two years under its influence with small doses of iodide of sodium. Valerianate of zinc with ext. valerian., small doses of ext. of nux vomica and pulv. rhei are a combination which has also proved valuable. This case is a curious and instructive combination of the neuroses of asthma and vasomotor disturbances, and will, it is to be expected, eventuate in recovery with the completion of the menopausal adjustment.

The following case is one which, whilst presenting decided symptoms of the form of angina now under consideration, is a better illustration of the so-called spurious angina, which cannot be separated from this variety :—

CASE 4.—Mrs. X., aged thirty-five, a lady of nervous temperament and inheritance, and married to an extremely nervous man, was seen in consultation with the late Dr. Sansom and Dr. F. J. Smith in March 1895. In the course of seven years she had had eight children, and during her eighth pregnancy suffered from dyspepsia, vomiting, and tachycardia, the heart's action reaching 135. In September 1894, after some extra fatigue on a blackberrying excursion, she was suddenly seized with great breathlessness and severe palpitation, attended with swelling of the veins of the neck, causing her to feel as though her head would burst, and pain over the cardiac region, with a feeling of great precordial (epigastric ?) distension. These symptoms were attended with such utter prostration of strength that she was obliged to lie down or she would have fallen, although there was no loss of consciousness. The symptoms subsided in two or three minutes, but pain and tenderness remained in the cardiac region. A few days later Dr. Smith found the heart normal in position, the first sound peculiarly sharp and short, and the rate very variable from minute to minute, being especially more rapid during forced expiration, and slowed by forced inspiration. More or less similar attacks were of frequent occurrence, caused chiefly by fatigue or excitement. The patient described an attack which occurred in the Christmas week of 1894, after some extra exertion in entertaining her children, as beginning in the limbs " with the circulation going the wrong way " ; then a sense of the heart " turning over," and a feeling as though she were dying. To ordinary observers this seemed no exaggeration, and it was only by close examination that the absence of any cause of death was ascertained. During one exacerbation of the attack, witnessed by Dr. Smith, there was decided gaseous distension of the stomach. In the intervals of attack, sleeplessness, constipation, and coldness of the extremities were her most frequent complaints. Shortly before our consultation she had had a very severe attack, seizing her quite suddenly and causing her to fall down. On very careful examination no cardiac abnormality of any kind could be found. The pulse was rather quick, the pressure somewhat raised, varying in this respect even under examination. She was a tall, slender woman, with the neurotic characteristics already mentioned, and a somewhat patchy flush upon the cheeks. She greatly improved in health, and in July 1896 went to Switzerland and walked the hills as well as her companions, provided she did not hurry. The attacks of heart pain continued from time to time, but they were never severe ; movement of the left arm was apt to bring them on. Dr. Smith kindly sends me a note under date August 1908 : " Mrs. X. has

remained well ever since the attack, and enjoys excellent health, except for the worry of her household."

Diagnosis.—The diagnosis of these cases presents no great difficulty, and it is scarcely necessary to say how important a correct diagnosis is in view both of prognosis and treatment.

(i.) We have to deal with a patient of a neurotic temperament and physiognomy; often, not always, at a period of life too young for the more serious kinds of angina. Although very ill for the time, the aspect to an experienced observer is not that of a fatal seizure.

(ii.) On sufficient recovery, or in an interval between the attacks, no cardiac lesion is to be found. There may be some modification of the rhythm or some accentuation of the second sound, but the position, dimensions, and sounds of the heart are within the limits of health, modified only by functional disturbance. This somewhat difficult decision must be established by most careful palpation, percussion, and auscultation in the intervals between the attacks.

(iii.) The ambulatory tendency in the attack is a decided point in favour of its vaso-constrictive origin.

(iv.) It will usually be found, in persons subject to these attacks, that the arterial blood-pressure, as detected by the character of the pulse or instrumentally estimated, ranges high. It is, moreover, variable, and may vary during the time of examination. Other phenomena, indicative of instability of the vasomotor nervous system, may be observed.

Prognosis.—The prognosis of angina vasomotoria is, as a rule, very favourable. Under varied moral, hygienic, and medicinal treatment, and with lapse of time, the younger patients get well. Climacteric cases (Case 3), and a certain proportion of gouty cases also, end in recovery. A certain number of patients, however, lapse into the second category of angina; or if they lose their heart attacks, they still advance to the gradual establishment of cardiac hypertrophy and, usually, mitral incompetence; these phenomena being attendant upon that chronic high arterial pressure in the earlier stages of which the anginal paroxysms have intervened as incidental neuroses (*vide* Case 1). Lastly, in a very few cases death occurs in an attack. Some such cases have been recorded by Huchard, and amongst them must be included some of the cases of death in the water from cramp. In these latter, however, it is probable that the attack only proves fatal indirectly, the patient, rendered helpless by his cardiac seizure, being drowned.

Treatment.—The treatment of angina pectoris vasomotoria is prophylactic and medicinal. The mode of life of the patient must be considered, and there will usually be some defect to be remedied. Over-excitement, moral errors, dissipation, excesses in tobacco-smoking and alcohol are to be inquired for and corrected. Irregularity in meals, quick eating, and sitting down to meals when exhausted, are, through the dyspeptic troubles entailed, indirect exciting causes of the attack. Many persons towards middle life become overwhelmed with engage-

ments and worries, which make their lives quite unnecessarily hurried, and anxious beyond their nervous powers of endurance ; with a firm hand these extra causes of nerve-strain must be pruned down, and, after a sufficient rest and holiday to recover from them, the patient may with advantage return to regular work. It is certainly our experience that it is not the routine labour of the day, but the multifarious occupations and engagements of our so-called leisure which, especially with the class of patients under consideration, exhaust the nervous energies and bring about neurotic disturbances ; and it is often the worst treatment possible for such patients to cut them off from their business or professional work. Their labours may be, however, curtailed somewhat in two directions : first, to secure a moderate amount of quiet open-air exercise daily ; and, secondly, to ensure the return home in time to get an hour's quiet before dinner. When the heart's action is habitually quick, this hour should be spent lying down. On the other hand, the after-dinner hour should never be spent in sleeping, but rather in some quiet occupation, such as billiards. The dietary of the patient must be carefully inquired into, especially in those about the climacteric period or who present gouty phenomena, or venous plethora. In the latter class of cases a course of waters at Buxton, Harrogate, Carlsbad, Homburg, or Nauheim will often be desirable to start a cure, which subsequent care in exercise and diet will maintain. Cases of venous plethora with disposition to superfluous adipose tissue about the cardiac vessels, and defective cardiac tone, are those to which Nauheim exercises are especially if not solely applicable.

Finally, after a careful diagnosis, the definite assurance to these people that their distressing and painful symptoms are not dependent upon cardiac disease, and are not of a dangerous nature, will do much to relieve the attacks by withholding that element of panic which tends to intensify them. A grave admonition upon the folly and futility of worrying will often have a good and lasting effect.

The medicinal treatment is of considerable importance. It aims at lowering any excess of arterial blood-pressure, which is not corrected by the hygienic and dietetic measures spoken of ; a careful regulation of the bowels is of the first importance, and should include a small dose of mercury once or twice a week, followed by a saline. In highly neurotic persons, especially those about the menopause, a little hydrobromic acid and bromide of sodium may be prescribed for a time. Iron may sometimes be given in anaemic cases, but it is rarely well borne, and must be given in small doses. Arsenic is a valuable remedy, and may be usefully combined with valerian ; for instance, in a varnished pill containing one tenth of a grain of arseniate of iron with two grains of extract of valerian after food three times daily. Neither quinine nor strychnine is, as a rule, well borne by these patients—hop, calumba, or chiretta are better tonics ; but in the neuralgic bouts, to which they are subject, quinine and phenacetin may be usefully combined.

During the attacks we have to guard against the tendency to fly to

stimulants and sedatives. Brandy or subcutaneous injection of morphine will give relief, but they leave the patient on a lower plane and less resistant to fresh attacks than before. Perhaps very hot water, slowly sipped, is the best domestic remedy ; but such patients should have at hand a draught of soda, ammonia, or ammoniated valerian, cardamoms, and chloric ether, to be slowly sipped on the oncoming of an attack. A minim or two of one per cent trinitrin solution may often be added to the draught with advantage, or a trinitrin lozenge taken at the same time. A rest in bed of twenty-four hours is usually necessary ; and sometimes in anaemic or neurasthenic cases a more prolonged complete rest is desirable ; but it is important as soon as may be to get the patient back to the ordinary routine of life with the restrictions above laid down.

I venture to introduce here a table which I drew up for a clinical lecture some years ago, giving an instructive comparison between the main features and tendencies of vasomotor angina and spasmodic asthma.

COMPARISON OF ASTHMA WITH ANGINA PECTORIS VASOMOTORIA SIMPLEX

Asthma.	Obstructed respiratory function from spasmodic contraction of bronchioles.	Angina.	Obstructed cardiac function from spasmodic contraction of arterioles.
1. Excitation of spasm	Peripheral, visceral, central (haemic).	1. Excitation of spasm	Peripheral, visceral, central (haemic).
2. Special pulmonary lesion	None in early stages.	2. Special cardio-vascular lesion	None in the early stages.
3. Chief factor	Bronchial hyperaesthesia, a neurosis.	3. Chief factor	Cardiovascular hyperaesthesia.
4. Consequent lesion	Emphysema and its consequences.	4. Consequent lesion	Atheromatous thickening, dilatation.
5. Attendant lesions	Many and various, none of them essential.	5. Attendant lesions	Any variety of heart disease, none essential.
6. Prognosis	Depends upon attendant or consequent lesions present, *i.e.* upon integrity of motor mechanism.	6. Prognosis	Depends upon integrity of the heart, *i.e.* soundness of motor mechanism.
7. Family history	Includes some one or more of various neuroses, asthma, epilepsy, hysteria, diabetes, insanity, chorea, Graves' disease, etc.	7. Family history	Includes similar neuroses.

Within the scope of each malady certain cases are included which require special explanation and nomenclature.

II. ANGINA PECTORIS GRAVIOR

A. SECONDARY CARDIAC ANGINA.—**Etiology**.—The etiology of this group of cases of angina pectoris gravior is, in most respects, precisely the same as that of the preceding form of angina, in so far that it

includes all the causes of vasomotor disturbance which, by constricting the arterioles, brings about cardiac embarrassment. In addition to this purely nervous mechanism, however, we have further to take into account those diseases and degenerations of the heart which constitute the real pathology of secondary cardiac angina, and place it, in contrast with the preceding group, amongst the most grave of cardiac disturbances.

Sex.—Whereas the vasomotor anginas were found to be more common amongst women, the graver forms of the present category are vastly more prevalent amongst men, according to Forbes and Walshe, as ten to one. It is difficult to estimate the proportion of men to women affected. The older physicians (whose statistics are represented by Forbes and Walshe) would only reckon those cases in which cardiac degeneration, mostly of coronary origin, was present. Their cases were, moreover, completed cases which had ended fatally. Huchard, dealing with 277 cases of cardiac angina terminating fatally, finds that about four-fifths were men. These cases include the older ones and others added to them. The cases I have observed and would place in this category, namely of vasomotor angina associated with diseased heart, would give approximately 2 males to 1 female, whilst taking all cases of angina in which the heart is distinctly diseased, the numbers in my cases would be 57 males to 13 females, or about 4 to 1.

Taking, for convenience, the three classes of angina together, the following are the conclusions that appear just with regard to sex. Huchard records 141 cases of " pseudo-angine," of which 43, or 30·5 per cent, were men and 98, or 69·5 per cent, women. He also records 277 cases of fatal coronary angina (as already related), of which 224, or 80·8 per cent, were men and 53, or 19·1 per cent, women. Prof. Osler records 40 cases of his own in which there was only 1 woman, or 97·5 per cent men, and 2·5 per cent women. Among 96 cases of my own of all kinds seen during the years 1898-1908, there were (1) 26 cases of angina pectoris vasomotoria simplex, of which 6, or 23 per cent, were males and 20, or 76·9 per cent, females. (2) 36 cases of angina pectoris vasomotoria gravior, of which 25, or 69·4 per cent, were males and 11, or 30·5 per cent, females. (3) 34 cases of primary cardiac angina, of which 32, or 94·1 per cent, were males and 2, or 5·9 per cent, females. Taking (2) and (3) together there were 70 cases, of which 57, or 81·4 per cent, were males and 13, or 18·5 per cent, females.

The reason for the increased prevalence of the graver forms amongst men—much less in my experience than has been supposed—is clear when we observe that all the causes tending to earlier degeneration of the vascular system, namely, physical toil, gout, syphilis, alcoholism, and so forth, are more prevalent amongst them ; and that the causes of increased arterial blood-pressure—for instance, mental strain and gout—are more frequent and abiding in them as they approach middle and late middle life.

Age.—Except in cases in which the angina is attendant upon valvular heart disease, when its appearance may be in comparatively early life,

secondary angina occurs within the years of commencing and advancing degeneration, more particularly between forty and seventy; my cases give fifty-seven as the mean age for men, and fifty as the mean age for women. Dreschfeld stated that the coronary arteries are often athero- matous when other arteries shew as yet no sign of disease; and this he attributed to the high pressure of the circulation in them. He related one case in a boy of twelve, who died suddenly from this affection. Beyond sixty-five the field of liability to the disease has become somewhat exhausted, and the conditions of life become less favourable to its manifestation.

TABLE shewing age-distribution of Angina Pectoris (males and females) in percentages of total cases.

Age.	A.P.V.-M. Simplex.		A.P.V.-M. Gravior.	Primary Cardiac Angina.	
	Douglas Powell.	Huchard.	Douglas Powell.	Douglas Powell.	Huchard.
Under 20	0	14·1	0	0	1·08
Between 20 and 30	[1] 19·1	36·1	2·7	0	1·8
Between 30 and 40	[1] 34·6	22·6	8·3	0	15·8
Between 40 and 50	[1] 23	14·2	5·5	14·7	[2] 31·4
Between 50 and 60	7·6	12	[3] 44·4	[3] 41·1	21·3
Between 60 and 70	[4] 15·3	0	[3] 36·1	[3] 23·5	16·2
Between 70 and 80	0	0	2·7	20·5	9·7
Between 80 and 85	0	0	0	0	2·1
Total cases	26	141	36	34	277

Heredity.—Family predisposition is very decided, and this is explained by the fact that the disposition to cardiac and vascular degeneration, and to those diseases, such as rheumatism and gout, which lead up to them, are all very hereditary.

Previous Diseases.—Gout, syphilis, plumbism, alcoholism, and perhaps tobacco, may all be said to be important factors in the pathological history of the lesions associated with angina; also valvular disease of

[1] Majority of cases occur between ages of twenty and fifty.
[2] Cases with vasomotor element associated with valvular disease probably account for earlier percentages both of my statistics and those of Huchard.
[3] Majority of cases under both these headings occur between fifty and seventy.
[4] The percentage at more advanced age is probably accidentally high.

rheumatic origin. The uric acid diathesis, alcohol, and tobacco also favour a chronic or intermittent high arterial blood-pressure; besides favouring arterial degeneration, they cause disturbances of innervation which, in earlier life, tend to bring about the first variety of angina, and later to excite attacks in connexion with degenerated conditions of heart.

Syphilis is not only a cause of local or general arteritis, but may lead to the formation of gummas or gummatous infiltration of the heart. Dreschfeld has found that congenital syphilis attacks the aorta and coronary arteries more often than is supposed; causing endarteritis, which may lead to contraction of the lumen.

Glycosuria.—Diabetes has not been shewn to have any etiological effect in angina. Cases are met with in association with glycosuria, but are dependent rather on the degenerative changes consequent upon the gouty condition with which this state of urine is connected.

Uraemia.—In chronic uraemia, especially when due to contracted kidneys, anginal phenomena are very frequently manifested,—a fact which is not surprising when we recollect the persistent high blood-pressure which is a feature of this disease.

Physical Exertion.—The most common proximate cause of the graver forms of angina is physical exertion, although some organic arterial or cardiac change will almost invariably be found behind it. The ordinary history of the first attack of angina is that it began during some effort, a quick walk up an incline, hurrying to catch a train, mounting a ladder, running a race, or after a fatiguing walk. The patient may often have exerted himself to the same or a greater extent before, but the time had come when the reserve power of the heart, weakened by insidious disease, had been overstepped.

Pathology.—The forms of heart disease which constitute the serious factor of this second group of angina are several. First, there is fatty infiltration of the heart, independent of coronary degeneration. Secondly, there are those forms which depend upon permanent constriction of the coronary arteries. I have elsewhere described coronary atheroma and the forms of heart disease secondary to it; namely, fatty degeneration, false or fibroid hypertrophy, fibro-fatty change, fatty infiltration of the heart, infarction of the heart, syphilitic arteritis extending to the cardiac substance. Thirdly, there are certain valvular diseases of the heart, especially aortic regurgitation, aortic stenosis, and mitral stenosis. Fourthly, diseases of the aorta,—atheroma and aneurysm.

Of these the three most commonly met with are simple fatty infiltration of heart, fibroid or fibro-fatty hypertrophy, and aortic regurgitant valvular disease, often associated with dilatation of the vessel. Perhaps aneurysm ought also to be included.

(i.) The least grave form is simple fatty infiltration of the heart in which the fibres of the heart are not primarily diseased, but are merely more or less toneless and atrophied, being hampered in action by the intercalation of adipose tissue deposit, the result of imperfect metabolism (see p. 113). The patients are usually of sedentary habits and disposed

to good living; they are generally gouty, and uric acid or its allies are the most common proximate causes of the vasomotor spasm that starts the cardiac attack. The mechanism is precisely the same as that considered under the preceding heading. The coronary vessels of the heart may or may not partake in the more general arterial spasm, but they are not diseased. In short, these cases have the same etiology as the preceding, with an embarrassed heart in the background; the organ is more or less enlarged and its cavities dilated.

(ii.) The much more grave cases, in which the heart is enlarged and its walls thickened by fibroid or fibro-fatty changes, are almost all secondary to narrowing of the coronary vessels. The texture of the organ is impaired, its circulation is impeded, and cramp and paralysis of the muscle are apt to ensue upon any sudden accession of blood-pressure from vasomotor spasm causing dangerous or fatal angina. Thickening of the valves or aorta from chronic endarteritis is often associated with the textural changes in the heart-muscle. These cases merge in their pathology with those of the next group, of primary cardiac angina.

(iii.) Anginal seizures in association with aortic regurgitant disease are rather common. The excessive strain to which the small vessels are subjected with each beat of the heart following upon very complete relaxation in this disease may be a cause of vasomotor spasm, as it is a frequent cause of capillary haemorrhage. The valvular disease may not be associated with coronary narrowing or degenerative disease of the heart-muscle; but the more marked the dilated hypertrophy of the ventricle, and the less, therefore, the reserve power of the heart, the more readily induced and the more dangerous in character do the attacks become.

Owing to the great reserve capacity of the left auricle and pulmonary veins, mitral regurgitation is incompatible with the mechanism of the vasomotor variety of angina pectoris now under consideration; and the establishment of mitral incompetence by progressive endarteritic changes and mechanical dilatation of the left ventricle, which may be observed to occur in the course of some cases of coronary angina, is accompanied by cessation of the anginal attack.

Excessive endocardial pressure as a cause of angina is, however, not necessarily due to contraction of the vessels of the periphery. In cases of aortic and mitral stenosis, and in some other heart diseases, the ventricle is subjected to habitual strain in maintaining the circulation; and any extra exertion may overstep the limits of reserve power and bring about anginal symptoms. An excellent example of the angina now under consideration was related by Anstie in the case of a boy who, while running, suddenly cried out with severe pain in his chest, and died. On post-mortem examination he was found to have great narrowing of the aortic orifice.

(iv.) Sir Clifford Allbutt regards the anginal phenomena in a considerable number of cases as attributable to a painful distension of the first part of the aorta, which is the seat of an inflammatory lesion. The aortitis may be of rheumatic, influenzal, or other infective origin, or it

may be atheromatous. In the cases of infective source the general blood-pressure is normal, or may be depressed. In those of atheromatous nature there is generally a more or less extensive arteriosclerosis, and the blood-pressure is heightened. In either case there may be no pain or discomfort during repose, but with exertion arterial pressure is raised, and with distension of the aorta substernal pain is experienced, which, if the exertion be continued or increased, becomes more intense, radiating along the usual lines of reflection, and may by its severity oblige the patient frequently to rest in his walk, or to stop altogether. Josué holds similar views, and questions the importance of diseased coronaries in the pathology of angina. He regards the morbid changes in the coronary arteries as associated with the aortitis, to which, under condition of distension, he attributes the pain. He points out the richness of nerve-supply from the cardiac plexuses of this portion of the vessel.

In atheromatous disease of the aorta with dilatation, and especially with aneurysm, anginal seizures are not uncommon. In most cases aortic regurgitation is well marked and will account for the attacks ; in some we have to seek for the explanation in disturbances of cardiac innervation through pressure upon or stretching of the cardiac plexuses.

B. PRIMARY CARDIAC ANGINA.—*Syncope Anginosa.*—In this group of cases the symptoms are solely dependent upon causes in the heart itself. To them the apt term of Parry, "syncope anginosa," should be restricted. Their essential pathology is cardiac cramp and paralysis, due to narrowing or occlusion of one or both coronary vessels.

In an additional chapter to his most recent work Huchard has given a careful précis of 185 cases of fatal coronary disease ; in 20 there was disease of the coronaries without mention of contraction ; in 83, disease of both coronaries ; in 49, of the left coronary ; in 18, of the right coronary ; and in 15 the coronary affected was not specified. In the 165 of these cases in which there was obliteration or stenosis, it was due to atheroma or thrombosis in 158 cases, to embolus in 5 cases, and to compression in 2 cases.

It is thus evident in how large a proportion of cases of fatal angina coronary arterial disease is present in a marked degree ; and that there is much to justify the opinion of some pathologists, from the time of Jenner to our own day, who maintain that coronary disease is the essential pathology of angina pectoris.

I have endeavoured to controvert this opinion by shewing that the more important symptoms of angina pectoris are frequently met with without any coronary or other cardiac lesion (vasomotor angina pectoris) ; secondly, that in many cases of grave cardiac disablement, even including a considerable but indeterminate proportion of the cases of coronary disease above enumerated, the characteristic features of the anginal attack are dependent on the same vasomotor causes remote from the heart ; that is, upon paroxysmal constriction of the peripheral vessels

telling back upon the enfeebled heart, which latter, however, is concerned in the often fatal issue. Lastly, these cases having been eliminated, there remains a residuum of cases belonging to the present category in which the heart, and the heart alone, is the primary as well as the final factor in the anginal seizures.

It will be obvious that cases thus separable for more precise pathological consideration must overlap and merge into one another, as I have endeavoured to make clear in the diagram on page 160, and that clinically it may sometimes be difficult to classify a given case precisely. This matters nothing if our pathological insight into the disease for prognosis and treatment be assisted by the recognition of the three types.

The heart of primary cardiac angina is enlarged, but it is often less so than in the cases of the preceding category; its texture is more or less damaged by fatty or fibroid degeneration, or, it may be, by both combined. The coronary vessels, one or both, are narrowed or occluded by disease : they may be obstructed at their aortic origins by the degenera tive endarteritis about them ; or they may be converted into thickened calcified tubes ; or again, at some portion of them thick calcareous plates may almost close their calibre. Superadded to this condition thrombosis of the vessel may take place at any point. In some cases one of the vessels may be closed by an embolus ; or a syphilitic growth may thicken its walls to complete occlusion. Patches of extreme anaemia, haemorrhagic infarction, or softening affect the territory of muscle corresponding to the completely or almost completely occluded vessels.

The mechanism of the angina in these cases is tolerably simple, and precisely analogous to anginal seizures in muscles of less vital importance with which we have now for some time been familiar. Restricted supply of arterial blood has long been recognised as a cause of cramp in skeletal muscles, and affects by choice those muscles which are most in use ; thus, old people, whose arteries are degenerating and whose muscular blood-supply is wanting in elasticity of accommodation, not infrequently complain that, after walking a short distance, they are pulled up by more or less severe cramps in the legs or thigh muscles. Sometimes after a tiring day they will wake up at night with cramp pains in the limbs. In other cases, instead of cramp their muscles fail in power, and they can walk no further. Similarly in the history of anginal seizures, we are frequently told that the patient's first seizure occurred while walking perhaps faster or more laboriously than usual ; that the attack caused him to stop short, and that only after a time could he with great care and caution get home. Subsequently a less effort will bring on a similar attack, and sometimes the attack will come on at night after a fatiguing day. In such cases the factors are probably the same as in those of cramp in the leg muscles, namely, restricted irrigation of the heart through contraction of its arterial supply, impaired removal of waste products, cramp or paralysis ; but the muscle involved is in the centre of life, and its temporary disorder is apt to have a fatal issue. In 1809 Allan Burns distinctly described the effect of constriction of vessels from disease in causing failure of

power, with or without pain, in muscles during action. Burns compares a heart with narrowed coronary vessels to a limb round which a ligature has been applied with a moderate tightness. Moderate and equable muscular action can in both cases be carried on without distress, but under any pressure of labour, fatigue and exhaustion set in. In 1831 Boullay described loss of power in the limbs of the horse attended by painful cramps, associated with partial or complete occlusion of vessels supplying the parts ; the blood-supply being only sufficient for the muscles during rest or slight exertion, but insufficient to secure the nutritive changes adapted to increased muscular exertion ; to this condition he applied the term " intermittent claudication." Sir Benjamin Brodie in 1846 noted this incapacity of rigid or thickened arteries to secure that fluctuating blood-supply to muscles which is necessary to repair the varying waste during rest and severe or slight exercise, as met with not only in the muscles of the limbs, but in other muscles also ; and he, in this connexion, alludes to the observations of Jenner and Parry, which were supposed to prove that angina pectoris is due to ossification of the coronary arteries. " In examining the bodies of persons who died from the disease (angina pectoris) in question," Brodie says, " I have sometimes found ossification of the coronary arteries to so great an extent that they were converted into complete bony tubes, while there was no disease of consequence elsewhere. When the coronary arteries are in this condition, they may be capable of admitting a moderate supply of blood to the muscular structure of the heart, and as long as the patient makes no unusual exertion the circulation goes on well enough. When, however, the heart is excited to increased action, whether it be during a fit of passion, or in running or walking upstairs, or lifting weights, then the ossified arteries being incapable of expanding so as to let in the additional quantity of blood which under these circumstances is required, its action stops and syncope ensues, and I say that this exactly corresponds to the sense of weakness and want of muscular power which exist in persons who have the arteries of the legs obstructed or ossified." In 1858 Charcot, apparently unaware of the preceding observations, described this condition of vessels as a cause of pain in the limbs in the human subject, and as a premonitory sign indicative of a tendency to senile gangrene.

Dr. Parkes Weber relates a remarkable case—one of a series—that he has observed of spastic contraction of the minute cutaneous blood-vessels of the foot preceding muscular cramp-like pains in the muscles of the instep or the calf of the leg. No pulsation could be felt in the dorsalis pedis or posterior tibial artery on the affected side, and Dr. Weber regarded the patient, a Polish Jew aged fifty-two, as the subject of arteritis obliterans. This combination of vasomotor irritability in the territory of the vessels affected with arteritis obliterans causing cramp and paralysis of muscles would be analogous to the mechanism of some forms of cardiac angina. The spasm of the small vessels is probably contributory rather than essential to the production of muscular cramps (angina cruris), the impeded circulation through the main artery of the limb (as

through the coronary vessels of the heart) being the real cause. Dr. Weber associates excessive smoking with the obliterative arteritis which he had seen in 9 cases in male Russian Jews between the ages of thirty-two and fifty (*vide* p. 559).

Whilst Brodie mentions intermittent pains as amongst the symptoms of this partially obstructed circulation, he does not speak of it as an essential or constant symptom; and there can be no doubt that, as pointed out by Dr. Parkes Weber, the cramp pains and loss of power are both attributable to accumulation of waste products in the muscles; they can both be induced in healthy persons by over-muscular fatigue, but they are more easily produced in muscles whose vascular supply is inadequate, and whose tissue renewal is therefore sluggish; and, finally, in angina pectoris from diseased coronary arteries the heart-failure may or may not be attended with painful cramp. The key to the whole mechanism is supplied by Dr. Gaskell, who points out (*a*) that the lymph-fluid of all tissues in a condition of inactivity is alkaline; (*b*) that the natural activity of a muscular organ is invariably accompanied by dilatation of its blood-vessels; and (*c*) he argues that, nervous influences apart, this dilatation of blood-vessels is brought about by the diminished alkalinity of the lymph-fluid of the tissue during its activity which relaxes their muscular coats. Under conditions of diseased, rigid, and narrowed vessels this arrangement for flushing tissues with blood while in action is obviously interfered with.

In the case of syncopal attacks to which the name of "angina sine dolore" has been given by Gairdner, the phenomena are those of paralysis without cramp; some of these cases are, however, no doubt cases of extreme fatty degeneration; on the other hand, the syncope anginosa of Parry is heart-failure with cramp; whilst there may be many intermediate attacks of more or less severe cardiac cramp without actual syncope.

It is certain that the same morbid condition of the coronary vessels of the heart which constitutes the pathology of primary cardiac angina may extinguish life, not with a paroxysm of acute anginal suffering, but with a prolonged intermission terminating in fatal syncope. These syncopal attacks may be attended with some anginal phenomena, and especially with that sense of failure and impending dissolution alluded to in the letter from Heberden's "unknown" patient as occasionally taking the place of his more ordinary anginal symptoms, and described by Gairdner as characteristic of a group which he would separate under the term "angina sine dolore." It is to be remarked, however, that these attacks of painless angina are not infrequently the final phenomena in the fatal issue of angina pectoris; that is to say, a man may have had several attacks of severe angina, and finally die with a painless syncope; such attacks are not to be distinguished from the attacks of prolonged and often fatal intermissions of the heart's action observed in cases of fatty degeneration. I would rather regard them, therefore, as detached symptoms of the heart-failure with which angina, in common with some

other heart affections, may terminate, than as constituting a separate group. It will probably be found, on further investigation of the pathology of these syncopal cases, that there is occlusion of the branch of the right coronary vessel in whose territory is contained the area of the septum auriculorum, whence the rhythmic contractile impulses of the heart are supposed to originate (*vide* p. 14).

We have now well-nigh traversed the field of anginal pathology, and yet it may be complained that little has been said of neuralgia or neuritis as a cause of the symptoms. Without venturing to deny the possible occurrence of neuralgia, whether central or originating in the grey matter of the cord or cardiac ganglia, or the possibility of a neuritis of the cardiac plexus or nerves as causes of angina, I am convinced that they are of the rarest occurrence in connexion with this group of symptoms. I have seen instances of neuritis, and perhaps of neuralgia of the cardiac nerves, in diphtheria, influenza, pericarditis, and so on, but in such cases the symptoms are not those of angina. There is no question that morbid changes can and do arise in the nerves and plexuses connected with the heart, and that perversion of the cardiac rhythm and pain connected with the heart's action may arise therefrom ; but the question is how far it can be shewn that any appreciable number of cases of angina pectoris have in this cause an exclusive mechanism. With regard to Nothnagel's views of the vasomotor origin of angina I am in full accord ; but here the neurosis is a general or central one, in which the heart is only involved by a reflected mechanism, so to speak—although in some cases of tobacco angina the coronary vessels may themselves partake in the spasm and so embarrass cardiac circulation. Anstie, Eulenburg, and others maintain that angina is a neuralgia or a neuritis affecting the cardiac nerves, exclusively or essentially. Eulenburg, excluding all organic changes as conditions sometimes associated, but not essential, describes four varieties of cardiac neurosis as causes of angina, namely—

(i.) Disease, injury, or irritation of the automatic nerve apparatus of the heart.

(ii.) Some such affection of the regulating apparatus of the heart.

(iii.) Similar influences affecting the sympathetic apparatus.

(iv.) Disturbances of the vasomotor nervous system.

The late Dr. Anstie, by ingenious reasoning and by comparison with other neuroses, maintained that angina is a pure neurosis, interchangeable in families and individuals with asthma and gastralgia, and due to an irritation of the vagus nerve, or to a primary affection of the spinal centres in the upper dorsal region rapidly involving the vagus through the cardiac plexus. There are, however, but few observations of a conclusive kind in favour of angina being a cardiac neurosis.

In almost all the cases of fatal angina pectoris in which nerve lesions have been found, other conditions have been present, which may be regarded as having played at least a more important part in the fatal issue. Thus, in Heine's case, quoted by Eulenburg, in which a post-

mortem was made by Rokitansky in 1841, the right phrenic and left
vagus were found to present pigmented nodular changes, the patient
died of syncope rather than of angina, and probably at the present time the
pathology would have been traced to the bundle of His.   Lancereaux, in
1864, found those cardiac ganglia which were adjacent to thickened and
contracted coronaries to be granular.   Putjakin found changes in the
cardiac ganglia in angina ; but he found similar changes in other cases of
heart disease also, and quotes others who had found them in such cases.
Haddon, in 1870, found the left phrenic nerve in a case of angina
entangled in an enlarged gland and its elements changed ; but the aorta
was atheromatous and dilated.   Peter, in 1883, found the cardiac
plexuses undergoing inflammatory changes and disorganised ; but old
pericarditis and a dilated and thickened aorta were also present.   Ray-
mond and Barth, in 1891, found similar changes in subjects who had never
suffered from angina.

In influenza, towards the close of the illness, cardiac seizures of a
decided anginal kind are not very uncommon ; but these seizures, in my
experience, have been of the nature of vasomotor angina and associated
with an enormous discharge of (previously retained) urea—enough to
produce a dense precipitate of crystals on the addition of nitric acid.
Fatal attacks of multiple neuritis in influenza, and attacks of general
paralysis resembling diphtheritic paralysis, have also been met with, but
not associated with angina pectoris (in my experience), as, in multiple
neuritis, might well have been expected.

In the face of such facts, positive as regards the heart, negative or
subordinate as regards the nerves, it seems to us futile to contend, with
Eulenburg, Anstie, and others, that angina is a neuralgia of the heart in
any other sense than that it is a painful affection of that organ.   It is
true that in the course of pericarditis anginoid attacks are sometimes,
though rarely, met with, their gravity depending upon that of the
cardiac lesion.

The precise nature of the pains in angina has given rise to consider-
able discussion and is not yet perhaps fully understood.   Setting aside
pure neuralgia of the cardiac plexuses or of central origin as a possible
cause of occasional attacks of anginal suffering, the origin of the pain is,
as we have seen, most commonly either a stretching or compression of the
peripheral nerves of the cardiac muscle or endocardium, or a cramp
affecting a limited area of the cardiac muscle.

The intensity of the irritation may be only sufficient to affect the grey
matter of the cardiac ganglia or (to use Dr. Allen Sturge's expression) to
cause a commotion in it : in which case the pain remains localised in the
heart, and consists of a more or less dull and distensile suffering or oppres-
sion.   In other cases the intensity of irritation or the nervous suscepti-
bility of the patient may be so great that the painful impression passes
beyond these narrow bounds and extends to the cord itself, to be reflected
down corresponding nerves.

It has been shewn by Dr. Head, partly from clinical observation and

partly from a consideration of the work of Allen Sturge, Gaskell, Ross, Dean and Bradford, and Edgeworth on the cardiac innervation, that the sensory nerves of the heart are in relation with the spinal cord from the first to the eighth dorsal roots : namely, auricle, with fifth to eighth dorsal ; ventricle, second to fifth dorsal ; ascending aorta, first to third dorsal, and third and fourth cervical. In the early tubal form of the heart the auricles are placed below (posterior to) the ventricle, and their nerve-supply is therefore lower in the cord. The nerve-roots most central to the paths of pain from the heart, which as a rule receive the first and most intense impressions, are the second dorsal roots—although the painful commotion may extend or overflow to higher or lower centres.

The painful impressions upon the root-centres are referred to the corresponding surface areas of nerve-distribution, and taking the left ventricle as the most common primary seat of pain, and the second dorsal as the chief recipient of the disturbance when intense enough to pass beyond the cervical cardiac ganglia, we can account for the most common reflected surface pains. These are well illustrated in a record given to Ross, by an intelligent patient, of the reflected pains suffered during an anginal attack :—" The pain is described as starting at a point a little below mid-sternum, then shooting between the shoulders at the level of the second dorsal vertebra, and darting down the inside of the left arm to the elbow ; at the same time a feeling of great tightness was experienced over the second ribs below the clavicles on each side, and a degree of pain was similarly felt down the right arm." This distribution corresponds closely with the sensory distribution of the second dorsal nerve and its intercosto-humeral branch. In other cases the reflected pains extended to more or less of the whole area of sensory supply from the brachial plexus. It is observed by Head that during an attack of angina a notable degree of tenderness is felt over certain areas—the precordial, dorsal, and supra-orbital. This tenderness is neatly explained by Allen Sturge, who remarks that ordinary tactile sensations transmitted to irritated sensory roots become painful impressions ; and that in angina the sensory roots of the dorsal nerves corresponding with the left ventricle are undoubtedly irritated. The supra-orbital pains referred to by Head as constant in angina and in some other heart pains are most difficult to interpret precisely, as are similar pains well known in association with other visceral disturbances. My attention has not been attracted by these head pains in angina ; and although of much pathological interest, they are naturally obscured by more urgent symptoms.

Symptoms.—(*a*) *Secondary Cardiac Angina ;* (*b*) *Primary Cardiac Angina.*—The symptoms of the two varieties of angina pectoris gravior have so much in common that it will save repetition if we include them in one description, and afterwards endeavour to discriminate between them in diagnosis and prognosis, and for the purposes of treatment, with the help of a few illustrative cases.

The patient, most commonly a man, and usually between forty and sixty-

five years of age, is quite suddenly seized whilst under excitement, or engaged in some exertion not unusual or excessive to him, or even whilst in bed after a somewhat fatiguing day, with severe pain in the precordial region. The character of the pain varies. It may be most acute and agonising, of a rending character; or accompanied by a sense of constriction as though the heart were gripped or the thorax were severely pressed. Its onset is always sudden, but the pain is sometimes rapidly ingravescent rather than reaching its height at once. Having its principal seat within the precordial region, usually at the lower mid-sternum, the pain radiates in most cases upwards to the left shoulder and down the arm to the elbow or wrist; sometimes similarly to the right shoulder or to the chin and throat, but rarely in a downward direction. This radiation is not essential, and varies with the intensity and seat of the pain. The countenance becomes pale and assumes an anxious, panic-stricken expression, sometimes betraying acute suffering, and that apprehension of death which is more or less a feature of the attack. A cold sweat bedews the brow and the coloration of the lips is livid. Whatever he may be doing, the subject of true angina stops short and rests—sitting, stooping, or leaning forward against any support that may be at hand. The breathing is first disturbed, oppressed, and restrained by the pain, then panting or sighing; there is a sense of air-hunger, and the patient will motion attendants aside, although himself he dare not stir; fanning is grateful to him. The pulse may be but little changed, yet it is sometimes tightened. It may be small, hard, thready and irregular. As a rule, it is not markedly quickened, sometimes decidedly infrequent; but in these latter cases on listening to the heart it will often be found beating twice to each radial pulsation. During the attack the heart-sounds are, as a rule, distant, feeble, and of purely valvular character, the first sound resembling the second (fetal characters); adventitious sounds, such as murmurs, may or may not be present, but they have no necessary relation to the anginal paroxysm. The intensity of the attack may last only a few minutes and rapidly subside; but it sometimes returns in a series of wave-like recurrences through a period of an hour or more. There is often flatulent distension of the stomach, eructation of flatus giving some relief; but such distension is an attendant phenomenon arising during the attack, and has no necessary causative relationship to it. Much exhaustion ensues upon an attack, and a sense of having received a severe shock, from which, however, recovery takes place with varying rapidity; some patients resume their usual business the next day, some even continue the business in which they were at the time engaged. It is rare for a patient to faint with true angina, except in cases of fatal syncope.

It often happens that a patient having experienced a severe attack feels afterwards, on taking exercise, that at a certain point the premonitory symptoms of cardiac oppression and pain are felt; these become more severe as he proceeds on his walk, and oblige him to stop before all the completed phenomena of radiating pains and the rest are manifested.

Such being a description of the clinical features that may accompany an ordinary attack of angina, let us glance at the brief records of a few well-marked cases as they come before us for diagnosis and treatment.

CASE 5.—J. P., aged twenty-seven, a draper, had suffered from rheumatic fever at the ages of nine and fourteen, and several times since. He had been several times laid up with heart trouble, pain, and dyspnoea. He had never had dropsical symptoms. He was admitted into Middlesex Hospital on 5th October 1896 on account of paroxysmal pain chiefly referred to the lower precordial and upper epigastric regions. He presented no marked dyspnoea and no oedema. He was a fair-haired man of spare build, with visibly pulsating carotids and a regular, full, collapsing pulse of 100 beats per minute. The heart's apex-beat was in the 6th space, one inch outside the nipple-line, was heaving in character and diffused over an extended area, the dulness extended upwards to the third rib, and laterally to an inch and a half beyond the right border of the sternum. A to-and-fro murmur was audible over the base of the heart, the diastolic being the more prolonged and conducted down the sternum in the usual manner.

The patient had frequent attacks of pain, sometimes three or four a day, of which paroxysms the following description is characteristic :—On November 5th while in his usual condition, being examined by myself in the presence of some students, an attack of pain came on, attended with quickened action of heart and diminution in size and increase in tightness of the pulse—the diminution in size proceeding almost to extinction, a mere tightened thread being felt under the finger. While the patient leaned forwards on the bed, forcibly pressing his chest against a chair placed in front of him, the action of the heart remained so powerful as to give a visible impulse to the chair, and the neck vessels were observed to throb strongly. Two amyl capsules were inhaled, with the result of immediate relief to the pain and restoration of the pulse to its full volume. The attacks were all similar in character, and were relieved by one or two minim doses of nitroglycerin solution. He somewhat improved generally, was able to get about the ward, and finally left the hospital at the end of November.

This was an excellent example of aortic regurgitant heart disease with vasomotor angina ; and it is most instructive to note the powerfully labouring heart, the strongly throbbing large vessels in contrast with the contracted and almost pulseless smaller vessels ; and again the rapidly restored equilibrium of circulation under a remedy which relaxed the arterial spasm. The imminent peril of such a case could not be doubted, nor could the propriety of the term "angina" be questioned by any one who witnessed the paroxysm. I have recently witnessed a succession of paroxysms of a precisely similar character in a patient with aortic regurgitant disease, but in a more advanced stage. And such occurrences are very frequent, although one may not be at hand at the moment actually to witness them. The case which led Sir Lauder Brunton to suggest nitrite of amyl as a treatment for angina was doubtless of this character.

CASE 6.—A gentleman, aged about fifty-four, engaged in anxious and pressing business, had been for some years the subject of gouty glycosuria ; he

had complained for the past five years that occasionally while walking over London Bridge he would be seized with sudden pain at the heart, causing him to stop instantly, and either stand still or lean against some support. After a few minutes he would get on slowly and carefully, being stopped once or twice on his way. He had had several of these attacks, and an ether and ammonia draught containing 1 minim of nitroglycerin gave him relief. His heart was distinctly and considerably enlarged, but there was no murmur present, until two years later, when a mitral murmur appeared ; since that time he has had no similar attack. I may say that this gentleman's mother died suddenly in a railway carriage, and his sister dropped dead of heart disease. My belief is that his mitral incompetence served as a safety-valve against excessive endocardial pressure, and so has kept him free from attack. A few years later this gentleman suddenly died from a syncopal attack.

This case presented the ordinary characters of a fibro-fatty heart secondary to impaired coronary circulation, in which a heightened blood-pressure occasioned by exercise in association with arterial spasm gave rise to the attack. Dilatation of the vessels releases the intraventricular pressure, and when, in the course of the malady, the mitral valve became incompetent, the reflux into the capacious pulmonary venous system spoiled the mechanism of anginal suffering and the patient finally died of syncope.

CASE 7.—A man, fifty-eight years of age, a bootmaker, was admitted into hospital with signs of heart-failure and a history of attacks of cardiac pain. There was a history of rheumatic fever in early life and of two attacks of rheumatic gout ; also a doubtful history of syphilis. He had been in fair health up to three years before admission, when he began to suffer from sudden attacks of pain in the region of the heart, extending to the shoulder and down the left arm. The pain recurred at intervals of a few weeks, was generally attended with profuse sweating, and left the patient in a state of great prostration. Except at these times he did not feel ill, and he kept at his work until a very severe attack occurred three months before his admission ; from this time he had suffered from cough, shortness of breath, and dyspnoea on lying down, and had been incapacitated for work. During the last month he had observed his legs becoming dropsical.

He was a grey-haired, largely built, spare man with livid coloration, oedematous trunk and extremities, and orthopnoea. The pulse was 120 per minute, weak, irregular, soft, and compressible, and physical examination revealed an increased area of cardiac dulness, especially to the right and downwards, and a weak and diffused impulse with muffled sounds, the first sound at the apex being prolonged without distinct murmur. The lungs were moderately emphysematous, with oedema at the bases, and there were signs of slight effusion into the right pleura. The liver was increased in size, the urine scanty, 1030, acid, and contained a trace of albumin. The case as it now presented itself was one of enlarged, dilated, failing heart, with secondary congestive oedema of the tissues and organs in a man with a history of illness beginning with anginal seizures. On the eighth day after admission, at 4 A.M., the house physician was called to him, and found him pale, distressed-looking, and streaming with perspiration, suffering from a sudden seizure of great pain in the precordial region, and

spreading as before to the shoulders and down the left arm, with short, laboured breathing ; the pulse was very small, scarcely perceptible, but very hard and thready. It became full and soft under nitrite of amyl, but the symptoms did not immediately abate, and he recovered but slowly in the course of a few hours. On the two following days he had similar attacks, the first slight, the second severe, and on the third day he woke at 4 A.M. as if in pain, groaned, and was dead in a few minutes.

On examination the heart was found greatly enlarged, the enlargement being principally on the left side ; the auriculo-ventricular orifices were dilated, the ventricles thickened, their texture firm. The valves were practically sound ; there were some atheromatous points in the aorta. The right coronary artery was notably contracted, slightly atheromatous, and at a little distance from its orifice occluded by fibrinous clot. The left artery, fully patent at its commencement, was considerably thickened, and at a point rather less than an inch inwards was closed by organised clot. This latter was the thrombosis of older date, although the clot in the right coronary did not appear to be of quite recent formation. The texture of the heart shewed fibro-fatty degeneration.

Here again was a case, also of a mixed character, in which the heart itself was more actively and primarily involved, but was yet associated in the anginal attacks with some degree of arterial spasm during three years. The signs of heart-failure rapidly developed after the severe attack three months before admission, during which the thrombosis of the left coronary doubtless occurred. Death finally took place soon after the more complete occlusion of the right coronary.

CASE 8.—A military man, aged fifty-five, quite recently home from India, who had never previously complained of any heart symptoms, and had dined with familiar friends in apparent health and good spirits the evening before, was walking quietly with his wife when he was taken with pains in his chest, and was brought in a cab a short distance to my house. Observing him to be very ill, my servant shewed him at once into a side room, where I found him sitting on a chair with his hand pressed to the cardiac region, with pale pinched features beaded with perspiration, cold extremities, and small, thready, and irregular pulse ; a half-suppressed groan and the expression of his countenance indicated the severity of his sufferings. After a restorative and a tablet or two of nitroglycerin, he was gently moved to a sofa in another room and was able to lie down. The pulse became more regular and less thready—it never had the complete characters of vasomotor spasm. I gave a little morphine subcutaneously, and left him, returning, however, every few minutes. Thirty-five minutes from the time of his entering my house I found that he had just vomited slightly. His pulse had become regular and of better force, and he expressed himself as for the first time feeling decidedly easier. He had hardly finished speaking, however, when with an exclamation of pain he partially raised himself with his hands clasped to his breast, and fell over on his left side livid, insensible, pulseless, and with breathing arrested in expiration. By slapping with cold water, and other means, some deep, convulsive respiratory movements with loud groaning expirations were excited, but the pulse never returned. The jugular vein became slowly distended and could not be emptied by pressure towards the heart.

In all the painful circumstances I could not but assist the friends in avoiding a necropsy, and the exact lesion remains obscure ; there was an appreciable general cardio-vascular degeneration, and most probably the case was one with early atheromatous changes encircling the aortic orifices of one or both of the coronaries. But I cannot doubt that some sudden thrombosis of diseased coronaries started the cardiac spasm, and the notable manner in which the jugular became filled, resisting pressure onwards, suggested cardiac paralysis, beginning at the right heart, as the final event.

This is an example of an attack without previous symptoms of primary cardiac angina, fatal in the first instance. I could not tell the history of this case ; there was none.

Case 9.—A gentleman, aged fifty-seven, of commercial pursuits with many public duties, sent to me November 30, 1907, on account of some severe attacks of cardiac oppression which he had recently experienced. He was a largely-built man of liberal habits and leading a strenuous public life, was also accustomed in his leisure to vigorous exercises. He had enjoyed good health, and an attack of ptomaine poisoning in July 1906, which was attended with discharge from the bowels of a considerable quantity of blood, was his first serious illness. At the end of the following December and on another occasion a fortnight later he woke up at 4 A.M. with great dyspnoea and oppression of breathing. On examination I found his lungs emphysematous with some bronchial catarrh and slight oedema-rales at the bases. Heart large, the apex-beat outside the nipple-line, overlapped by lung. Cardiac impulse and sounds also too manifest at the ensiform cartilage. A systolic murmur was audible at the apex. No oedema of legs. Urine free from albumin. The case was regarded as one of fibroid degenerated heart-texture with probable stiffening of the papillary muscles, and atheromatous coronaries. The attacks of cardiac asthma were anginal. Needful restrictions as regards his diet, work, and the character of his exercises were laid down, and a carminative with trinitrin prescribed for his attacks, for which he had also had some oxygen inhalations ordered by his medical attendant. He appeared to improve much for a time, but died suddenly from heart-failure six weeks later, in the fifth attack. In none of the attacks was there any pain, simply great difficulty in inspiration and profuse sweating. The final attack lasted ten minutes ; it occurred at night a fortnight after the preceding attack.

Case 10.—A gentleman, aged fifty-three, was engaged in mercantile business, but also leading an active public life, and, in addition, much engaged with church work on Sundays. His father had died quite suddenly after a quiet walk about his farm, when talking to a labourer, and without any previous illness. His mother was alive at eighty, and two brothers and a sister were alive and well. On the 14th of September he was at his office, and on his way to the lavatory was suddenly taken with severe mid-sternal pain, and leaning against a desk, slid down to the floor in momentary unconsciousness. He got up and the pain rapidly passed. He describes its character as if having swallowed very hot fluid, which stuck half-way down the gullet. He felt very ill, as if he might die. There were no eructations, sickness, nor disturbance of bowels. The heart's action was irregular and the dimensions of the heart extended. The blood-pressure was low, 90 to 100. He rested two or three

days, and then went to Sidmouth for Nauheim treatment. The patient for a
week or two previously had felt a similar but much less severe pain on walking
soon after taking food. The pain on these occasions did not radiate down the
arm. On his return from Sidmouth there was not much improvement, and he
took further treatment at Harrogate. The blood-pressure rose to 120, and a
systolic murmur was now first heard at the apex. He still suffered occasional
attacks (two or three days a week) of milder pain on walking after food, which
would cause him to stop and, when better, to walk more slowly. On examina-
tion, January 19, 1908, the pulse was 88 with occasionally an intermission.
The vessel was somewhat thickened, tension moderate. Heart's superficial
dulness commenced at fourth rib, $\frac{1}{4}$ inch to the right of margin of sternum,
and the apex-beat was $\frac{1}{2}$ inch outside the nipple-line and somewhat diffuse.
A short systolic murmur was heard outside the apex, and a separate short
systolic murmur at the level of the third rib over the mid-sternum. Urine
reported not to contain albumin. There was perhaps a slight enlargement of
the liver. The knee reflexes were active. This case had been regarded as
dyspeptic, but unquestionably his medical attendant was right in his view that
it was one of true primary cardiac angina : the symptoms occurring, as is so
common, on first exercise after a meal. But it will be observed that the grave
attack had no reference to food, and the evidence of cardiac changes not
dependent upon valvular lesion is also very significant.

CASE 11.—A merchant, aged sixty-seven, had some months previously
suffered from a peculiar attack of giddiness, the ground appearing to move up
to his eyes, when he sank powerless into a chair, and remained there a quarter
of an hour, with no loss of consciousness. A few weeks later he was suddenly
seized with intense darting pain in the region of the heart which made him call
out, and was accompanied by a sensation of " tumble over " of the organ. He
had frequently suffered from attacks of giddiness accompanied by slight pain in
the left fronto-parietal region, which lasted for a little time beyond the giddiness.
He had only once or twice, however, suffered from the more severe vertigo since
that first described, and on three occasions only had he experienced the acute
pain at the heart.

On examination a systolic mitral *bruit* was heard, and a slight systolic
aortic murmur also. The action of the heart was quiet. Some degenerative
changes were noted in the vessels.

Two years later this patient died suddenly within a few hours of a severe
attack.

Diagnosis.—The diagnosis of angina pectoris gravior, with a sufficiently
careful consideration of the symptoms and signs, can be made out with
accuracy in almost every case.

The age, sex, and hereditary history of the patient ; the preceding
health record with respect to such diseases as syphilis, alcoholism, gout,
rheumatism ; the effects of strain as calculated to induce premature arterial
degeneration ; the evidence of such degeneration in the radial or other
vessels,—bearing in mind the fact that in the coronary vessels such changes
sometimes arise earlier than in other vessels ; and exact estimate of the
dimensions, functions, and valve-sounds of the heart, which determine its
muscular and valve integrity or otherwise ; these are the points upon which

diagnosis depends. Any evidence that the heart has yielded before the blood-pressure, or that it has become hypertrophied yet with signs of failing circulatory power as registered at the pulse, or that the mitral or aortic valves have become thickened or incompetent, furnishes us, in the presence of attacks of painful heart-failure, with proofs that such attacks are of the graver form of angina

In discriminating cardiac pain from that of hepatic or renal colic, of flatulent colic in the stomach, and of rheumatic neuralgia of the chest-walls, it may be briefly observed—

(*a*) That renal colic is a pain often of sudden appearance, of considerable duration, situated in the flank on one side, reflected downwards towards the groin or testicle, and accompanied or followed by characteristic changes in the urine. There are no heart-phenomena.

(*b*) Hepatic colic is also often of very sudden and agonising onset, the pain being situated at the epigastrium or right hypochondrium, reflected across the abdomen and through to the right scapula, sometimes to the right shoulder-tip. The associated phenomena are nausea and vomiting, sometimes jaundice. The cardiac phenomena are nil, or only such as are attributable to acuteness of suffering. I have met with one case in which hepatic colic was complicated with an attack of fatal angina, which it apparently excited ; and I am occasionally seeing another case in which distinct hepatic colic occurs, attended with the passing of biliary grit, and at other times cardiac pains of an anginal kind accompanied by faintness and oppression.

(*c*) Flatulent colic of the stomach may be associated with angina and in some cases may excite it. Otherwise the phenomena observed are localised in the stomach, and consist of flatulent distension with eructations of wind ; the pains are epigastric, hypochondriac (left), and interscapular.

(*d*) Neuralgic rheumatism of the chest-walls presents features rather suggestive of pleurisy ; the pain is unilateral, increased on breathing, and the dyspnoea arises directly from the pain unaccompanied by restlessness and gasping symptoms of air-hunger.

(*e*) The passage of a clot through the heart into a branch of the pulmonary artery is attended with symptoms at the moment indistinguishable from angina. The paroxysm is, however, almost immediately followed by cough and the expectoration of dark, more or less clotted blood, significant of pulmonary embolism.

In our endeavour to distinguish between vasomotor and primary cardiac forms of angina pectoris gravior—a diagnosis of some importance, both in regard to prognosis and treatment, and often presenting very considerable difficulty—we have to keep in view the following points :—

1. The presence of a high and especially of a variable degree of blood-pressure in the intervals between the attacks, and of a tightened radial vessel during the attack, would be decided evidence in favour of vasomotor spasm as an important symptomatic factor in the case.

2. The presence of an aortic regurgitant valve lesion or of aneurysm, or the recognition of a weak and large heart from fatty infiltration, as distinguished from fatty degeneration of the cardiac muscle, would be strong presumptive evidence of the angina being of the secondary cardiac form (Case 5).

3. The recognition of dyspepsia, of constipation, of present gouty phenomena, or of mental emotion as factors in the causation of the attacks in any given case, would also favour the diagnosis of its vasomotor incidence.

On the other hand—

4. The absence of the vasomotor phenomena referred to under headings 1, 2, 3 would suggest primary cardiac angina ; but it must be remembered that in the circumstances of an attack in a person of nervous temperament, some increase of arterial tension may well be present and intensify the symptoms.

5. That the earlier attacks came on during exercise not of a violent kind, and that the symptoms steadily increased during exercise, obliging the patient to stop, would suggest primary cardiac angina (Cases 6, 7).

6. The presence of a large fibroid hypertrophied heart failing before a normal blood-pressure without any murmurs being as yet present, would signify primary cardiac angina (Case 7).

7. The presence of an apex-murmur, shewing incompetence of the mitral valve in cases of the kind under consideration, indicates degenerative thickening of the mitral and probably also yielding of the ventricle before the blood-pressure : regurgitant escape of blood from the ventricle militates against the mechanism of vasomotor angina. Hence mitral murmur in association with angina is in favour of the attack being of primary cardiac origin (Case 10). An aortic systolic murmur would point in the same direction.

8. Sudden syncopal attacks with or without pain, and generally fatal, are usually of primary (coronary) cardiac source (Case 8). It is to be remarked that such attacks are frequently preceded by more recognisable anginal seizures coming on during exercise (Case 6).

Prognosis. — The prognosis of angina pectoris gravior is always serious, but varies according to the nature of the heart condition which lies behind the symptoms.

(*a*) In cases in which the heart condition is one of muscular atony with a variable degree of adipose infiltration and mechanical dilatation of the weakened ventricle, the prognosis is decidedly encouraging. Doubtless some of these cases pass in later life into group *c*.

(*b*) In cases of valvular disease of the heart with vasomotor spasm, the attacks, although much more hazardous, are not commonly fatal ; and are susceptible of great relief from treatment.

(*c*) When, still in cases of secondary cardiac angina, a recognisable element of arterial spasm is present, but with a large fibroid or fibro-fatty heart in the background, in which the coronary vessels are presumably

diseased, a sudden fatal termination may be feared ; although even in such cases the end may be averted by judicious care and treatment.

(*d*) In cases in which the symptoms are of primary cardiac origin a fatal termination within a short period is inevitable.

Treatment.—In popular and even in professional estimation heart disease is too often used in a sense of very exaggerated prognostic significance, and when the name Angina is used a fatal issue is a foregone conclusion.   Yet, as a matter of experience, with the more enlightened and rational treatment of modern times, and the use of some new remedies and the better handling of old ones, there is no class of serious diseases which are so amenable to remedial measures as those of the heart.

It may truly be said that we have enlarged the field of angina, including therein a large class of cases which were not comtemplated in the description of Heberden and Parry, and which would be excluded by some modern writers of great authority, such as Sir William Gairdner. This matter has been duly considered ; all merely functional diseases of the cardiac vascular system are excluded from the present category of angina pectoris gravior, and yet we shall find that treatment is not merely to be regarded as palliative or expectant.

Treatment is of undoubted value in angina.   Much may be done, not only to relieve symptoms, but to remedy the conditions which underlie them.   Taking first the anginal paroxysm, there are certain prominent symptoms that call urgently for relief, if relief be possible, namely :—

(i.) Pain.   (ii.) Arterial spasm if present.   (iii.) Stenocardia and cardiac muscle failure.   (iv.) Shock and air-hunger.

Pain is almost always due to one or both of two conditions, namely, distension or muscular cramp of the ventricle.   When first called to a patient in the midst of a paroxysm there is no time for careful examination even did the condition of the patient permit of it.   A tightened, thready pulse, with obviously labouring or it may be paralysed heart, urges upon us the immediate use of the vascular antispasmodics, nitrite of amyl or nitroglycerin.   Five to twenty minims of nitrite of amyl may be inhaled, or one to five minims of a 1 per cent solution of trinitrin given.   If there be violent or forcible heart-action, stimulants are better avoided ; but if there be flatulent distension, a draught of aromatic ammonia, soda, cardamoms, and chloric ether may be given with the amyl inhalation or the nitroglycerin drops ; if such means be not to hand, some very hot water with a little peppermint essence or brandy may be sipped slowly.

In cases in which there is marked heart-failure, ether by preference, or brandy, in doses of 20 drops to ℥j., in which one to two minims of trinitrin (1 per cent solution) may be dissolved, should be injected hypodermically.   When the pain is not relieved by this treatment, arterial spasm having thus been eliminated as its cause, the use of morphine subcutaneously is indicated ; due care being exercised with regard to the dose in view of the possible presence of kidney disease ; if this factor be

excluded, the degree of pain would regulate the dose, and the combination of atropine would be useful as a heart-stimulant.

The free use of oxygen inhalation is of very great value in all cases in which cardiac failure is a marked feature. The remedy has a double value in satisfying and relieving the air-hunger (due to impaired circulation through the lungs), which is often so marked a feature ; and in securing the circulation through the usually constricted coronary vessels of over - oxygenated blood, which stimulates nutritive changes in the muscle and secures the removal of effete and half-changed materials which embarrass its function. In administering the oxygen, however, all personal co-operation on the part of the patient must be avoided ; the naso-oral muzzle must never be used, but the gas must be directed over the mouth and nostrils by means of a glass funnel attached to the tubing held a few inches away so as to leave the patient to breathe a highly oxygenated air at his ease. Oxygen inhalation is particularly indicated in those cases in which morphine is found necessary ; and, when the paroxysm is over and sleep induced, the gas should be allowed from time to time to fortify the air immediately about the patient's mouth and nose. It is best to let the patient choose his position for himself, and adopt that which is most comfortable and helpful to him.

After the acute seizure is over, the whole case must be carefully investigated with a view to an accurate diagnosis ; and the treatment to be adopted must depend upon the conclusion arrived at.

It is needless to say that in all cases the causes of angina must be reviewed, and all pernicious factors in the case in hand, such as excess in diet, tobacco, alcohol, and other habits, eliminated.

The treatment of the different forms of heart disease will be found under the proper headings ; that suited to the vasomotor factor in any given case has been already touched upon. It only remains, therefore, to make one or two further observations with regard to treatment.

In the first group of cases of angina pectoris gravior, in which the heart is large, wanting in power, and embarrassed in action by fatty depositions about its surface and fibres, and is labouring against a high arterial resistance which is prone to acute increase, the line of treatment is a restricted but fairly nitrogenous dietary in three regular moderate meals ; root vegetables, sweets and starchy foods, and all sweet wines and beers being avoided. Fluid should be taken very sparingly at meal-times, and supplemented by a draught, preferably of hot water, taken between meals or shortly after food, and again either at bedtime or in the early morning, with a view to the excretion of effete materials.

Regulated open-air exercise is of the utmost importance—beginning with regulated level walks, proceeding to gentle inclines, and so on, but never overstepping the limits of cardiac power. Unquestionably Oertel's treatment for cardiac weakness and the Nauheim baths and exercises are valuable in this form of malady. It is only to be regretted that the latter treatment has been so " boomed " into popularity for every conceiv-

able form of heart disease and imaginary heart ailment as to discredit its use in appropriate cases.

The most important drug treatment of these cases consists in the judicious administration of mild mercurial laxative and saline aperients to reduce arterial blood-pressure ; iodide of potassium is also sometimes valuable to this end.   It is in this stage of the disease that Prof. Bradbury advises the use of erythrol tetranitrate, which he finds to exercise a more persistent influence upon the blood-pressure than other preparations of the kind.   In addition, strychnine and acid tonics, taken once or twice a day only, are sometimes of value.

In cases of valvular disease of the heart appropriate remedies must be employed.   When digitalis is employed it is often advantageous to combine with it small doses of nitroglycerin ($\frac{1}{200}$ to $\frac{1}{100}$ gr.), or of erythrol tetranitrate, to slacken the arterial resistance.

In cases of coronary disease and consequent secondary nutritional changes in the heart there is still much to be done : (*a*) by regulating the daily life of the patient, physically and mentally, on the capacity of his circulatory powers, and by insisting upon a leisured and level life, free from excitement, hurry, and physical exertion or fatigue, and yet occupied up to within the limits of his capacity.

(*b*) Small nutritious meals always to be preceded by a period of quietude or complete recumbent rest, and followed by an interval of quietude, but not of sleep.   A digestive and carminative medicine may be given before the chief meals.

Arsenic is the most appropriate drug for these cases, and oxygen inhalation from time to time, and especially in the night, is very valuable in those where respiration becomes shallow and inclined to the Cheyne-Stokes type during sleep.   In the latter cases strychnine is the most valuable cardiac tonic.   Small doses of digitalis or strophanthus are indicated when the cardiac power is much reduced and its rhythm irregular ; caffeine is also of great value, especially when the urinary secretion is scanty.   The liver function must be stimulated from time to time by a mercurial laxative.   Let it be said, in fine, that he who would treat angina pectoris in its multiform degrees with all the success that can be looked for, must take the cases in hand on broad lines in accordance with the well-defined principles of medicine, pursuing such lines into such detail as may be appropriate to each case.

<div align="right">R. DOUGLAS POWELL.</div>

REFERENCES

Historical and General : 1. ALLBUTT, Sir CLIFFORD.   *Brit. Med. Journ.*, 1903, ii. 22 ; 1906, i. 5.—1*a*. EULENBURG.   Ziemssen's *Cyclopaedia*, 1878, xiv. 31.—2. FORBES. *Cyclopaedia of Practical Medicine*, 1833, i. 81.—3. GAIRDNER.   Reynolds's *System of Medicine*, 1877, iv. 535.—4. GIBSON, G. A.   "Angina Pectoris," *Practitioner*, 1906, lxxvii. 289.—5. *Idem.*   "Some hitherto Undescribed Symptoms in Angina Pectoris," *Brain*, 1905, xxviii. 52.—5*a*. HEITZ.   *Arch. des mal. du Cœur*, Paris, 1908, i. 542.— 6. HUCHARD.   *Maladies du cœur*, 1899, ii. 50, 523.—6*a*. JOSUÉ.   *Arch. des mal. du Cœur*, Paris, 1908, i. 564.—7. KEITH and FLACK.   "The Form and Nature of the Muscular Connections between the Primary Divisions of the Vertebrate Heart," *Journ.*

Anat. and Physiol., London, 1907, xli. 172.—8. *Idem.* "The Auriculo-ventricular Bundle of the Human Heart," *Lancet*, 1906, ii. 359.—9. OSLER. *Lectures on Angina Pectoris*, 1897.—10. STOKES. *Diseases of Heart and Aorta*, 1854, 481. References to earlier and some current literature will be found in the above. **Experimental Observations with Reference to Angina :** 11. CHARCOT. *Progrès méd.*, 1887, sér. 2, vi. 115. —12. COHNHEIM. *Lectures on General Pathology*, New Sydenham Society, 1889, i. 35. —13. PORTER. *Journ. Physiol.*, 1894, xv. 121.—14. *Idem. Journ. Exper. Med.*, 1896, i. 46.—15. BRUNTON. *Pharmacology*, 1887, and *Lectures on the Actions of Medicines*, 1897, 321. References to earlier experimental writings will be found in the above. **Organic Nerve Lesions as causative of Angina :** 16. EULENBURG and GUTTMANN. *Physiology and Pathology of Sympathetic System of Nerves*, 1879, 97.—17. HADDON. *Edin. Med. Journ.*, 1870, xvi. 45.—18. LANCEREAUX. *Gaz. méd.*, 1864, sér. 3, xix. 432.—19. PETER. *Traité clinique et pratique des maladies du cœur*, Paris, 1883, 671. —20. PUTJAKIN. *Virchows Arch.*, 1878, lxxiv. 461.—21. RAYMOND and BARTH, quoted by Fraenkel. *Verhandl. d. Kongress für innere Medicin*, Wiesbaden, 1891, x. 228. **Neuralgia a Cause of Angina :** 22. ANSTIE. *Neuralgia and its Counterfeits*, 1871, 70.—23. EULENBURG. Ziemssen's *Cyclopaedia*, 1878, xiv. 41.—24. GASKELL. *Journ. Physiol.*, 1882, iv. 43.—25. HEAD. *Brain*, 1896, xix. 218.—26. ROSS. *Ibid.*, 1888, x. 355.—27. STURGE. *Ibid.*, 1883, v. 492. Eulenburg contains references to earlier writers, Laennec, Trousseau, Romberg, Friedreich, etc. **Vasomotor Angina —the Disease a Vasomotor Neurosis (?) :** 28. EULENBURG. Ziemssen's *Cyclopaedia*, 1878, xiv. 34 and 48.—29. GAIRDNER. Reynolds's *System of Medicine*, 1877, iv. 575. —30. NOTHNAGEL. *Deutsches Arch. f. klin. Med.*, 1867, iii. 309.—31. DOUGLAS POWELL. *Trans. Med. Soc.*, 1891, xiv. 267 ; *Lumleian Lectures*, Lect. i. 1898 ; *Brit. Med. Journ.*, 1894, ii. Eulenburg and Gairdner give the views of Cohen, Traube, Romberg, and Landois. **Intermittent Claudication a Factor in Pathology of Angina :** 32. BOULLAY. *Arch. gén. de méd.*, 1831, xxvii. 425.—33. BRODIE. *Lectures on Pathology and Surgery*, 1846, 360.—34. BURNS. *Diseases of the Heart*, 1809, 138.— 35. CHARCOT. *Gaz. méd. de Paris*, 1859, 202.—36. *Idem. Progrès méd.*, 1887, 99. —37. FRAENKEL. *Verhandl. d. Kongress f. inn. Med.*, Wiesbaden, 1891, x. 228.—38. HUCHARD. *Maladies du cœur*, 1899, 3rd edit. ii. 5.—39. OSLER. *Loc. cit.*, Lects. iv. and vi.—40. POTAIN. *Union méd.*, 1894, lvii. 181.—41. WEBER, PARKES. *Amer. Journ. Med. Sc.*, Phila., 1894, cvii. 531.—42. *Idem. Proc. Roy. Soc. Med.*, 1908, i. (Clin. Sect.) 44. **Toxic and Epidemic Angina :** 43. BEAU. *Gaz. des hôp.*, 1862, 330.— 44. GELINEAU. *Ibid.*, 1862, 454.—45. GRIFFITHS. *Compt. rend. Acad. des sc.*, 1895, cxx. 1128.—46. HUCHARD. *Maladies du cœur*, 1899, ii. 178, 186, 191.—47. WHITTAKER. *Twentieth Century Pract. of Med.*, 1896, iv. 442.

R. D. P.

OVER-STRESS OF THE HEART

By SIR CLIFFORD ALLBUTT, K.C.B., M.D., F.R.S.

THE subject of mechanical strain of the heart and great vessels, to which Peacock recalled the attention of physicians in the middle of the nineteenth century, has since that time undergone no inconsiderable changes of opinion. For my own part, although the problem is one to which I have devoted no little pains, I undertake the discussion of it with no less diffidence. And yet my opportunities have not been scanty : I have watched the conditions of the arterial system in Leeds in the laborious artisan, in Cambridge under the vehemence of youth ; and in both places I have had the advantage of consultations with colleagues whose practice brought them in closer contact with the

toil of the labourer or with the ardour of the undergraduate. To Dr. Michell of Cambridge I am deeply indebted, as will appear presently.

At the outset it is necessary to indicate a division of this subject into three parts : namely, static and permanent disease of the heart and arteries due, or attributed, to extraordinary effort; dynamic and temporary disorder of like origin; and thirdly, gradual and almost insensible spoiling of the vascular system due, really or apparently, to the wear of hard muscular labour during long terms of years, especially when the first freshness of youth is past. In all three series, moreover, the problem is complicated by various and obscure contingent influences. The first division, that of static and permanent injury, is distributed under the several heads of Cardiac Disease, chiefly under that of the aortic area (p. 418) ; and the third belongs more logically to the section on diseases of the arteries : thus it is mainly with the second division that this article must be concerned—with the field of dynamic change in persons of otherwise healthy body, who by single or persistent exertion may have urged muscular effort to degrees menacing or hurtful to the organs of the circulation, or in whom efforts of less severity have told upon a vascular system already somewhat impaired by other causes.

Now, before we can appraise any such consequences, we must endeavour to discover the limits within which the machinery of the circulation can adapt itself to those stresses which in various degrees we all have to encounter ; and in the next place to recognise the signs of any overstepping of these limits. Furthermore, as we thus step outside them, what, or how much, are we to regard as easily reparable trespasses ? what again as perilous transgressions towards or actually into the realms of disease ? It is, generally speaking, the experience of consultants to meet only with the cases in which these limits have been overstepped. It is because the patient has fallen into disease, or into grave disorder, that the more formal consultation is sought. And for these more overt conditions, however difficult it may be to unravel the causes of particular cases, we are not unprepared. What we are less prepared for, and this confession I will venture to make on behalf of physicians generally, are the cases which do not fall into the common categories of overt heart affection, but present functional disturbances of very various kinds and degrees of significance ; disturbances often indeed more stormy than those of graver lesions. But without an adequate experience of the particular behaviour of the vascular system in athletic men, and of the more superficial and transient perturbations common to all men, we cannot undertake to formulate maxims on which men are to be trained to strong exertion ; nor to indicate, negatively and positively, the rules of life which are to guide them while so engaged, and the earliest signs of slackness, in sound persons or frail, while under training or under excessive stresses whether of effort or of endurance. Yet disease must not happen, or only on very rare and sudden occasion ; there must be no ordinary probability of such a misfortune, no danger so considerable that parents and guardians should be constrained to set bounds to the enthusiasm

of their wards; or that young men themselves in their games should be beset by apprehensions and meticulous rules. We ought to be in a position to declare at once when perplexing or selfish precautions are also fantastic. Happily the margin of tolerance is so large, the play of the equilibrium of the circulation in sound persons is so wide—of the elderly we will speak later—that it is seriously questioned by learned pathologists, especially by those of the school of Leipsic, if it be possible, by any muscular effort practicable to the bodily frame, to push the healthy heart or great arteries beyond the limit of their reserve. It is alleged by these physicians that in so far as such an effect may become apparent in particular cases the parts were not sound, but had been beforehand the seat of some latent defect, mechanical or toxic. Morbific influences such as these, by their many contingencies and the obscurity of their symptoms, are as insidious as, on the other hand, they are fraught with issues of vital importance to all spirited young men, and of grave concern to those who have control of them in their callower years.

The sceptical critic, and I confess to some such bias, may plausibly maintain that hitherto physicians, by grandmotherly warnings against the free play of fiery youth, have done more harm than good; and, what is worse still, by laying hold of schoolboys and undergraduates who after games, somewhat too rash perhaps, may have complained of palpitation, anaemia, slackness of energy, and the like, and submitting them to exacting and otiose systems of treatment and restriction, in not a few cases have dressed up imposing phantoms of disease, have encumbered many precious months of education, and moreover have engendered an introspection and valetudinarianism which a more discerning management would have avoided. In the article on Functional Diseases of the Heart it is explained how manifold are the causes which, in young people especially, may set up cardiac perturbation; and how frequently these are attributable, not to any defect or hurt in this organ itself, but to its sympathy with eccentric disorders of multifarious origin, bodily and mental.

It is our first duty, then, in cases of alleged "heart-strain" to discriminate between heart-strain, fatigue, and contingent discords of other interpretation. For one case of disability due to strain, or even of sharp over-stress, there are fifty of secondary and incidental derangement. Notwithstanding, we must admit that to overlook a case of heart-strain by confusion with mere functional fretfulnesses of different meaning, might be a worse blunder than a few superfluous "cures." Difficult as discrimination may occasionally be, confusion of opinion in cases of a kind common enough in adolescence, and occurring under conditions which in respect of education, manliness, and vocation in life impose peculiar caution, must be rectified by precision of method, sagacity, and continuous revision of the data of experience.

The pioneer work of Peacock, da Costa, Traube, Seitz of Zürich, Roy and myself, and many others, has on the whole been verified; but it has certainly been pushed beyond the degrees which these observers

would have approved.   After a period of uncritical appreciations of physical signs, especially by the Nauheim school, and—for such disorders as these at any rate—overwrought therapeutical schemes, a reaction has set in which, as is usual in the course of opinion where the facts are intricate, is veering to the opposite extreme.   The researches of de la Camp and his colleagues, able and important as they are, carry us surely too far if they dictate to us that by no voluntary effort are the dimensions of the heart substantially altered.   Are not the ortho-diagraphists in their turn assuming the somewhat unreasonable dictation which in the Nauheim school we have deprecated.   In their valuable method I am personally unskilled ; but my not infrequent opportunities of seeing the work of others convince me that as yet there is almost as much uncertainty in the appreciation of magnitudes by orthodiagraphy as by percussion.   Of moving objects its pictures are fugacious, and the apparent dimensions are still subject to deceptive space-relations.   For my part, no orthodiagraphy can invalidate such an observation as this which follows ; and it is but one of many, closely observed and attested by physicians of unbiassed experience in such matters.

CASE.—Mr. T., aet. 17, well built, but tall for his years, not in training nor in perfect health, went in the summer of 1908 on a high expedition with an experienced member of the Alpine Club and a competent guide. Even before reaching the glacier level he felt breathless, but after a short rest seemed ready to proceed. An hour or two later, however, he knocked up, and was with difficulty conveyed to a mountain restaurant, where fortunately he met with Dr. Waugh of Kensington, who examined him carefully.   His pulse was then 136 to 140, but regular. After some rest a mule was obtained, and upon it he reached a lower inn, where I met him.   He was then utterly unable even to sit on the mule.   We laid him at full length on a bench in the fresh air.   The pulse was still very rapid ; he was prostrate and mentally torpid ; his face was cold and rather livid, and the limbs were cold.   The respiration was shallow and frequent.   I dare not do more than examine the front of the chest, where I was startled to find the cardiac dulness extending far beyond any debatable margin ; it passed for two thick finger-breadths beyond the right sternal line.   The veins of the neck were full and fluctuating (I had no means of course of recording their oscillations).   The soft and flat abdomen did not suggest any rise of the diaphragm.   The impulse was diffuse, but in his position I could not define the axillary borders.   Later, when he had reached his hotel and his bed, I noted an extension to the left ; and Dr. Waugh, who met me in the evening, stated that on his first examination he had made the same note. He also definitely, and quite independently, corroborated the enormous dulness to the right, an exorbitancy which, we agreed, could not be accounted for by any degrees of pulmonary retraction or diaphragmatic elevation within common experience.   The patient was observed by Dr. Waugh and myself until he was fit for removal to a stage nearer home.   From the second day the dull area to the right diminished gradually and decisively, and by the third or fourth day it had wholly disappeared, and with it most of the cardiac symptoms. The temperature, which on the first evening was about 102° F., continued to be febrile, or subfebrile, for some days after the disappearance of the heart

disorder; and about the middle third of the right lung behind was a dullish patch with some bronchial crepitation, which in our opinion must have been antecedent to the excursion. This patch did not clear up for two or three weeks, during most of which time he was kept to bed. With the disappearance of this pulmonary trouble Dr. Waugh informs me recovery was soon established; and on re-examination, at Christmas 1908, he found the recovery in all respects complete.

Now this is a very definite example of heart stress of the kind which I described in my 1870 paper, and which was verified a few years later by Roy and other workers on the subject, and often since. I have chosen it because it emphasises the question discussed by the Leipsic school, and especially by Albrecht, if in such cases of yielding the heart was at the time of stress in a perfectly normal condition? At that time our patient was suffering seriously from a "neglected cold"; but to the best of our judgment not specifically from influenza.

In a previous paragraph I have alluded to methodical and intelligent training. Happily upon this part of the problem we are all agreed; namely, that in respect of the safety of the heart training is not only of the first importance, but is all-important. It is not perhaps too confident a statement that a properly trained person, man or boy, need never fear cardiac strain. Mistaken as in many ways the old systems of training were, still by prescribing regular work and certain thumb-rules, they enabled veteran oarsmen, and other University "blues," to give a very good account of themselves, both in their sports and in later life (Morgan).

Until lately, many observers of the circulation under physical stress have been following static disease back to its causes; endeavouring thus to disentangle the element of strain from other adverse implications. Recently the more promising method has prevailed to detect in the healthy heart the effects for good and ill of stress; their significance and their relation to strain, whether for or against it. Both methods have their validity; but it is evident that we cannot begin to define the effects of disease until we have gained some knowledge of the capacities and confines of health. As man and the higher animals in the course of ages have not won any immunities from the supremacy of the circulation of the blood, and as man himself, minute by minute, owes his life to its integrity, the necessary margin of safety, which cannot therefore lie in any independence of the activity of this supreme machinery, must have been attained by great enlargements of this capacity and adaptability. As the animal mechanism attained many wide and various powers of survival, yet scarcely even a momentary independence of the heart, this organ must itself have attained an enormous endurance and resources almost illimitable.

Seeing, as I have said, that the pioneer work in the investigation of strain of the heart fell within the sphere of the healer of the sick rather than of the physiologist, the study of the subject was confined to the sphere of disease. Opportunities of watching the behaviour of the heart,

stressed indeed, but within the confines of health, did not generally fall
to the lot of the practitioner.   And I may repeat that among physicians
themselves consultants are appealed to after some mischief, by whatever
cause or causes, is perpetrated ; whereas family physicians are in more
continuous touch with their charges, and are more intimately cognisant
of all their doings.   My own experience is of the former and remoter
kind, and is therefore more exceptional and ambiguous.   Within the
last few years, however, devotion to athletics, occupation with the
chemistry of the body, the appointment of medical officers of schools,
and other such changes have enlarged our opportunities, and have
brought to us not only a great accession of physiological knowledge, but
also a more fruitful experience of current conditions ; so that we are
approaching these difficult problems with greater advantages.   The im-
mediate result of these opportunities has been, or I think ought to have
been, to open the eyes of the physician more fully to the vast range of
cardiac accommodation, and to make him hesitate in pronouncing too
readily upon cardiac strain.

A series of experiments upon the blood-pressure of persons engaged
in muscular work was projected for the years 1895-96 by the late
Professor Roy and myself, but my colleague's unhappy and ultimately
fatal illness stopped this and other investigations of the kind.   One
rather curious result, however, came out in the course of the more or less
desultory observations which we had made upon athletic men in Cambridge
and elsewhere, namely, that in them, as a rule, the habitual blood-pressure
ranges low.   Observations upon men given to arduous muscular work, but
recorded at intervals of complete or comparative rest, seemed to indicate a
rule that in them the arterial pressures range habitually under the average.
In my own person a few weeks of mountain-climbing or a cycling tour
are always followed by a lower range of blood-pressures, lasting for many
weeks.   The subjective impressions of experienced clinical observers must
be taken for what they are worth ; but if a succession of observers skilled
in the pulse agree that the radial pressures of a certain set of men seem to
them to run low, this agreement deserves consideration.   Dr. George Oliver
says : "Observations with the pulse-pressure gauge have shewn that,
when other indications are favourable, the lower ranges of pressure are
not only more salutary, but are very often compatible with the highest
health."   Dr. Michell's records will appear presently.   The well-known
rise in blood-pressure in advancing years may, in part at any rate, be
the converse of this proposition.

It was on grounds chiefly clinical, then, that I stated that muscular
exercise tends in the long-run not to raise, but even to reduce, the mean
arterial pressure of the twenty-four hours.   On the other hand, the blood-
pressures of men who, as athletic habits are laid aside, lead sedentary
lives without denying themselves at least as great an abundance of
food, are prone to rise.   By abstinence this disposition might be pre-
vented ; but I am generally assured by brain-workers that they need, or
at any rate desire, a somewhat liberal diet.   For my own part I admit

that when occupied from day to day in brain-work I crave for food far more than when engaged in vigorous exercise in the open air.

If, then, we assume a trained man to be working continuously with his muscles at a uniform but moderate rate, the total daily pressure in his arteries would probably lie somewhat under the mean of that of ordinary citizens ; and the output of twenty-four hours, if increased at all, would run on the whole at a lower rate of frictional resistance. If, on the other hand, we assume the same man to carry eight bushels of wheat up a flight of steps every ten minutes, although still the mean of his pressures and output for twenty-four hours may not be excessive, the maximum pressures, that is, the initial rise at the outset of each effort, may be very high. Again, if we take another man, one who does not carry sacks hour by hour and day by day, but is engaged as a check-weighman, and takes a sack up occasionally ; in him the maximal arterial pressure, as he shoulders the sack, will be driven much higher than under the same effort more regularly undertaken by a porter whose thoracic capacity, blood-volume, and vascular distributions are used to such recurrent stresses. And if a clerk from the office were fired occasionally, by emulation of the porters, to shoulder a sack, his defect in the automatic adaptations to such exercises might cause so sudden and relatively so great an increase of arterial pressure as to embarrass, to dilate, and even to strain his heart ; possibly indeed to rupture some part of it.

A few years ago, in the case of some athletic undergraduate suffering from cardiac disturbance, I was associated with Dr. Michell of Cambridge, and then became aware that Dr. Michell was not only making a clinical study of the subject, but also, by modern methods, such researches upon the healthy as well of course as upon the failing subject, as to contribute to this inquiry data of which we stood in great dearth. Some little time after Roy and I, and subsequently Dr. Michell and I, had been watching and gauging the healthy undergraduate in his exercises, certain German observers began to test healthy men under conditions of artificial stress, and to their results I shall presently refer. Moreover, Dr. James Kerr had most kindly carried out some tests for me upon school children ; and meanwhile the well-known researches of Zuntz, Chittenden, and others were throwing light upon the associated problems of metabolism.

As Dr. Michell's results in their kind stand almost if not quite alone, I have substituted his manuscript for certain parts of my own, as follows :—

Dr. MICHELL'S REPORT.—I propose to consider first the influence of continued hard physical exercise on the heart of the young man, and to shew that the athletic man cannot be judged and treated by the standards which are successfully employed with regard to the non-athletic man ; and then to point out the earliest signs and symptoms of overwork which will be followed, if unrecognised, by heart "strain." With very few exceptions the material on which the following report is based is derived from notes made on present or past undergraduates of the University of

Cambridge, many of whom have kindly allowed me to examine them at intervals for my own information. Of the individuals examined 1200 were rowing men, 410 football players, and a few running men. The cases on which the notes on atrio-ventricular regurgitation are founded are selected from the total number of athletic men who have fallen under my notice. Each individual was examined twice on the same day, between 8.30 and 11.30 A.M., and again in the evening, two hours after the cessation of all exercise.

The points which stand out most prominently, as the men are followed through the years of their healthy athletic lives, are :—(1) The progressive reduction in the frequency of the pulse ; (2) the progressive increase, in each succeeding year of residence, in the percentage of men who shew this reduced pulse-rate ; (3) the progressive decrease in the difference between the frequency of the morning pulse-rate, before exercise, and the evening after exercise ; (4) the gradual increase in the size of the left ventricle.

I. The average pulse-rates of the three several years in the men examined are :—First year, 69 ; second year, 64·5 ; third year, 56·8. If the school athletes be subtracted from the whole number examined the rates are :—First year, 74 ; second year, 68 ; third year, 58·3. When a picked team of men, some of whom at least have been taking part in some form of exercise for more than three years (for example, a University Eight), is examined, the average morning frequency may be 52 ; the rate in some individuals being 48 or 46. At the onset of a febrile attack in an athletic man in active work this infrequency does not disappear immediately ; indeed a rate of 70 may be an indication of severe fever, for after forty-eight hours the rate generally rises suddenly, without necessarily meaning that the patient is any worse. After recovery the heart has the rate and rhythm of the healthy non-athletic man ; and some weeks or months must elapse before any hard work can be undertaken, the length of this interval depending on the kind and severity of the illness.

II. The progressive increase in the number of men who present this slow pulse-rate is shewn in the following table :—

Percentage of Men with a Pulse-rate *below* 65 to the Minute.		
Age.	Below 11 Stone.	Above 11 Stone.
18	40 per cent	35·29 per cent
19	40·81 ,,	44·70 ,,
20	59·09 ,,	60 ,,
21	64·86 ,,	66·23 ,,
22	84·21 ,,	71·42 ,,

The first two lines are influenced by the facts of many freshmen who had already taken part in athletics, and shew relative infrequency. The

corresponding figures for 280 men who had not previously been athletes are :—Below 11 stone, 16 per cent; above 11 stone, 15 per cent.

III. The progressive *decrease of the difference* between the pulse-rates of the morning and of the evening :—

	Undergraduates *under* 11 Stone.		Undergraduates *over* 11 Stone.	
	18 Years.	19 Years.	18 Years.	19 Years.
1st year . .	11·68	10·3	12·5	10·35
2nd year . .	9·2	9·4	11·1	8·5
3rd year . .	8·37	9·0	7·9	8·2
4th year . .	...	7·7	...	7·8

The maximum blood-pressures of these men is as follows :—When just awakened and lying in bed, 95-100 mm. Hg; after rising, and while moving about the room, 100-115 ; while standing still, 105-110 ; in the middle of the day, 115-120 ; two hours after hard exercise, and immediately on lying down, 130-125 : from this highest point the fall becomes rapid, so that after the man has been lying down for ten minutes it is very often 106-110. To get an accurate early-morning pressure the observed must know the observer sufficiently well to feel neither surprise nor annoyance when (half asleep) he feels the armlet of the sphygmometer being placed into position. The blood-pressures have been taken by a modified Riva-Rocci manometer, which was pumped up until the pulse-wave disappeared, and did not return after keeping the pressure up for one minute, while the artery continued to feel empty to the finger.

The mouth temperature of these men, when they take hard exercise, rises 1° to 2·5° F. above their normal. When successfully trained, the blood shews a continuous rise in the number of red corpuscles to about 6 millions per c.mm., and a corresponding rise in the colour-index. If the weight be steadily falling, or has fallen and remained lower than is usual, these increases are succeeded by decreases. The same fall is seen when the man has overworked himself and wearied his heart ; and when he is "stale," or becoming stale ; and appears, if I may judge from the experience of 43 cases, to be the earliest sign of incipient unfitness.

The heart-sounds of the healthy athlete differ from those of the healthy man who is not athletic ; thus, the first sound may be represented by the sounds "L-lumb"; its pitch is lowered, its volume greater, and its beginning smaller than its continuation. This is true both of hearts which do, and of those which do not present reduplication. In a man lying in bed and just awaking after a good night's rest, the second sound in the aortic region is absolutely lowered in pitch, and is below the second sound in the pulmonary region. After exercise it rises to be absolutely higher in pitch, and equal to, or above, the pulmonary. Its volume is greater, both absolutely and

relatively to the pulmonary, and this difference becomes more marked as the day advances.

Indications of Overwork.—(i.) The earliest sign of overwork is a rise of the pulse-rate in the morning before exercise ; the next sign is a rise of the pulse-rate in the evening after exercise ; thus the relation hitherto existing between these two rates of beats to the minute is disturbed, and the difference between them is increased : for instance, a heart which is passing from the healthy hardworked to the overworked state may beat as follows : — (1) M. 57, E. 64 ; (2) M. 66, E. 64 ; (3) M. 68, E. 95. (ii.) The second stage occurs in men who have been taking part in athletics long enough to have developed a typical healthy athletic heart, but have then overdone themselves, generally by some one great effort.   In men whose athletic life has been shorter, the first of these stages passes directly into the third.   Of beats which make up the frequency to, say, 95, some arise from the descending limb of the pulse-tracing which precedes them.   The enhanced blood - pressure remains stationary for a longer, though a variable, time after the patient has lain down ; and the gradient of the fall is more prolonged than in health.   The sphygmometer will usually shew the higher pressures to be about 140 mm. Hg ; at times, however, the pressure is found to be below the normal.   I have seen these differences related apparently to weariness of the right and left ventricle respectively, and in hearts which are equal only to very gentle exercise.   (iii.) While this is occurring, another change in the rhythm has appeared ; the sounds have changed their time-relation to the events of the cardiac cycle.   In the place of "L-lumb-dup," followed by a lengthened pause, we get an approach to equalisation of the intervals, which becomes more evident as the overworked condition of the heart becomes more marked.   The heart has begun to "space."   (iv.) Experience has taught me that it is impossible to exaggerate the importance of the observation that a heart is "spacing"; it means that rest is imperative, and if it is not taken, other signs will appear which signify that the case is passing out of the class of wearied hearts into that of "strained" hearts.   The apex snaps at the chest-wall ; the first sound loses its "boom" and its length, and rises to a high pitch, and the daily excursion of the apex ceases.

The importance of these signs is that, when the rhythm of the strained heart under observation is compared with the rhythm of the normal healthy non-athletic heart, they remain (or can be easily made manifest) after the "spacing" has ceased to be obvious.   At this stage the patient will probably obey directions as to taking rest, but he is more likely to rest two or three days only, and then to seek a second opinion.   This rest, however, is usually sufficient to allow the heart to beat without "apparently spacing."   The patient therefore presents himself to the consultant with a pulse-rate of about 68 to the minute, the beat steady and well sustained ; he then is run about the room for two or three turns, when it will rise to about 85, still being steady and, as compared with the standard of the non-athletic man, well sustained.

The apex-beat, however, does attract attention, and is reported to be sudden : the pulse, too, does not settle until thirty seconds have passed ; but, as the sounds are clear and of good volume, the patient is merely advised to take things easily for a week or two.   He does so, as far as he knows how to do it, and then reverts to his old ways.   If he really has taken things easily he may revert successfully ; but, as a rule, his return is temporary, and he is driven to seek advice again.   Now it can be shewn that his good success or ill success depends on whether it is the right or left ventricle which is "strained."

(v.) This is the fitting place to record the existence of a point beyond the apex at which the percussion-note becomes flattened and empty as compared with that given by the chest-wall still more to the outer side.   (This is true for the formation of chest and lungs in most young men, but a round chest may not have this point.)   If this point can be determined, and indelibly marked on the skin in the morning before exercise, it can be shewn that in the evening after exercise the apex-beat and the mark nearly, or quite, coincide ; the "apex" having moved out.   After a night's rest the apex is found again within this point ; it has moved back again.   When the heart is strained the behaviour of the apex is different ; it goes out to, or near, the above-mentioned spot, but it stays there.   At whatever time of day it may be sought, the beat can be found at, or near, this outward place.   This can very often be easily proved, in spite of variations in the distension of the lungs.

The earliest signs of recovery are the return of the apex to its normal behaviour, and a fall in the blood-pressure.   The cases which go beyond the condition just described fall into two classes which are fairly well defined, though not entirely distinct.   The number of these cases is relatively small.

In the one (*a*) the patient is usually an athlete of short experience and youthful years ; the history of ailment covers but a short time, a day or two, and is somewhat as follows :—Until two days ago he felt quite well ; then he had pain over the left side of his chest, or a fluttering sensation, or a vague discomfort which appeared to be in his heart ; he wanted to vomit, or did vomit, after exercise, and was short of breath. Examination of these short - history men shews a waviness over the cardiac area, epigastric pulsation, a feeble apex-beat felt over a small area, a tender spot over the sternal ends of the second, or sometimes the third, intercostal space, a small twanging first sound, a second sound high pitched, empty, over the pulmonary cartilage, and over the aortic cartilage often sudden, short, and below the pulmonary in pitch but above it in volume.   In some cases, however, it is unchanged, and in these the condition had passed off when gentle exercise had been substituted for hard.   If the examination be made after exercise, but before rest has been taken, the precordial waviness will not be seen ; the epigastric pulsation may not be seen or, more likely, is definitely decreased ; the apex-beat is well defined, but sudden and vibrating ; and in about half

the cases there is dulness in the second right intercostal space, and a systolic murmur along the left side of the sternum from the fourth to the second interspaces. I have heard a similar murmur in patients whose vessels were sufficiently relaxed, for example after influenza, to bring about cessation of the pulse at the wrist or ankle when the limb was raised about 18 inches from the horizontal, and the murmur has disappeared when the relaxation of the vessels disappeared. A radial tracing and a cardiogram shew this sudden and vibratile apex-beat to be diastolic, and to be followed by a systolic fall. Such a train of signs and symptoms as this is at times supplemented and completed by another of great importance, namely, engorgement of the veins of the neck. This series of events is the form of strain peculiarly prone to occur in untrained but otherwise healthy men who undertake violent exercise at short notice.

(b) In the other class the history covers a longer time; the patients are older and more experienced athletes who, for some reason they believe to be adequate, for example, to obtain a "blue," have overdone themselves for a long time, perhaps for a year. The patient has not been feeling well for some ten days or more; on some days he felt better than others, especially in the early part of the week. Last night, after he had been in bed two or three minutes, he felt his heart "thump," and thought it was going to stop. Whenever he stays quiet he can feel his heart beating irregularly; or he has a feeling that there is something in the left side of his chest which is not in the right. For some time, say two or three hours after exercise, he feels better; he has a stabbing pain now and then (which he indicates with his finger in the fifth space in the neighbourhood of the apex-beat). He has lost weight quickly during the past week or so. Examination shews in this patient the cardiac dulness increased outwards on the left side; a rhythm which is irregular in frequency, owing to grouping of the beats; and an impulse which is sudden and heavy. His heart-signs are not much changed in character from those usually heard and seen in the healthy non-athletic man. His blood-pressure is about 120 mm. Hg, and begins to fall as soon as he lies down. This class is very much smaller than that first described. In the stage beyond this the condition is aggravated, and other symptoms and signs appear, as follows :—The force of the pulse becomes markedly irregular; beats are dropped as well as grouped. The dropped beats correspond to contractions of the ventricle which have not propelled sufficient blood into the aorta to make a sensible pulse-wave in the radial artery. Where the pulse-wave which has been dropped should have appeared a sphygmogram shews a small smooth rise. These abortive ventricular contractions are accompanied by one sound only, small, tone-less and distant, which in time corresponds to the rise of the small elevation. These contractions cannot be felt on the chest-wall. At times the force of the pulse is diminished by a sequence of some half a dozen rapid beats of the heart, which can be felt only as irregular tremors of the chest-wall accompanied by small high-pitched sounds.

The region of the apex-beat is so sensitive to pressure that the patient will say that he thinks he will be sick if the stethoscope be applied to the region again. While the patient is standing the blood-pressure falls rapidly to 100-90 mm. Hg, or even less, and the pulse is easily felt by the finger to be dicrotic. These patients are liable to feel faint, or even to faint during physical examination, and to do so again and again after being brought round. The horizontal position does not always prevent this.

In the stage which follows another sign is added, namely, regurgitation through (i.) the tricuspid valve, (ii.) the mitral valve. The number of these cases occurring in trained men who were previously healthy is very small indeed. By far the larger number are seen in untrained men who have attempted tasks which should have been left to trained men. Both these complications tend to disappear if treated appropriately from the first. This is especially true of tricuspid regurgitation, which not only disappears, but does so rapidly; for example, in two days. The murmur may disappear, or there may never be one.

(i.) After tricuspid regurgitation has ceased, as is shewn by the cessation of the reflux wave in the internal jugular veins, the heart steadily settles down into a condition of apparent health ; but experience of these cases shews that it is still unable to adapt itself to any marked increase of the work it has to do. On gentle exercise gradually increased, such as walking at $2\frac{1}{2}$ miles an hour, or horse riding, when the horse is quiet and no jumping or galloping is tried, it will improve ; but if it be pressed at all, it will shew signs of reverting to the impaired state from which it had apparently recovered.

(ii.) Mitral regurgitation does not shew any tendency to disappear suddenly or indeed quickly ; the condition of the heart responsible for it begins to improve in a month or so, and continues to do so steadily for about a year, when recovery may be and often has been complete.

I have purposely said nothing about dilatation which persists, because I have only seen it in men who had not only taken strenuous exercise while untrained, but had also a history of either diphtheria or rheumatic fever.

Treatment.—When the right side is affected and the affection has occurred on the day the patient is seen, or on the day before, it is always worth while to put him in bed. All these hearts have a strong tendency to revert to the rhythm they had before they had been overworked, and anything which relieves them of work increases this tendency. As the blood-pressure in these cases is high, the conditions under which the heart has to contend are improved by reducing the arterial pressure ; with this object diuretin and a hot bath, or a hot pack if the heart be "beating against the chest," should be prescribed. I have usually begun with a dose of 15 grains of diuretin, and have then been guided by the effect produced. Sometimes it acts decisively in a very short time. In one man, as the result of two such doses of diuretin given with a four hours' interval, the pressure sunk so low that he had a sighing respira-

tion, dimness of sight, and a feeling of impending syncope.   Diuretin
has the great advantage that it lowers the blood-pressure, by relaxing
the blood-vessels, not only without depressing the heart, but with dis-
tinct increase in the volume of the first sound, and the absence of any
cumulative effect.

The hot bath should be given at full length, and be as hot as the
patient can stand it ; he must be lifted out of it on a blanket, and not
allowed to get out himself, if the effect desired is to be attained.   I have
given the hot pack when the bath was either too small to allow the
patient to lie at length, or was too far away from his bed.   Both these
methods work well, especially with the help of an intelligent patient, who
can be told and understand what " all this fuss is about," as one of them
said to me.   The justification for this " fuss " is the importance of doing
away with the small hard pulse indicative of constriction of the arterial
system as soon as possible, in order to avoid a vicious circle in which the
temporarily feeble heart is further enfeebled by working against a per-
sistent and raised peripheral resistance, which reacts on the heart and
increases its enfeeblement, the final effect being a sum of these factors.

With this treatment a heart which has shewn regurgitation (I am
not here concerned with the " safety-valve " action on the right side)
will, in a short time, cease to shew it : thus, in two days the heart will
settle down, the pain will cease to radiate, as it often does, to the left
shoulder and axilla, and will disappear ; the first sound will be lowered
in pitch and raised in volume, but the murmur heard at times along the
left side of the sternum does not always pass away so soon, and when
it remains becomes more distinct ; but eventually it disappears.   This
murmur is of great prognostic value ; it indicates the cases which are
most likely to relapse if the heart be again tried by hard exercise,
although it may have appeared to be quite recovered.

When the patient leaves his bed, which he does as soon as the heart
has quieted down, he must be told to take gentle exercise only ; to be
careful not to try to do anything suddenly or strongly ; to avoid stairs,
or when this is impossible, to take care that he rests on every third or
fourth step to allow his heart to settle down ; and to avoid all excitement.

Furthermore, he must be impressed with the necessity of abstaining
from tea, coffee, cocoa, tobacco, and alcohol.

When the left side is affected the patient should remain in bed as
long as the heart's rhythm continues to return towards that of the
healthy athletic heart.   When progress ceases, he may be allowed
gradually to do more and more until he reaches the stage of moving
about deliberately on the level.   The murmur, if there be one, will not
disappear, but rather become of greater volume and lower pitch if further
rest be taken, while the first sound becomes definitely of smaller volume
and almost toneless, a mere rap, and any exertion tends to produce or
does produce a turbulent rhythm.

Whether the right or the left side be affected the patient is wise in
time, who during the earlier days, limits strictly the amount of food, solid

and fluid, to that absolutely necessary to keep down hunger and thirst. As time goes on he will find that this amount decreases ; and as long as there is evidence of regurgitation he would be especially well advised to be very careful not to exceed this amount of food ; at any rate for so long as the heart continues to improve. He should also avoid the articles of diet indicated above as harmful. It is evident that regurgitation of this type is different in kind from that which affects athletic men who have suddenly given up exercise and taken to sedentary work, too often without making any difference in their diet. In a very few cases the regurgitation persists on the left side. These are considered elsewhere.

In a few cases, when the heart's rhythm is very greatly disturbed, diuretin acts slowly if at all, and morphine guarded by strychnine in hypodermic injection is necessary to quiet the heart. In these cases the blood-pressure varies within wide limits. The relative anaemia, which appears so soon and so quickly, is beneficially affected by a mixture containing glycero-phosphate of manganese and haemoglobin.

The after-treatment of these cases is concerned with two different states which may exist together or separately. In the one the heart must be treated ; in the other the patient. Digitalis and strychnine, which are used in the treatment of ordinary heart disease, sometimes give rise to bad effects in these cases. When the pulse is small, frequent, and the blood-pressure low, digitalis is sometimes efficient and sometimes not ; it affects the hearts of these patients more easily than in others, and it is difficult to get and to maintain the desirable rhythm, because another rhythm is prone to occur, in which beats are dropped and the interval is followed by a thumping beat. Reduction of the dose does not often result in the reappearance of the desirable rhythm. The addition of strychnine to digitalis enables the desirable rhythm to be obtained with smaller doses of the latter drug, but the mixture is apt to be followed by an increase of the patient's sensibility to the beat of his heart and by a sensation of tightness in the left side and middle of his chest. These sensations make a patient restless, and the last makes him really anxious. In the condition just referred to, and in the further stage of it in which with a jerky and variable pulse and an enfeebled beat there is also a marked liability to a fall of blood-pressure, which is quick and sudden and frequently occurs without change of position, *Cereus mexicana* acts efficiently. It increases the strength of the contractions of the heart, which become regular without causing the blood-pressure to rise sufficiently to necessitate the use of the reserve strength of the heart to overcome it.

Epitome of a Typical Case of Affection of the Left Side.—A seasoned athlete, aged twenty-two years, seen in November. The chief points were : The depth and length of the first sound of the heart. The pulse-rate, 54 in the morning and before exercise, 59 in the evening and after exercise. The blood-pressure, which fell to 108 and 105 mm. Hg in the morning and evening respectively after lying down for ten minutes. An impulse, which was a sustained press,

felt 3 inches and $3\frac{1}{4}$ from the left border of the sternum in the morning and evening respectively. When seen again in June, the pulse-rate was 59 and 68 ; impulse less sustained and $3\frac{1}{4}$ inches from the edge of the sternum. The increased frequency of the pulse and the greater difference between the morning and evening rates caused me to warn him to rest. He did not do so. Seen again in November ; pulse-rate 68 and 66 to 113 in the morning and evening respectively. Impulse felt about $3\frac{3}{4}$ inches from the left edge of the sternum, both in the morning and the evening. Blood-pressure 130 in the morning ; 140 to 110, irregular, and most often at the low figure, in the evening, even when standing. The first sound was raised in pitch, shortened, and lessened in volume. The region of the apex-beat was so sensitive to pressure that he asked me not to apply the stethoscope a second time lest he should vomit.

Epitome of a Typical Case of Affection of the Right Side.—An athletic freshman aged eighteen and a half years ; when seen in November the apex-beat was in the nipple-line $3\frac{7}{8}$ inches from the left border of the sternum. Pulse-rate 68 and 78. Blood-pressure 130 mm. Hg when standing ; 118 after lying down for fifteen minutes. Seen again in May, in the evening after very strenuous exercise taken without any previous steps to get fit after a rest which had lasted a year. He complained of pain along the left side of the sternum at the level of the 3rd to the 5th costal cartilages ; consciousness of the beat of the heart and difficulty in breathing when he tried to do more than stroll along. The apex-beat was $4\frac{1}{4}$ inches from the left border of the sternum and kicking. The pulse-rate 72 and spacing ; in the morning 88 and not spacing. The blood-pressure 148, 142 after lying down fifteen minutes ; 130 next morning and apt to rise in leaps without any obvious cause. Epigastric pulsation and precordial tremor visible over the ventricle and the right auricle. The two former were not visible next morning, the last remained for thirty-six hours. The first sound was twanging and thin : in the morning lower pitch and larger volume. The second sound was a high-pitched toneless rap, particularly over the second left costal cartilage, propagated to the left mid-axillary line ; in the morning it was lowered in pitch and not propagated. The external jugular vein was prominent in the evening and ceased to be so in twenty-four hours.

R. W. MICHELL.

In order to interpret the meaning of our clinical observations, we must define the word *strain*, so freely yet so loosely used by many writers. In an earlier essay I used the title "Over-strain," which is, of course, absurd ; over-stress is an intelligible term, but all strain is over-strain. Over-stress may end in strain, or it may not ; it may produce only a transient harm, no more than a passing fatigue. But the harm of a strain is perhaps always permanent ; even if all apparent disability pass away for good, some alteration of texture probably remains, slight and partial as it may be. If slight and partial, the heart may be practically none the worse ; the lesion may ultimately consist only in a few strands of lower fibre, or some intimate molecular alterations microscopically imperceptible.

In such a structure as the heart—and the same is true, *mutatis mutandis*, of the arteries—we have at present to regard two properties

especially, namely tone and elasticity. Tone is a "vital" function; elasticity a physical quality. In his essays on tone, Dr. Gossage, and implicitly Dr. Waller before him, have suggested that this property may be transmutable with certain other properties of the heart; that in a measure tone may, as it were, absorb contractility, or contain "reserve." If so, as by "fatigue" tone is abated, other properties may be irregularly released in a more or less temporary way; and as the fatigue passes off, and tone pulls itself together again, the balance of the cardiac synergies may be restored. With strain, a more physical change, it is not so; this injury is not to be got over with a mere recovery of tone by rest and nutrition. For example, if we take a watch-spring in one hand, holding it upright, and with a finger of the other hand bend down the upper end in a bow till it meets the lower, by this compression, if the quality of the steel be good, no molecular alteration takes place in the spring; the molecules of the metal, being still within the mutual pull, do no more than revolve about each other without escaping from the orbits of mutual attraction: on release, they rapidly resume their original positions. But if the steel be defective, or the bending so acute as to exceed its elastic limits, they do not return, or they return but partially; so that the primary mutual relations of the molecules are not entirely re-established. The molecules are now dislocated, and the spring has taken a new and permanent "after-strain," or set. Strain then, the effect of stress beyond the elastic limit of a material, and perhaps of reiterated or over-riding stresses near it, results in a new set. In living tissues, however, over-stress may amount to strain or it may not; if the over-stress has been only to the degree of tone abatement, but not beyond the elastic limit of the particular fibre, it will not have taken a new permanent set; on the restoration of tone its molecules will recover their mutual space-relations. Probably tone depends directly not on the tissue itself but on a continuous—or virtually continuous—stream of energy from the nervous system, whereby a tension-curve in steady concert with other coefficients is maintained; a curve lying far within the elastic limits of the particular tissue. Mere fatigue of a muscle or other animal fibre, therefore, is not comparable with fatigue in a metal: in a metal it means molecular rupture or segregation, and a new set; in the heart or artery it means only a functional abeyance, the molecular structure being virtually intact. To the segregative or detritive alterations of advancing years another name must be given.

What then are these all-important elastic limits underlying the tone of animal fibre? In answering this question I may assume as common knowledge that the limits vary widely with the kind and age of the fibre; that in the child or youth the limits are astonishingly wide; but that with age they become narrower and narrower. In a certain suffocating baby Dr. Lloyd Jones tells me he found at first no cardiac change, whether of area or sounds. As it grew worse the cardiac area doubled, extending on both sides of the mid-sternal line, and a loud diffused systolic murmur appeared. As under chloroform the suffocating spasm subsided, and the

cyanosis passed off, the murmur ceased, and the heart soon returned within its normal boundaries.　　Moreover, in respect of the myocardium, by some substitution as yet ill understood but due to age or disease, muscular fibre may be supplanted by fibre of a lower kind, generally connective fibre, whose elasticity may be higher but is contained within much narrower limits.　　The superior and young fibre has less resistance to deformation, but it has a remarkable resilience ; it recovers even from extreme deformation quickly and entirely ; the older fibre, or fibre of lower grade, has a higher resistance to deformation, but far smaller range of resilience.　　Connective-tissue fibre therefore, when substituted for muscular fibre, as we find commonly in old hearts, gives to the part a higher resistance, but at the cost of resilience ; so that once over-stressed the molecules of this fibre never recover their former positions ; deformation is permanent, there is a new and permanent set.　　Thus in the heart there are three modes of dilatation : active adaptive dilatation, to receive larger volumes of blood (*vide* " Aortic Disease," p. 439) ; passive but remediable dilatation, by abatement of tone, acuter over-stress, or more gradual fatigue ; and the irremediable dilatation due to strain.

Hence it appears that if we find in older persons that a ventricle of the heart under some relatively excessive stress has dilated, we have but too good reason to fear that, consisting of an older or in part of a lower fibre, its narrower elastic limits may have been transgressed, and that it may never wholly return to its form, nor recover its molecular integrity. In a young person, on the other hand, in whom is no history of rheumatism or other infection, we are justified in hoping that even an enormous dilatation may prove not to have exceeded the wide elastic limits of the fresh resilient texture ; and that in a few hours, or in a day or two, as in the case of Mr. T., the molecules will recover their primary equilibrium, and the textures their primary form.　　Thus after a reasonable time the heart should be ready again to cope with the ordinary tides of functional demand.　　It is possible indeed so to stretch even a new india-rubber band as to exceed its wide elastic limits, and to strain it, but in ordinary uses we rely on its extremer adaptability ; with an old one, on the contrary, we are tender, knowing that its limits have probably become much narrower.　　In old rubber, as in old tissues and in the eternal hills, an intimate molecular degradation is in perennial and unsleeping activity.

Once more, in steel or in india-rubber we know that both elasticity and resilience may vary greatly under treatment.　　By peculiar treatment we can make steel of the high elastic resistance but narrow resiliency of a ball-bearing, the brittle molecules of which part company abruptly on slight torsion ; or we can make it supple like the spring and the india-rubber.　　In either of these materials also by various treatment we can produce corresponding differences of elastic endowment.　　So with the heart ; besides the gradual alteration of molecular constitution which in lapse of time slowly accumulates in muscle, steel, rubber or any material, the heart at all ages is susceptible to many other modifying

influences, extrinsic and intrinsic, especially to those which in the animal body are known as toxins. Thus the elastic limits of the myocardium may be so altered as to impair its capacity for the large functional variations to which, especially in young persons, it is exposed. We have reason to know that such baneful influences are only too frequent ; that overt or covert attacks of an infection, manifestly or insidiously, often so impair the cardiac muscle, and so reduce its resiliency, as to render it liable to yield prematurely or unexpectedly. It is but too clear that under such conditions over-stress and even strain may be set up in young hearts as in old, and prove in its consequences, if not permanent, at any rate very tedious. It is scarcely necessary to add, that what is thus true for young hearts is more baneful still in old ones ; but infections are less rife among elder people, less liable to pass unnoticed ; and in any case they touch the hearts of persons less occupied with severe exertion.

It is in the acuter cases of strain that this toxic factor can be indicated more precisely ; in chronic cases stress is so intimately confused with the toxic factor—such as the abuse of alcohol, microbic infections, and the like—that it is often exceedingly difficult or even impossible to distribute its due weight to each of these several causes. For example, many interesting observations concerning strain of the heart have been made upon soldiers ; yet there is perhaps no class of persons in whom the various factors of cardio-arterial disease, including improper equipment, are more difficult to estimate severally. On the other hand, however, the part of stress in the causation even of chronic cardio-arterial diseases comes out plainly when such cases are considered in numbers large enough, if not to eliminate, yet greatly to reduce the risk of error: when, for instance, we contrast large numbers of persons engaged in laborious callings with large numbers of those whose pursuits are mechanically less urgent ; when we compare forgemen, hodmen, navvies, wharfingers, Cornish miners, or Tübingen wood-cutters, who have no monopoly of vice, with clerks, professional men, or even with persons occupied in the open air but not in heavy muscular exertion. The part of stress, mixed as it still is with other factors, is made evident, again, in the comparison of the cardiac affections of men with those of women and children. Moreover, we shall not forget that unusual exertion may reveal a latent defect in a heart which in ordinary circumstances, or for some time at any rate, would have passed as sound. In men beyond middle life a breakdown of the heart is often thus acutely determined ; but in later life it is usually very difficult to distinguish between lesions due mainly to variations of stress and resistance, and those due to the tooth of time.

For the matter of cardiac physics, and especially for the coefficients of cardio-motive energy, the reader is referred to the article on this subject (p. 11) ; but it is for us here to consider the modes in which muscular effort may affect the heart and greater vessels. And for this purpose some further classification must be made. We must divide acute strains from chronic ; the effect, that is, of some instant or brief stress

upon a sound heart from the deferred effect of a more protracted labour : we must divide heart stresses in the young from those in elderly men ; thirdly, we must distinguish injuries of the valves from those which in the first instance affect the walls of the heart.

Stress-injuries to the valves fall chiefly on the aortic area ; as these are described in the article on Aortic Disease they may be dismissed from this place. Disabilities of the myocardium, in so far as they are attributable rather to disease than to exertion, have also their appropriate article (p. 105). There remain for consideration here, then, acute over-stress in young persons and acute over-stress in elder persons ; in both classes affections of sound hearts, or of hearts in which disease, if present at all, was latent ; and thirdly, chronic over-stress in older persons. I am not satisfied that chronic strain apart from disease by infection or mechanical defect, occurs in young persons, such, for example, as schoolboys. In alleged cases of strain, apparently chronic, other factors, sheer fatigue, infections more or less latent, anaemia, dyspepsia, insufficiency of sleep, the worries of lessons, puberty, inherited nervousness, and other such incidents have to be taken into account, and offer us indeed very complex problems for diagnosis and for treatment.

To avoid a return upon the subject, I may say here the few words I have to say on these cases of *Fretful Heart.* They form a large part, indeed a predominant part, of the number of those regarded as "over-strained at school," "victims of school-runs," and so forth ; youths who come up to a university at an introspective age, disturbed by the incidents I have touched upon rather than described, slackened by suspensions of exercise and discipline, alarmed by doubts about heart disease, discouraged by subjection to Nauheim or other imposing, and in these cases much over-wrought, methods of treatment, and perchance with records of intermittent albuminuria. Now if a case be such an one as I have sketched—one of fag, indisposition, or nervousness rather than of heart affection—it must be taken at once out of this category, not only for clear diagnosis but also for efficient treatment. The physician should do his best to wean such a youth from introspective and valetudinarian habits, not to preach to him on perilous expenditure of unmeasured energy. His mind must be diverted from his heart, temperate exercise may be beneficial even at once, incidental causes must be averted, and mind and body refreshed as a whole. Instability of tone, vasomotor and cardiac, is a feature of most of them. For further consideration of cases of this kind, however, the reader is referred to the article on "Functional Disease of the Heart" (p. 496).

Physiology of Stress.—In now turning to consider the effects of heart stress, as discriminated from these manifold functional disorders, we must see how far contemporary physiology can carry us. It is true that clinical experience teaches us many things of which physiology can give an imperfect account or none ; yet to go with physiology as far as we can is not only to tread so far on relatively firm ground, but also to provide a discipline and a preciser terminology. Ignorance of physiology

and the use of loose or dog-greek words and phrases leave the physician too free to "pan out." Let us begin with blood-pressures.

If the reader will take note on starting for some exercise in which the pulse can be watched, such for instance as a hill climb, he will find that, after a variable interval, usually two or three minutes, the radial artery will open out rather suddenly to something like double its former capacity, a movement presumably universal in the large musculo-cutaneous area. This expansion must signify a like fall in the peripheral resistance, and usually corresponds to an ampler output. Thus an absolute fall of arterial mean pressures may be established; but in the heart's work there are other factors besides pressure and expansion, of which rate and output per minute concern us especially. If, as the conditions of the work are equalised, the pulse be watched a little longer, this amplitude falls a little, the artery is perceived to close down on a somewhat smaller content, and the respiration is easier.

To gauge *blood-pressures* during ordinary muscular exercise has hitherto been found impossible; no sensitive instrument can withstand the racket.[1] What has been done therefore is only to measure the effects of brief efforts, such as lifting weights; those of respiratory stresses, as in Valsalva's method; and those of passive movements, as in massage. But it is obvious that in none of these trials are the systemic effects of exercise demonstrated continuously in their integrity. Massage, over considerable areas, at first raises the blood-pressure; then by redistribution of the blood and increasing velocity the pressures fall to normal or less (Brunton and Tunnicliffe). To increase the pressure suddenly by some 20 per cent, as by lifting a weight, gives the adaptive machinery of the system no adequate opportunity of counteracting the stress. In forced breathing, with the body at rest, the periphery is not opened. In massage the respiration is not on trial. Not only is the field of experiment thus narrowed, but in man the manometer gives us with fair approximation the maximum pressure only; to record the arterial pressures of a cardiac cycle we need of course the duration of the maximum pressure as well as its degree, and also the degrees and durations of minimum and mean pressures. Some observers believe that when all adjustments have come about—a stage reached much sooner in well-trained young men—mean arterial pressures fall to moderate levels. A high maximum pressure may be of short duration in the cardiac cycle, and as the periphery is thrown open, minimum pressures may fall considerably. Moritz is of opinion that during exercise systolic pressures are continuously excessive; but the latest attempt—so far as I know—to grapple with this inquiry is that of Strasburger, who has recorded pressures instantly on the cessation of doses of work (*vide* p. 224). From these researches he concluded that the maximum and minimum

---

[1] Experiments on animals at work (horses and dogs) with a manometer in the carotid, can hardly be trusted, even when performed by Zuntz (and verified by Kauffmann), who recorded a gradual fall of mean pressure (by some 10-15 mm.) during a slow pull uphill. Speaking generally our instruments can give us only static when we want dynamic records.

pressures stand in no constant relation to each other ; their curves shew no parallelism. Strasburger in his previous researches seemed to me not to pay sufficient attention to the variations of the periphery ; he had shewn, however, that " diastolic pressures " under muscular exercise may fluctuate by 100 per cent. Thus in the same person on different trials, and again in different persons, the mean pressures and the work-quotient per minute must vary considerably. Roy and I, with his mano-meter, took curves from various persons while they held steadily 10 kilos in the hand for as long as possible, and found the initial rise to range about 25 per cent ; as the experiment continued the wave-apices would vary from 110 to 150, and wave-bases from 75 to 110. Dr. L. Hill likewise finds arterial pressures to exceed ordinary levels for the first ten or fifteen minutes of exercise ; then, as the periphery opens out, to fall. His records suggested that at moments the pressure might be even doubled. Thus Sir James Barr also, with the sphygmograph, says that the upstroke, at first oblique and broad at the summit, becomes more abrupt and vertical ; the summits are sharper and narrower, and the falls more precipitous. On the whole it seems probable that during exercise the mean arterial pressures must exceed that of rest, but not enormously sustained, nor without compensation ; and the heart, after exertions which have drawn upon its reserve, then enters into the phase of low rates and low pressures demonstrated by Dr. Michell (p. 200). Thus, if we take the daily round of trained men, arterial pressures, by long intervals of reduced friction, may on the whole not exceed, if they do not fall below, those of healthy sedentary men. During the rest the volumes of blood coursing through the repairing muscles may be profuse, but they flow at a low resistance. We are apt to conceive too uniform a notion of blood-pressures, to regard them as moving with a piston-like action on reciprocating planes ; whereas a better comparison would be with the waves of the sea, or with the wafting undulation of a large bird.

There is no reason then to assume hypertrophy of the left ventricle, nor excess of wear and tear, unless the work be continually such as to drive the maximum pressures to the limits of tone and elasticity, limits which in the young we know to be very spacious. With older persons the conditions are not so safe. In them, as years advance, these limits, even in health, are gradually diminishing, and, as the muscular powers do not diminish in proportion, men passing their youth are too often tempted to persist in exercises which in their youth were reasonable and wholesome. However, partly by instinct partly by common sense, the maturer men usually fall in with certain precautions. Although we can scarcely admit that estimates of minimum or mean pressures are as yet precise enough for capacity-quotients, yet from other researches we do know that as men grow older maximum pressures for equal efforts tend more to bounce ; probably because the universal readjustments are not so fluid. The cardiac reserve is also less ; many experimenters have found in elderly persons that during exertion arterial pressures may begin decisively to fall. As regards output, without trustworthy rate and

resistance data, the beat-volumes of the left ventricle under exertion cannot be closely calculated. But the evidence of Haldane and Smith's respiratory gas-tensions goes to shew that in exercise output increases largely. Roy and Adami demonstrated that on enlargement of the ventricular cavity it might increase, or at any rate not diminish, even if the residual blood in the ventricle on each stroke also increased. After the initial stage of exertion, however, orthodiagraphic shadows seem to indicate that the contraction-volume of the left ventricle does not increase but diminishes rather; but this can be shewn only in persons under the Valsalva experiment, not in exercise. In the initial stage, before the periphery expands, arterial pressures jump up; the output of the left ventricle increases in greater ratio than the fall of peripheral resistance and the relief by sweat; then, while the musculo-cutaneous areas are thrown open, probably the splanchnic area, so far at any rate as muscular compression and thoracic aspiration are concerned, is constricted. Thus the blood is delivered in large volumes to the right heart and the widely distensible pulmonary artery, then often mistaken for the left auricle. In this phase, unless by training the thoracic muscles and associated nervous and other mechanisms are prompt to throw the lungs open fully and quickly, the right heart is subject to excessive stress for a period longer or shorter in proportion to the training and capacity of the individual, and the left ventricle may not fill. At such periods the orthodiagraphic records may correctly reveal a diminution in its size. Drs. L. Hill and Flack found that on administering O_2 to the livid athlete the radial artery became quickly fuller, and frequency was reduced. As Dr. Keith has just shewn, the alveolar expansion of the whole lungs is not so simple a matter as we have supposed (p. 219); and the problem is further complicated by the vasomotor constriction needful to support arterial against venous pressures. As we may note in ordinary valvular disease, venous pressures cannot be allowed to predominate, nor even distantly to approach equality with the arterial; we instinctively feel that efforts which fix the chest, even in young persons, must be of short duration. Training, then, apart from diet, is something more than an economy of muscular adaptations; it is also, in the circulatory concert, a development of the muscles of the thorax, an unfolding of the lungs, and a swift play of vascular and other nervous, and probably also chemical, reflexes. My observations on angina pectoris have led me to think that much of the adjustment of arterial pressures depends on a peculiar sensitiveness to tension in the first or suprasigmoid part of the aorta.

One more coefficient there is in exercise which has received no attention. In mountain-climbing it is now proved that the red corpuscles are not only more apparent but positively multiply. Yet if, for instance, at M. Vallot's observatory, the reds are found to attain 8,000,000, the increase of friction must be very great—at first sight the increase approaches 50 per cent. This condition may be one reason of the somewhat peculiar facts of mountaineering physiology. And further researches have shewn

that the need for more oxygen, imperative as it is at high altitudes, is relatively considerable during exercise at ordinary levels ; so that here, too, the reds are said to increase by some 15-20 per cent (p. 201) ; an increase of friction not so considerable, yet no negligible fraction. The frictional coefficient is modified no doubt by other variables, such as the enlargement of the blood-channels, and alterations of the form and volume of the reds by decarbonisation (Bence), but it is not annulled.

As with pressures so with *rate* ; under exercise the pulse of course is much accelerated, but to degrees which vary widely for different individuals. On the whole the acceleration is much more moderate in trained persons, and passes off sooner. In Dr. Michell's notes we observe that, as with pressures so with frequency, the periods of acceleration are compensated by intervals of retardation. As regards acceleration, Dr. James Kerr, during his residence at Bradford, kindly made some observations for me on fifty-five children, unselected except for health, age, and sex, who were all put to the same short fast run. Twelve boys and twelve girls were eleven years old ; twelve boys and twelve girls were thirteen. These were untrained ; the remaining seven were boys, aged twelve and a half, who had been carefully trained for games. All the care possible was taken to avoid nervousness. Before the run the pulse was taken quietly during sitting, standing, and then sitting again. Their runs were timed ; but a few of the girls, who seemed to be flagging before quite completing their test, were stopped. It was found best not to take the pulses till half-a-minute's rest had calmed the agitation ; then a count was made, and repeated in $2\frac{1}{2}$ minutes. The children aged thirteen, and the trained boys were counted a third time, in $4\frac{1}{2}$ minutes. The mean percentage acceleration of pulse-rate was :

	Time-record in seconds.	Percentage Pulse Acceleration.		
		$\frac{1}{2}$ Min.	$2\frac{1}{2}$ Min.	$4\frac{1}{2}$ Min.
For the girls of eleven	$35\frac{1}{2}$	32	13	...
For the boys of eleven	$29\frac{1}{4}$	16	6	...
For the girls of thirteen	19	30	11	9
For the boys of thirteen	$16\frac{2}{3}$	19	7	3
Trained boys of $12\frac{1}{2}$	$16\frac{1}{7}$	10	0	0

Some other older untrained boys were tried, and a few pupil-teachers ; but these figures are of less value. The great difference between boys and girls is notable ; perhaps because of the difference in respiratory activity. One conclusion is that at the age of thirteen the pulse-rate ought in five minutes to return to or near the rest figure ; and here comes out a marked difference between the trained and the untrained. The seven trained boys, although a little younger, and run to the same time-record, presented half the acceleration of the untrained boys, and a third of that of the untrained girls ; and in $2\frac{1}{2}$ minutes they had settled down to the rest rate. I find on the notes that in two of the eleven girls and in one of the eleven boys the pulse, taken at the half minute, was very irregular, though in rate not more frequent than in

some others. Of the girls of thirteen, in four the pulse became irregular, and in two boys of this age ; in one boy and one girl, "very irregular." In one boy aged thirteen (untrained), the note is "hurried and grouped beats." It is incorrect to state that the cardiac rhythm is never upset by exercise. The highest rate—at the half-minute—was 172 ; this was in an outsider, a boy of fourteen from a Higher Grade School. In this respect the pupil-teachers, who were probably sedentary students, came out badly ; of seventeen, two had, at the half-minute, rates of 148 and 152 respectively ; in none was the rate under 128 ; in seven it exceeded 140 at this time ; in nine the rate at twenty-four minutes was still over 100 ; in six it had not reached the rest rate in the last count at forty minutes. In $4\frac{1}{2}$ minutes it had reached the rest rate in all the trained boys ; indeed, in the boys of thirteen, probably all active little fellows, the remaining excess at that hour was small. In this class of girls at the $4\frac{1}{2}$ minutes, the rate still ranged, in most of them, about 100 ; in one only, or perhaps two, had it then fallen to the rest rate.

I have said the highest rate noted was 172 ; in eight minutes this pulse had fallen to 112 ; in sixteen minutes to 108 ; in twenty-seven minutes to 92 ; in thirty-two minutes, however, it had risen again to 100—an unfavourable sign : the boy was not well. The next highest rates at the half-minute were (leaving out two irregular pulses not easy to count) 152 and 148 twice, in three teachers, one male, the others female. Among the runners aged eleven and thirteen the half-minute rate moved in the girls around 120, in the boys a little lower. Omitting one apparently nervous boy, only one of the runners aged thirteen was found up to 120 at the half-minute ; the rest were three or four beats above or below 100. These observations of Dr. Kerr therefore corroborate the conclusion of Trautweiler and some later observers in this field ; that 172 is the extreme limit of pulse acceleration in the normal mechanism, and that 160 is rarely attained. If the limit of 170-172 be crossed, we have to do with an altered mechanism ; perhaps with some affection of the medulla ; perhaps with a shift of the cardiac rhythmical centre, and the condition may be called Tachycardia (p. 523). But to call mere frequency of pulse, under this limit, "tachycardia" is either mere pedantry or, falsely, to suggest some new concept.

A very careful study of rate under exercise (chiefly with the ergostat 1000-10,000 Kgm-meters) appeared by Stähelin in 1897. Like Kerr and Christ, Stähelin usually found no arrhythmia, except an occasional extra-systole. In the few exceptions, as in those of Dr. Kerr, perhaps the heart was not quite sound ; it may have been touched by some infection. It is said that effort never causes arrhythmia in the normal heart, but as it appeared in more than one of Dr. Kerr's cases, and some of my own, I think it may result from unequal tensions of blood in the two ventricles (*vide* p. 228). Christ made physical examinations in his test cases, and often found well-marked extensions of dulness ; in some convalescents murmurs became audible (*vide* p. 205). Stähelin also found great individual differences in the active and rest rates. In some, after

the easier work-tests, the return to rest rate took about two minutes; in others as much as ten to fifteen minutes longer. After the heavier exertions a few recovered their rate in five minutes; others took fifteen to twenty minutes to calm down. Training improved the figures; a liberal supply of alcohol set them back.

Dr. Baelz once told me, among other remarkable facts concerning the Japanese rickshaw coolies, that after a 50 miles' non-stop run, the pulse would return to the normal rate almost at once on resting.

On kymographic curves taken after work, the excursions were much wider, and dicroty was much increased, and lower down. This I think signified not so definitely an increase of output as slackened vascular walls. Certainly it did not imply increased blood-pressure. Broadly speaking, Stähelin concluded that immediately after exertion the pulse-rate should fall about three beats per ten seconds; but the fall is more rapid in the first thirty seconds than afterwards. The ordinary acceleration was nearly to double the rest rate; but as the amount of blood on the move is more than doubled (Zuntz), the output must be increased, and therewith the heart's work. In exercise, as contrasted with mere postural changes, the velocity of the blood stream is certainly increased. It is asserted that even before the periphery opens, the output may be sixfold! Such quotients must be received with caution; and I think, indeed for men in fair condition a doubled rate is excessive. In Dr. Kerr's trained boys the increases appeared to be much less; but the first counts were made, it is true, after half-a-minute's rest. Sir Hermann Weber, during an ascent at the beginning of his holiday, noted the initial rise of his pulse-rate to be from 74 to 122; but after a week's active walking the rise was only from 74 to 105. When climbing hills my own habitually slow pulse of about 56 will rise, when I am in training, to rates between 90 and 110.

Respiration.—The advantage of training is well illustrated on the side of respiration also; training is not for the limbs only. After severe exertion in an untrained man the rate may keep up between 30 and 40 for half an hour. In a trained man it should not be more than 30, and in a quarter of an hour should be down to about 20. Omitting the unsettled factor of the red corpuscles, the oxygen-capacity of the blood per unit-volume—the chemical rate—does not change much in active work; so that the absorptive capacity is still represented fairly well by the ordinary difference between the venous and the arterial blood. The greater demand therefore must be provided, or largely provided, by more heart work; that is, by the passage of more unit-volumes through the circulation. I say "largely provided" because we have seen again and again that, in meeting this need, the heart is aided by many readjustments. Stimulation of the peripheral end of a muscle-nerve produces considerable increase of respiratory movement, even when the muscles concerned have been separated from the sensorium by cutting the afferent paths. The net advantage of the many variables, however, varies with different persons, and in the same person on

different occasions ; especially in respect of training and no training. Dr. Waller, Tigerstedt, and more recently, from the clinical point of view, Dr. A. Morison have carefully discussed the effects of the respiration upon the functions of the heart. The Haldane-Smith method of estimating blood volumes and total blood-mass has, by Plesch and others, been somewhat simplified. Moreover, Dr. Keith has published further observations on the mechanics of pulmonary alveolar expansion, and revised our common knowledge of the need of development, for hard exercise, of the respiratory muscles ; so that larger masses of blood may be received and passed onwards to the left heart again. It would seem at first sight that distension of the alveoli, by flattening the pulmonary capillaries, would impede the run of the blood through their meshes ; but if the increase of friction be so compensated that there is no fall of velocity, the blood exposed in more attenuated streams may be aerated more rapidly, especially if, as Bence states, oxidation by altering the shape and volume of the red corpuscles reduces the viscosity. Accordingly, although the velocity in the pulmonary artery is lower than in the systemic arteries, we shall see, under the head of hypertrophy, that in athletic men the right ventricle is strengthened. However, we must not regard this problem too mechanically. Respiration is not combustion, as the school-books too often assert ; recent experiment (Haldane, Pembrey, Zuntz and Geppert, Plesch) proves that the function is one of gaseous balance in the alveoli ; and probably the alveoli themselves, by a specific endowment of their epithelium, have some share in the function, in which the associated nervous centres also are engaged. Dr. Haldane regards the function, in its integrity, as more than mechanical or even chemical, and distinguishes it as vital.

Now, to pursue these recent investigations, the ordinary oxygen-difference between arterial and venous blood, at a pulse-rate of 70, is about 33 per cent, the equivalent in the adult of about 210 c.c. O_2 per minute ; on this basis the cardiac output has been calculated at about 43 c.c. If a man does no more than rise from rest to walk on the level, he doubles his consumption of O_2 ; and he doubles his O_2 consumption again when he doubles his pace ; or if, at the same pace, he rises 100 metres in one kilometre (Zuntz and Schumberg). Therefore the heart, by acceleration and otherwise, must provide and cope with largely increasing volumes ; it must cope with considerable increases of rate, of beat-volume and of time-volume ; conversely the O_2 estimate may be regarded as a fair test of such additional work, and the moment output begins to fall trouble begins.

To discriminate the respiratory coefficient so far as may be from other factors, we may turn to observations on the mountains ; for example, to M. Vallot's laborious researches on Mont Blanc. In round numbers, at 5000 metres (= 400 mm. Hg), the oxygen of the atmosphere falls to about half that at sea-level ; and the temperature very nearly ·5 C. per 100 metres rise. On the summit water boils at 84·3° C. Now on Mont Blanc, Vallot, testing trained against untrained men, found that

in untrained men the respiratory enlargement amounted to 45 per cent as compared with Chamounix; in trained men only to 30 per cent; thus the trained men had previously acquired a fuller thoracic capacity of 15 per cent. Furthermore, the capacity of the untrained man did not improve immediately—indeed, at first, it retrograded somewhat; it was during the second week that it developed and surpassed its initial record by 80 per cent. And on returning to Chamounix, some of this acquired excess of capacity was retained for a considerable time. Even the trained man, by stopping at the summit, developed 50 per cent more of acquired capacity. These facts may be illustrated by the example of the rabbit which, as compared with the hare, is a creature of no endurance; on alarm, it has but to scuttle into a stuffy burrow. Accordingly, it is a much shallower breather, and cannot live at very high altitudes, where it survives for a few days only; its organs being found, after death, in a state of "fatty degeneration."

Orthodiagraphic records have shewn that in this enlargement of the lungs—which by some has been called without evidence an hypertrophy, by others, in vague confusion with disease, "emphysema"—the chest-walls and the diaphragm move in all radial directions from the ideal thoracic centre. Moreover, by this method, M. Vallot's observation that some fraction of this acquired magnitude persists for a considerable time after the cessation of the excessive demand has been corroborated. Now in lungs thus amplified there is a corresponding increase of residual air. At first sight this seems a disadvantage; but Dr. Haldane quickly explained to me that, on the contrary, by this reservoir the gaseous exchanges are made less intermittent, more continuous; that the reservoir acts as the bag in the bagpipe, or the comparatively voluminous and elastic arch of the aorta in the arterial tree.

The most important condition in the refilling of the heart during diastole is, of course, its own previous contraction; but how far the heart itself exercises suction upon the blood as it enters is a problem which, as yet, is far from being solved. In the well-known experiments of Goltz and Gaule, negative pressures in both right and left ventricles were recorded in dogs, ranging from 100 mm. of water in the left, and 10 mm. in the right, to three or fourfold numbers. Dr. Rolleston's experiments also indicated that the minimum pressure in the ventricles may fall below that of the atmosphere, wherein he more or less corroborated the results of Goltz and Gaule; but Rolleston found also that negative pressures depend upon too many variables to be constant. Many of his tracings indeed shewed no fall below the atmospheric pressure at the time of experiment. Tigerstedt also, in the last edition of his work, agrees that the conditions of an effective suction are so many and complicated that at present no accurate estimate can be given on the matter.

As regards atmospheric pressures, which in mountaineering vary so widely, it is obvious that the pressure of the atmosphere on the extra-thoracic veins must be greater than that which, through the lungs, can

be exercised on the veins within the chest; thus, these veins and the heart must be distended in proportion to the difference.   In inspiration this difference must be increased; the negative pressure within the chest must be increased, and in some proportion to the depth of the inspiration.   The intrathoracic veins, the auricles, and the pulmonary artery must be distended, and if a sufficiently deep inspiration were held on the circulation must cease.   In expiration, on the other hand, the negative pressure on the chest falls, and the access of the blood to the thoracic veins is slackened.   Even the systole of the heart itself, by which movement some of the blood is driven out of the chest, must exercise an influence in the direction of suction towards itself, or, more accurately, towards the great venous reservoirs.   Whatever values then we put on these several factors, we perceive that violent exertion must be attended by a considerable oscillation of pressures in the thoracic veins and right heart.   The thick-walled ventricles and the aorta in which blood-pressure is high will be least influenced; but the right auricle and the vena cava, which are thin-walled and almost at zero pressure, will be sensibly affected, and thus the amount of blood-flow to the right side of the heart; and in its turn the left side also will quickly be affected.   The left side, then, will drive forward a larger quantity of blood soon after inspiration, a smaller quantity soon after expiration.

By his colorimetric method, Plesch, in Krause's laboratory, has endeavoured to apply the Haldane-Smith method to clinical use. He has calculated again the consumption of O_2 per minute, the percentage of CO_2 in the blood of the right heart or pulmonary artery, the percentage of O_2 in the arterial blood; and so the cardiac output per beat and per minute, the consequent demand for O_2, the total mass of the blood, and the circulation-time (*Umlaufszeit*).   This he puts with a pulse of 65, at 55 seconds; and the quantity moved at about four litres. A practical illustration of these processes is presented in the experiments of Dr. L. Hill, and others, who, advancing upon the old device of preliminary deep breaths, have administered oxygen to athletes during and after exercise, with marvellous effects of refreshment or restoration.   Dr. Hill gave "three bags" of oxygen to footballers at half time, with the result that they played up fresher than at the beginning.   At the end of a game a player not in good training, by whom "eight bags" had been inhaled (*i.e.* "a four-minute inhalation"), said, "I felt as if I had not played at all; and next day I had no sense of stiffness, which is quite different from my usual feeling after a game when not in training."

Hill observes that in football and other hard games the faces of all the players become bluish.   It appears then that, great as is the cardiac reserve, much larger as its work may be in comparison with the work of rest, effective as, with the co-operation of lively nervous centres, it may be in the quotient of frequency and amplitude (time and beat units), it does not, after all, supply the requirements of the blood in severe exercise, not fully even in trained men enriched in red corpuscles.   For, as on the

contraction of a skeletal muscle its blood-vessels open out so widely that it can deal with at least one-third more blood than when at rest, and as even at rest the skeletal muscles may hold something like a quarter of the blood in the body, much larger charges of blood are forced successively by the muscles beyond the valves of the veins and into the sphere of the respiratory suction. It is evident, then, how heavy must be the disadvantages of the untrained man. In him the blood is thus heaped up at the gates of the lungs; so that it is on the right side of the heart that the stress falls, as I demonstrated in 1870. Roy verified this conclusion, and it seems now to be generally accepted. Some recent writers, it is true, have been again attributing "heart-strain" to the left ventricle; but I shall shew that this chamber suffers not much, perhaps very little, from high intra-aortic pressures, but rather from the fatigue of enhanced output per second, or from more specific toxic influences. Dr. Michell's outward percussion-shadow in the tired heart (p. 203) is not a strain, nor even a forcing under excessive tension; it is the atony of fatigue. The man himself is overworked or stale; pass your finger down his cheek and a red line will follow; you may write upon his abdomen. That the left ventricle, when so enfeebled, is more obnoxious to strain from accumulating residual blood in diastole is true; but, happily, before this time comes about, corporeal neuro-muscular lassitude brings the exercises to an end.

It is difficult to reconcile these needs and calculations of large outputs, even if residual blood were minimal, with the orthodiagraphic records of de la Camp and later workers in this field. Moreover, the large mass of notes on physical signs by a multitude of clinical observers cannot be set aside as wholly, or even largely, fallacious. The discrepancy probably lies, as I have suggested, in the partial conditions of the Valsalva and other artificial circulatory compulsions under which the experiments are made. The clinical experience of changes of heart-volume under actual exercises, as we shall see under the section of Symptoms, are far too weighty to be set aside under the arbitrary conditions of the laboratory. That in the Valsalva position the blood is hindered on its way round to the left ventricle is pretty certain; this, however, in the exercise of trained men, is but the preliminary stage, and one on which release by compensatory adaptations soon follows. This (the Valsalva) test throws more light upon the interesting observations of Dr. Macnaughton upon the dilated right hearts of choristers.

Circulatory Coefficients.—The conclusive measure of cardiomotive potential, if by any ready method we could calculate it in practice, must be the time-volume—the volume of blood delivered into the arteries per minute, the sum of the beat-outputs, each of which, approximately speaking, will be inversely as the pulse-rate. As tone slackens, the residual blood on each contraction will increase, and the less will be the output per minute. The number of beats, then, multiplied by the mean quantity per beat, will give the work done; but it does not give us the total cost of the tale of work, for in so far as the aortic pres-

sures may be enhanced, the labour of the delivery is so much the greater. Even in the trained man all these coefficients are higher in work than at rest, though, as the circulation is habituated to the demand, the many and sensitive adaptations become so concerted and balanced that, whatever the gale, the heart runs on a comparatively even keel.

If, then, we are to understand the complex conditions of the circulation in increase of muscular activity, we can do so only by analysing the coefficients, by whose concert a larger output of blood is delivered in unit of time. These coefficients are output per beat (O), frequency (F), "pulse-pressure" (P), and width of channels (W). By the researches of many investigators, especially of Marey, we have been brought nearer to this kind of knowledge. F is easily determined, P is approximately calculable, and for O we have in the laboratory the Haldane-Smith-Plesch methods, which may soon be still more simplified and improved for clinical purposes. For the moment we must proceed with what knowledge we have, and try on the threshold to avoid the confusion of these coefficients, or of any of them one with another ; for instance, the too common assumption that output can be measured as pressure, or conversely. The fourth coefficient, width, is that which still eludes us ; expansion and contraction in areas larger and smaller depend on fluctuations of the vasomotor system (W), which are as pervasive and influential as they are elusive. Indeed, upon this unknown factor depends the balance between pressure and output. Sometimes this mechanism dilates to relieve the heart ; at other times, apparently as critical, it contracts, to keep up the arterial pressure-head, even at the cost of velocity and of cardiac energy. This inconsistency probably depends on alternations of the splanchnic and musculo-cutaneous vascular areas. But if we find the "pulse-pressures" fairly constant we may perhaps disregard it. Tone is a function of vessels as well as of heart ; and in atony of vessels pressures may fall to syncope. Thus perhaps is to be explained the readier supervention of "heart-strain" in Europeans, and even in hardworked coolies, in the tropics (Carnegie Brown), or in hot atmospheres (Fisher). It is not proved, nor very probable, that it is the heart which is primarily concerned in this exhaustion. The disability does not consist in a strain, but in a vasomotor languor (+ W) which lets down the pressure-head, and may well be of central origin. I saw recently, with Mr. Wingate, an undergraduate who had cycled under a blazing sun from Ely to Cambridge with only a small thin cap on his head ; and on arrival he sculled on the river. During the evening he felt excessively irritable, and then collapsed into bed. On my visit, in the next forenoon, Mr. Wingate warned me against raising him from the pillow ; and, indeed, on merely raising his shoulders, by way of experiment, the face blenched and the radial pulse so promptly went out that I dropped him in no little alarm. Such failures, in various degrees, had already happened under Mr. Wingate's eye. We dismissed the diagnosis of "heart-strain" for vasomotor exhaustion,

probably central in its seat; and gave a relatively good prognosis, which was, fortunately, justified. In lesser degrees such cases are not rare, and are not to be classed with " heart-strain."

It is rather by the simpler and more ordinary functions that we may attempt to ascertain the parts of the various factors which I have enumerated. On recumbency in health output falls, and with it fall frequency and pressures—minimum pressures chiefly, maximum in less degree. After a meal, rate and output both rise, but pressures depend on the attitude of the visceral nervous system. For instance, if these peripheral vessels are slack, but full, the minimal ("diastolic") pressure may keep up, or even rise. It has long been known that on setting a man to work, minimum as well as maximum pressures and frequency rise at once; Dr. Williamson's records of systolic pressures rose to 160-170 mm. But from Masing's well-known papers we have learned that even in the vigorous heart these changes happen in very various degrees; so what we need, namely, output per minute, may positively fall. Krehl's pupils, especially Tiedemann and Höppner, have repeated these experiments; and from the sum of opinions we may take the rule to be that the quotient $F \times O$ rises 30 per cent, the quotient $O \times P$ 12-20 per cent. As these values seem not to change much during the first five minutes of rest, they come within the reach of instrumental appreciations which, during exertion, are impracticable. It is interesting to learn also that what holds thus for the healthy heart, holds also for diseased hearts in which—as in many cases of valvular defect—compensation is fairly well established. But, after all, the elusive factor of vasomotor activity (W) is always secretly modifying the sum of events. For instance, Tiedemann, in distinguishing between voluntary fatigue ("einfache Muskelermüdung") and cardiac fatigue, reports, what we may call the cardiac paradox, that in well-marked heart fatigue ("ausgesprochene Herzermüdung") pressures may even rise during this five minutes, whilst in mere muscular tiredness they cease to rise, and may fall. In the midst of such complexities we have no data or criterions to guide us. However, if in this five minutes' period the rise of the $O \times F$ quotient falls below rest value, as Tiedemann found in certain convalescents, the heart is weak. And, indeed, if it be slight or transient, there is matter for suspicion; for output may be falling while the other factor of frequency is not falling, and, indeed, in fatigue may rise so substantially as not to diminish the quotient, although notwithstanding the pulse may be wobbling and the time-output deficient.

As to pressures, the quotient $P \times O$ should still be standing at 12-20 per cent, but if the heart be weak it may fall to rest value, or below it. In the following 10-15 minutes—individuals differ a little—the quotient of all three ($F \times O \times P$) should reach the normal, voluntary weariness or not; time volume, therefore, should be at rest value; for although F be still plus, O and P have fallen; and in the weak heart, if O and P be still lower, frequency runs abnormally high. Thus also in the variations of age and sex the quotient $F \times O$ is fairly constant, as the frequency,

though it preserves no regular inverse ratio to output, yet runs generally to the contrary.

As a good example of what training will effect, I may refer to the observation of Dr. Gordon (under Dr. G. A. Gibson's direction) on a "champion club-swinger." The record was: heart slightly hypertrophied; maximum blood-pressure (two days before performance) 105 mm., and the diastolic pressure 70 mm. ; at the conclusion of the exhibition, 115 mm. and 80 mm. respectively. The pressures fell to the ordinary level in one hour. The pulse never varied at all in rate (twelve hours' swinging), but stood at 84 before and after (not taken in morning in bed). *x*-rays revealed no cardiac change. In two football players after very severe effort the systolic pressure had fallen 25 mm. Selig also, in four fatigued football players, found a fall of the blood-pressure by 20-46 mm. and a dislocation of the apex outward (*vide* p. 203).

In the light of these facts we have now to consider the phenomena of *fatigue* in experimental and in ordinary work. Every striated muscle has a point of tension which gives its worth-maximum. The skeletal muscle can at a pinch contract with twice the energy of an ordinary effort; the heart-muscle, however, which is of different construction and has also a special blood-supply, is capable of very much more than this. The blood goes round the heart many times for once in the voluntary area, so that it is in the skeletal area that fatigue arrives first, and damps down the exertion before the healthy heart is placed at any great disadvantage. And when it does suffer, the depression may come indirectly through its nervous elements, especially, perhaps, through the associated centres. It does not seem probable, nor does experience dictate to us, that ordinary catabolites, even if accumulating in the heart and reducing its energy, would act upon its structures corrosively, as infective poisons only too often do. Fatigue is part of the experience of every man ; yet its nature is ill understood. It contains a volitional, a psychical, element which is beyond analysis. The man who is wearied by a few miles' solitary walk in monotonous ways expends many times the energy with comrades, on the hills, in the hunting-field, or on travel, and is cheerful at the end of it. It is said that some of the staleness of training is attributable to the boredom of the whole business. I think athletes, like geniuses, need long fallows. Fatigue—or, rather, the sense of it—seems to be a conservative provision more or less proper to the voluntary neuro-muscular system. We do not hear of a fatigued diaphragm, and only by analogy do we speak of fatigue in the viscera. Da Costa (18) endeavoured, chiefly by clinical history and results, to discriminate between this nervous or psychical element and direct exhaustion or degeneration of the myocardium. Klemperer made the curious observation, which I think has been corroborated, that hypnotic suggestion of work raises arterial pressure for the moment almost as much as the work itself ; and that even in a subject awake concentrated attention or intense idea (Vorstellung) of work has a similar effect. Kornfeld noted that an attempt to walk in a straight line for a sufficient distance raised the pressures more

than a free walk of the same length ; but surely the line-walking occupied
more muscle, or at any rate, in persons unused to the trick, muscle
less economically applied (*vide* p. 225, and also Zuntz and Schumberg).
Habit and training knit up the automatic economy of muscular
synergies. Morton Prince investigated the factor of fatigue by summon-
ing, flurrying, and hustling certain firemen selected as healthy. In
seventy-seven of them before engagement in hard work, violent
heart's action and tremor came on ; and, in some of them, temporary
enlargements of the heart—both right and left—so considerable that,
alive as he was to percussion fallacies, the evidence seemed undeniable.
He quotes Munro, who carefully noted similar transient effects before
surgical operations. Vexatious work, according to the admirably com-
plete and controlled experiments of Zuntz and Schumberg, dissipates
energy, as measured by oxidation. A sore foot on the march caused an
additional discharge of O_2 by 18 per cent. "March fatigue" produced
shallow, but more frequent respiration, and more oxidation ; but if the
work and purpose were completely changed, this excess was reduced.
The fatigue of disheartenment, as in a defeat or losing game, is well
known. Some men cannot play a losing game ; they fall off in both
energy and dexterity. I never have been able to agree with German
physicians that gymnastics, valuable as they may be as an accessory drill
for raising the muscular level all round, are better than games in which
the psychical element is far more abundantly engaged and inspired.
Moreover, our preceptors ought never to forget that to train the muscles
only has some danger of degradation about it.

In the first stage of exercise, then, the stress is upon the right
ventricle ; a block which in the trained man is swiftly relieved by
enlargement of the thorax and lungs ; then in their turn the left
chambers have to cope with the abundance. And, for the moment at
least, arterial pressures do rise suddenly and enormously ; soon, however
—and here again more rapidly in the trained man—relief is given by
expansion of the peripheral neuro-muscular areas, and resistance in
the aorta falls. Drs. Edgcombe and Bain have noted during exertion
a fall after the initial rise ; notwithstanding, it seems ascertained that
mainly during the whole course of hard exercise arterial pressures
keep up on all planes, maximal, minimal, and mean, above the normal.
As with all the organs of the body, so the heart is capable of far more
work than ordinarily we demand of it. For instance, if constrictions in
other areas were not more than an equivalent balance to the cutaneous
and neuro-muscular expansions, arterial pressures might chance to fall,
even perilously. At all hazards the "pressure head" must be kept up.
So imperative is this condition that even in the diseased heart, so long
as it is fairly competent, the pressures in exercise are maintained above
the normal of rest. A considerable fall of arterial pressure in exercise,
or as soon after it as the observation can be made, is said to be of
sinister meaning (p. 205) ; if the fault be with the heart it forebodes
its failure. But here again the fall, if moderate, may be due to some

relaxation of the periphery of which we have no measure ; or thoracic pressure may exceed abdominal pressure. The left ventricle has probably, in work, to deal with even a higher sum of peripheral resistance ; certainly with larger quantities of blood, — larger time quantities, and, if the exercise be active, and the frequency moderate, with larger output per beat. Whatever orthodiagraphists may say, it would seem that the volumes of the left ventricle, after the first or pulmonary stage (*vide* p. 230), must be increased, especially in trained men in whom pulse-acceleration is less.

From these considerations we may form some notion of the manner in which stresses on the left ventricle may increase and even become harassing. They come on after the lungs have opened, and when the heart has got into double swing ; and are exerted either by peripheral constriction or by residual blood accumulating in growing disproportion between contraction-volume and the output. Under the conditions of exertion we meet with three kinds of dilatation of the left ventricle (*vide* p. 439, Aortic Disease)—that of active adaptation, that of atony (fatigue), and that of strain—of stretch, that is, beyond the elastic limits of the tissue, a lesion which is more or less irremediable. Now on active dilatation we need not dwell, it is a physiological condition ; and Roy demonstrated that dilatation is not inconsistent with constancy of output. Of atony we have spoken in part, but we have yet to inquire anxiously if the dilatation of strain may occur, and if so, how readily, and why. In respect of the healthy heart, we have surmised that, before this stage is reached, general neuro-muscular and mental fatigue will have reduced the work. The left side of the heart is not so extensible, it is true, as the capacious reservoir consisting of venae cavae, right auricle, ventricle and pulmonary artery ; but in the young intact heart it is extensible enough. Of the transient systolic murmurs which arise in these conditions we shall speak in the section on Symptoms.

Heart fatigue is to be distinguished from systemic fatigue. In the voluntary system we know that fatigue consists in the accumulation of excretory products beyond the temporary capacity of excretion. Is atony of the heart of the same nature ? Is it " heart fatigue " ? This question is scarcely ripe for solution ; but loosely and provisionally we may answer in the affirmative. We read indeed of " autocurarisation " of the heart, but such fine words provoke suspicion— the suspicion that they are imposed upon us for knowledge. Still, what we recognise clinically as cardiac fatigue may be something of the kind. Juices or alkaloids derived from other parts, and supposed to be contaminated by products of muscular fatigue, have proved very poisonous to animals ; not only so, but in small doses immunity can be set up against them (Brunton), and training may partly consist in the development of anti-bodies to such metabolites. If thus something of their protective function in damping down excess of work is bargained away, on the other hand, by the training, more active means of dealing with them are also developed. The margins of endurance have been

pushed farther back. An endeavour was made to gauge such fatigue by measuring the length of the systole in proportion to the pause; but Dr. Thomas Lewis in a private letter tells me that he has been unable to determine such measurements, or to obtain from them even rough clinical service. Janowski believes that he is able to recognise these magnitudes, but that in health and at rest the length of the systolic wave is not a constant. The height of the wave is perhaps more to the point; or would be were we sure of a constant arterial calibre, and constant instrumental adaptation and inertia. Many of the published curves are worthless. Dr. Mackenzie would, I think, suspect the quality of any heart in which a pulsus alternans occurred—*i.e.* a drop in the height of a wave without a corresponding extension of diastolic rest. (*Vide* p. 21 and Fig. 9.) In respect of the fluctuations of exercise I am scarcely prepared to agree with him. I feel sure that I have perceived in myself short beats in excessive stress, especially on an occasion when I was both running and shouting with my college boat. But to make precise measurements even immediately after exercise is almost impossible: in tranquillity it is hard enough to prevent instrumental shifts and to allow for inertia. Stähelin, however, by many precautions, has made it seem probable that in cardiac fatigue the wave becomes more frequent, lower, and monocrotic; and that on rally it enlarges and regains a high dicroty on its low base line. In some cases he noted fluctuations of this kind in the period of recovery, but the difficulty in such observations of excluding respiratory and vasomotor waves is obvious. Still, when a pulse does not otherwise alter its form, Stähelin thinks these interferences may be disregarded. I may add here that Stähelin also rarely found arrhythmia; when it occurred it took the form of a smaller intercalated beat or a transient geminate group. We have seen that the finger tells us how in fatigue the pulse-rate falls more slowly and oscillatingly to the normal (Kerr, Masing, Gräupner, and others); so that, if the state of training be borne in mind, we are not without some handy means of guessing at the functional capacity of the heart. Yet individuals seem to me to differ a good deal in the time - relation of general and cardiac fatigue; in some persons cardiac fatigue treads closely upon the heels of general weariness. Dr. Oliver's observations on the courses of the lymph may be of importance in this connexion; but in muscular exercise the propulsion of the lymph must be greatly accelerated. The truth is some persons have a stronger or better balanced circulatory machinery than others. Athletes, as a rule, are naturally selected; but a few men, more ardent of temperament than strong in muscular frame, may suffer in the contest.

I have alluded to the curious observation that in the fatigue of healthy men, the first change of pressures, unless the fatigue be abrupt, is not in the direction of fall but of rise. Fatigue-products do not enhance pressure, rather the contrary: the rise is probably in the main another instance of the vigilance of the vasomotor centres in keeping up the pressure-head. To them the life is more than the heart. A fall of pressures by the

way of the vasomotor system is not asystolic but syncopic. But there is another factor also of no little incidental influence, namely, the arrears of respiration whereby the medulla is excited, and pressures are raised not —as we now learn—by plus CO_2 but by minus O_2.

Infections.—We have then to admit that it is not easy to discriminate between atony and fatigue; between the mere negative, the merely slackened and the poisoned muscle. And in so far as the poisons may be of its own household, "autotoxins," perhaps it is no great matter. In either case rest and time will tell favourably, as the many experiments on work by convalescents (from non-infectious diseases) have indicated. Pawlow stated, many years ago, that the accelerators fatigue first, when heart-capacity begins to fail. Virtually both conditions are often combined in atony; and, as tone in its descent is apt to resolve itself into other qualities (Gossage), into an excess of rate and of excitability, we meet with the quick and thumping—"irritable"—and fussily ineffectual heart. When, however, we have to do with extrinsic poisons, and especially with those which we call especially by the name of infections, the problem is a far graver one. Extreme stresses upon hearts under this detriment result only too often in strain, and this even in young persons. I have reason to suppose from cases of my own that such stresses may ultimately prove to be of fatal effect; certainly in a large number of cases they are very mischievous. It is of these cases, and of the chronic cases of older persons of whom I have yet to speak, that "Heart-strain" may properly be predicated; though in some of them no doubt the impairment may not go beyond deprivation of tone, in which case the recoveries are not so tedious. We must, I think, take the opinions of Albrecht and the Leipsic school with some salt; especially as they are founded largely on the evidence of the post-mortem table; yet by the demonstration in such cases of foci of myocarditis these physicians have done good service. Such foci, in the favourable and more frequent event, heal into patches of fibre which, if not extensive or ill placed, do little ultimate harm. Thus it is with ordinary cases; but if under such an infection a man undertook severe exercise he would disable his heart seriously, perhaps permanently. Protracted cases of this kind are by no means rare; and maturer experience inclines me more and more to refer the cases of heart-strain in young subjects to this category. Concerning older persons there is another story to tell presently. If the distinction between such a taint and atony be hard to make in the acuter period of the disablement, it becomes pretty clear in the subsequent course of it. To recover from atony is a matter of days or weeks, from strain a matter of months or years. After influenza Dr. J. Mackenzie has found the auriculoventricular interval increased; this may prove a valuable test for infectious contamination. Dr. Arthur Lambert of Harrow, who made heart-stress the subject of an M.D. thesis at Cambridge in 1904, and who has since kindly corresponded with me upon the subject, states that he has always regarded the specific fevers especially as the determinants of heart-strain; anaemia, general debility, and lack of training being subordinate

causes. "Up to 1903," he adds, "I was able to collect a number of cases of heart-strain, and during these years influenza was always with us. After 1904 we saw very little of it (heart-strain); indeed, during later years I have seen only one case of it." It has been shewn by orthodiagraphy, in diphtheria convalescents, that some dilatation persists long after all physical signs and symptoms of myocarditis have ceased to be perceptible, persists indeed until the patients pass out of observation.

This sketch, slight as it is, of the complex conditions of exercise may suffice at any rate to open our eyes to the thicket of difficulties in which lay and even medical counsellors become entangled when they undertake in journals and discussions to dictate about "strain of the heart."

Symptoms.—Under this head I may be brief, as many of the symptoms have been described or implied. They must be roughly divided between the stages I have discussed.

The symptoms of the first stage—the right-side engorgement or "Stauungs-Hochdruck" of Sahli—are not unlike those of mountain-sickness, and depend likewise on the delay in aeration of the blood. (*Vide* Art. "Mountain-Sickness," Vol. III. p. 243). Dr. L. Hill, as we have seen, states that lividity of the face is visible in all combatants in a severe game or sport; that is, even in trained men when the most intimate re-adjustments have come about, and no complaints are made. The capacious reservoirs on the venous side of the chest provide a large margin of toleration in the vigorous adult, but youngsters feel the instant pressure on that side acutely, if transiently. Their ribs are soft, and their thoracic muscles weak. But very soon the right ventricle pulls itself together, the systemic periphery opens, and second wind is established. This process, trying enough to an unsound or defective heart, to young boys, and to elderly men, is to the healthy heart of comparatively young adults perhaps never injurious; I have many times seen undergraduates and others, at the end of a long spurt of hard exercise, look ghastly; but I never saw a sound young man the worse for a temporary stress of this kind: if, as in a few cases which I have seen again and again in growing youths, dilatation of the right heart occurs, leading to cyanosis, panting, confusion, vertigo, or even vomiting, the oppression is generally sufficient of itself to stop the exercise in time. Even in children, whose frames are immature, how rarely is the brief but violent strife of whooping-cough attended with any ill consequences to the heart. A little boy—I try to speak always from cases—is injudiciously allowed to run with older companions; he turns blue in the face, drops exhausted, and feels sick, vomits perhaps; and then, having by two or three minutes' recumbency freed his circulation a little, jumps up again, plucky little chap that he is, and pounds along, to tumble over again in like manner.

> And often, ere the chase was done,
> He reeled, and was stone-blind.
>
> WORDSWORTH,
> "Simon Lee, the Old Huntsman."

So, a little late, at home he arrives; and strangely enough on the next day is none or not much the worse for his performance. Unwise as it is to allow such liberties, yet I have never seen very grave effects from them. The limits of elasticity in these lads are prodigious.

But as the boy grows up such liberties are less and less permissible. If he seriously and time after time overdrives himself, he will enter into the second stage—that of fatigue and exhaustion, remediable indeed, but which in the worse cases, especially in youths previously weakened, may merge into "strain." In many of these patients, as in Mr. T. (p. 196), it is impossible to say what may be the end of it. At the time of the fatigue he is prostrate, blue, and cold. The face bears an anxious expression, a weary ache—never, as Pawinski observes, a violent or anginiform pain—occupies the submammary region, and the respiration is quickened and shallow. The pulse is rapid, small, and thready, and I think undoubtedly in some cases arrhythmic, but with an arrhythmia of extra-systoles and grouped beats; though in some cases, and these of temporary severity, it may take the form of pulsus alternans and other signs of inadequacy. In this opinion Sahli, Janowski, and other trustworthy clinicians agree. In one case of this kind Dr. Michell noted a pulsus alternans, quite definite to the finger as — — — ∪ — — —˙ and so on. In an unbalanced state of the ventricles, and of the coefficients previously considered, this is easy to understand, for the residual blood in the left ventricle varies greatly with influx, diameter, and vasomotor instability. Blake and Larrabee describe in long runners, at the end of the race, a "moderate irregularity," which they add is "not rare." A patient and friend of mine, whose constitution had been shaken by haemorrhages in early adult life, took to the bicycle in middle age, and often rode hard and far. He complained to me that at times he had felt some discomfort from it. On careful examination, however, I found no sign of disorder; but I asked him to end his next hard ride at my house. I then found his heart as he dismounted, both irregular and intermittent, the arterial pressure low, and the right ventricle dilated. Fortunately he was soon well again, but repentant. In the acutest cases oedema of the bases of the lungs may be found on the following day; and if so the affair is not one of an hour or two. The patient, if young and vigorous, often recovers in a few days; but it may be a much more tedious business. On the next day he may be languid, drowsy or fretful, and without appetite; he slept badly, and complains of dyspepsia and flatulence. The stomach will be found distended in atony, which gives a metallic ring to the heart's sounds. The temperature may be raised a little in the mouth, certainly in the rectum. The heart is accelerated, and now, or a day or two later, is irritable and thumping, yet with a very small soft, generally very dicrotic, pulse. The dicroty is not a bad sign; it is better than a monocrotic wave of very low elevation. Other evidences of atony are found in the labile state of the vasomotor system, of which the "dermatography" is a ready test. There is some repletion of the splanchnic area, but not such as to cause faintness of the degree mentioned on p. 223. For

many a day or many a week afterwards, however, the pulse will run up as the patient rises from the chair, perhaps by 30-40 beats. In a patient in the convalescent stage sent to me by Dr. Finny, this rise was pretty consistently from 80 to 120.

It is generally by the subsequent history that the extremer cases of this stage can be discriminated from the third; namely, that of strain. Some observers, however, such as Dr. Lambert, think that a strain is marked at the time, by a sudden sensation of pain or faintness. This was the case with a medical patient, who told me that at the moment of strain he felt "a sudden faintness at his heart." And it is true that such is often the case; still the fate of others, in whom no such history was given, has by the graver disablement convinced the physician that the stress had gone beyond fatigue and atony, and had set up a molecular lesion. Or a history of infection, or the patient's age, may give the clue. If strain there be, however, the prognosis is not necessarily of the gloomiest ; such cases, after long intervals of disability, often end in virtual recovery, but probably never result in a restoration such as to put the man back, in respect of muscular exertion, where he was. Even after a fatigue breakdown, and far more of course after a strain, a state of nervousness, tiredness, and incapacity for any kind of work may persist.

For strain elaborate methods of cardiac therapeutics may be needed ; but to institute such methods after a mere fatigue breakdown (stage 2) is injudicious ; an introspective anxiety may get hold of the patient or at least costly, inconvenient, and unnecessary systems and thraldoms are imposed upon a patient who would do far better without these specific machineries. Dr. Morison has uttered a grave warning against the irrational exaggerations of the slighter symptoms of passing cardiac disorders or fatigue which have been fashionable of late, and of the no less exaggerated methods of treatment. In cases of doubt, borderland cases, careful attention to the age, history of infection, and character of the patient, and to the general run of his other functions, with an appreciation as exact as may be of the physical signs and clinical measurements, will after a while dictate to us the line to take with him (*vide* Dr. Michell's contribution, p. 202). In the severer cases we shall have only too much time for this discrimination ; for a case of severe fatigue may hang about for many months, and baffle all attempts at more than the mildest sports. The young man, or boy or girl, is fagged on slight effort, is short of breath, and with lost energy lies languidly inert. Dr. Eyre (of Claremont, Cape Colony) discussed such a case with me, which I refer to because in her the story was not one of a single determining stress but a long record of excessive feats, gymnasium shows, and other incessant games encouraged by foolish parents. This case was not one of heart-strain, but of utter fag. Of heart-strain the following is an example : —It followed (genuine) influenza, and was due to strong effort. An apex systolic murmur, to be mentioned presently, was audible. A long rest and specific cardiac treatment were required, and even then the heart remained very unstable. Palpitation and dyspnoea on slight exertion

were continually vexatious ; and once after a break-out into some game an acute attack of dilatation set in. At last he "got all right again," and went into the army, but within eighteen months was invalided out for "weak heart." This was an exemplary case of positive strain in an infected myocardium. The disablement of strain, if it seems to pass off, or even does pass off, does so hardly and slowly ; especially in men past their youth. The pulse long remains irregular and feeble, and the breathing embarrassed by slight effort. There is probably a large quantity of residual blood in both ventricles for a longer or shorter period. If the signs of dilatation persist on the left side, arterial pressures run low, and the mitral orifice may yield. A patient so strained may, indeed, return to the duties of a tranquil existence for some years ; or he may remain languid and pallid, unfit for much physical exercise, and in all the work of life soon wearied into fretfulness and depression of spirits. In the next stage of the disease albumin appears in the urine, and oedema about the legs and feet ; though even then the end may not be imminent. However, for these phases of the matter the reader is referred to the section on diseases of the myocardium (p. 105).

Chronic Strain.—When from young and healthy men we turn to persons no longer young, or, if young, injured by some infection, we turn from manageable, or comparatively manageable, disorders to diseases which may have the gravest issues. Dr. Mitchell Bruce's observations on chronic strain are very interesting. He discusses 40 cases. "It is unwise, ill-timed, ill-planned muscular exertion which does the harm." He comments upon the ill-effects of suddenly closing a period of great bodily activity, then entering upon a sedentary and self-indulgent life in a city, and from time to time in vacations dashing back into violent exercise again. He found in many such cases of chronic strain in later life that gouty disorder had some part in weakening the heart; and in other cases noted the baneful influence of syphilis, influenza and other specific fevers, tobacco, and so forth. I have quoted Dr. Lambert's experience on the incidence of influenza on the Harrow boys, and it stands in accordance with my own, and with that of all physicians, I believe, who have studied this chapter of disease. In the case of Mr. T. (p. 196) a neglected cold made a case of cardiac fatigue into a serious illness, though happily one which had a favourable issue. But, after infections apparently no more malignant, hard exercise has done more than this ; it has effected more than a cardiac fatigue with temporary atony ; by stretching a deteriorated muscle beyond its limits of elasticity it has damaged it, perhaps beyond recovery. The after-strain may be slight, or it may be severe and irreparable. It is not necessary to enumerate such instances ; it will suffice to point out that they are strain in the true sense of the word, not the mixed, indefinite, and far less serious cases which make the staple of current essays and congressional debates. Older men must remember that after thirty years of age their athletic exercises must be slower ; spurts which in the healthy youngster

rarely, perhaps never, damage the heart, are far more trying to his seniors. After the age of thirty-five then every man must tone down his exercises; he must enter for the slower sports—for the sports of the field, for golf, and so forth. And even these pursuits must be taken with reasonable moderation. Not infrequently the continuance of even these exercises laboriously and incessantly, if it do not imperil the heart immediately, which I admit it rarely does, is fraught with other evils, with risks of dissipations of nutrition and bodily reserves which are prone to end in tuberculosis, arteriosclerosis, or other remoter events which have no place in these paragraphs.

Happily, as I have said, even in older men, strain of the healthy heart is not frequent; they obey the hints of nature, and are content with less exacting stresses; but when strain does happen the results are often such that a year or two of treatment fails to remove them even so far as a virtual convalescence. The muscle, whose limits of elasticity have been insensibly contracting, is stretched beyond them, and its intimate structure has got a new set, and one either more awry or less susceptible of creeping back than after an infectious fever in a boy, in whom the fountains of youth are still springing. Prolonged stresses which are not good for boys, and in young men are tolerable only during a short adolescence of highest efficiency, to the middle-aged are murderous. Such a disablement was recorded lately in the medical journals of a man of the age of forty-six, who took to a tricycle, and after a brief apprenticeship rode from Brighton to London; and on his arrival was faint and cyanosed. The pulse was 140; the cardiac dimensions also were largely extended in both directions. An old friend of mine, when about fifty years of age, thus strained his heart by hard walking in hot weather on the Italian side of the Pennine Alps. He broke down and came home, when we found the dulness of the heart much extended transversely, and other signs of strain. The pulse was extremely irregular and intermittent, and these characters it never lost, though some fifteen years of a valetudinarian life remained to him before oedema and gradual encroachment of venous pressures ushered in the closing scenes of his life. Had I space to record them, I could give notes of many such cases of strained heart, especially in men who by years or by frailty were passing or past their prime.

The symptoms in the more chronic strain of elderly men are well described by Dr. Mitchell Bruce. We often find a large heart with feeble diffused impulse, and small and feeble muscular sounds but a ringing aortic closure. The complaint is of precordial oppression, palpitation, and sense of irregular rhythm. But, as Dr. Mitchell Bruce plainly says, in many of such cases there have been other and often even more potent causes at work than the muscular effort.

Again how suddenly and irreparably an elderly man may strain his heart is illustrated by the following case which I saw with Messrs. Hartley and Agnew of Bishop's Stortford in 1904. A burly man, aet. 52, athletic from his youth, and of impulsive temperament. Very temperate and under no

infection.   Insulted by a drunken workman, after a smart wrestle he
" chucked the man out of the office."   A few minutes later, on returning
to his desk, a severe aching set in about the lower chest, but never then
nor afterwards radiating to the arm.   It was a continuous ache, but so
acute that morphine had to be administered.   It did not cease for two days,
when I saw him.   While absolutely at rest he was then easy, but the
least exertion distressed him.   The pulse was at first 140, weak and
irregular ; on my visit and at rest, 120-130 ; it was more equable in
rhythm, but the artery was very ill filled.   There was a systolic murmur
at the apex, carried into the axillary and subscapular parts ; and coarse
crepitation at both bases.   I heard afterwards that he died in a few
months with the usual symptoms of gradual heart-failure.

Irritable heart, a state known to physicians in the long past, but made
more familiar to us by the descriptions of Da Costa and others in our
own day, is partially considered in the article on Functional Disease,
p. 519.   It seems to me that under this name two states are confused
which superficially resemble each other very closely, yet in kind are
radically different.   Superficially speaking, the more prominent symptoms
are palpitation, dyspnoea, and a sort of restless distress on muscular
exertion ; and the disablement in a severe case is so great that the patient's
active life is confined to the narrowest service.   In the article on
Functional Disease, these symptoms are described in so far as due only
to immaturity of bodily frame and development, temporary lassitude,
some psychical disorder, and so on ; that is, as " functional " and curable
cases.   In these patients the heart is fretful, probably ill endowed with
tone and reserve, but essentially sound.   But there are other cases, cases
which more properly belong to this article, which have been named the
" Soldier's Heart."   If the irritable heart of raw youth and of harassed
or irregular adult life is in its nature " functional " and curable, the
" irritable heart," dwelt upon more specially by Da Costa, Col. Myers, and
others, as notably frequent in soldiers, is a most obstinate and too often an
incurable malady which, however similar to the temporary disorder, must
on profounder analysis receive a different interpretation.   Unfortunately
we are almost without pathological data.   To take one instance out of
many : a friend of mine, of healthy body and inheritance, wholesome
habits and disposition, and still under middle age, in addition to some-
what arduous professional engagements, in a certain year cycled 13,000
miles.   This was some few years ago, and at the end of that term he
found himself, and no wonder, utterly exhausted.   His heart, which had
been vigorous, would fall into disorder on the slightest bodily effort.
After a long indisposition he made an apparent recovery, but even now,
although within the ordinary sphere of a professional life he is well, and
feels well, yet he is still unable to walk up a steep hill or to play any
active game.   He is then pulled up at once by palpitation and dyspnoea.
These cases must be attributable to heart-strain in the proper use of the
word.   Not suddenly, perhaps ; yet by incessant encroachment the myo-
cardium, rendered already more or less atonic by common fatigues, fatigues

often concealed under the pleasures of an ardent temperament, may be deteriorated gradually under the accumulated effects of tensions pushed to the extreme limits of elasticity rather than by any single occasion of strain. In their very interesting paper on the "Physiology of Marching," Zuntz and Schumberg stated that as the weight of kit was increased beyond 31 kg. the heart in 87·5 per cent (89 men) of the soldiers on a long march was lengthened in systole and shortened in the pause (p. 202); the pulse and respiration rate also ran on a higher range, and the pulse was more dicrotic. In two-thirds of the men so affected physical signs of increased cardiac area, chiefly to the right, and even some distension of the liver, were detected. After a thirty or forty minutes' halt these symptoms usually subsided; but if we suppose such stresses to be repeated continually over many months it is scarcely too bold an assumption to infer that the fatigued myocardium may yield, and its resilience become impaired, only too often permanently. The number of soldiers invalided out of English and foreign services a few years ago, before weight of kit and form of equipment were readjusted, was notoriously large. Dr. Tyson saw a number of cases of this kind at Sandgate in men who had gone out to the South African war untrained.

One of our graduates, Capt. M'Carthy, took up this matter of soldier's heart in a thesis for his degree. He obtained his materials at Netley. After stating that the modern valise equipment is less injurious to the young soldier than the old knapsack, which by its cross belts constricted the chest, he adds that the malady is still common enough nevertheless. He was able in a short time to collect twenty cases, and also to examine the first batch of twenty soldiers invalided from a campaign on the Indian frontier; and of these five were found to be patients of this class, though they were not included in his series. In dealing with his twenty cases, Capt. M'Carthy noted in each the age, total service, the trade of the recruit before enlistment, the habits as to tobacco and alcohol, the climates of foreign service, and the infectious and other diseases which he might have undergone. Fourteen of the men were in infantry regiments, three in the Royal Artillery, two in the cavalry, one in the Royal Engineers. At the date of examination two were under the age of twenty-one: fourteen were between twenty-one and twenty-five; four were of twenty-five years and over. "Taking the statements of the men as true," the average amount of beer consumed daily was from three to four pints. Other alcoholic drinks were taken but occasionally. The average amount of tobacco was three to four ounces a week, the tobacco being generally twist or plug. Twelve had suffered from syphilis; fifteen from malarial and other tropical fevers; two only from rheumatism of any kind. Some of the men figured, of course, in more than one of these categories.

"The patients state that while not exerting themselves they feel quite well and free from any shortness of breath; but as soon as they begin to march they are troubled at once with a throbbing sensation in the chest; and with this there is difficulty of breathing, followed in some

cases by faintness or giddiness. Rest may relieve for a time, but in most cases all the trouble returns shortly after returning to duty."

To take the symptoms in detail :—Cardiac pain was present in 17 cases, dyspnoea in 17, giddiness in 6, sleeplessness in 5, nervousness in 7 cases. Three cases were noted in which the men were unaware that there was anything wrong with the heart.

As to physical signs :—In 14 cases the pulse was regular while the patient was at rest, though in some of these it became irregular after slight exertion ; in the remaining 6 it was irregular even when the men were confined to their beds. In 12 cases the pulse during rest was below 100 ; in 6 it was between 100 and 115 ; in 2 between 115 and 120. The pulse rarely exceeded 120 when the man was at rest, but would always rise very rapidly, even to 140 or so, on his swinging the arms three times round the head. In nearly all the cases the pulse seemed to be of abnormally low pressure.

The area of cardiac dulness was increased in 14 cases ; but in some of them the increase was so slight that it was recorded with hesitation. In all the cases the impulse was diffused, and in many the apex was displaced—in 2 cases between 1 and $1\frac{1}{2}$ inches outside the nippleline. Abnormalities of the cardiac sounds were uncommon. In 2 cases the second sound was reduplicated at the base ; in 5 the pulmonary second sound was accentuated ; in 6 the first sound was sharp ; in 3, prolonged and booming ; in 4 cases there was a systolic murmur at the apex.

Capt. M'Carthy remarked on the history of malarial fevers in many of these men ; and he spoke of the evil effect of fevers on the cardiac muscle, and urged that soldiers recovering from these fevers should be exempted from drills and other manual work for several weeks after discharge from hospital.

Alcohol was the next cause on which he laid stress. As to tobacco, he says that men smoke more in the tropics where they loaf more ; and that the tobacco is bad and strong. On campaign the rations also are often necessarily short, while the labours are excessive. Finally, the author urges that tropical heat reduces the value of haemoglobin in the corpuscles of the blood, and leads to anaemia. If in this condition the soldier is called upon to do hard muscular work, is badly fed, and mayhap attacked by some infection, the deteriorated heart-muscle yields, and the man is invalided.

On the other hand, many men (not in the above list) have the arrhythmia, yet state that it has never been of any inconvenience to them whatever. " In fact, many such cases of disordered heart have been detected by me quite by accident while going through the usual routine examination, when soldiers come into hospital for other complaints, especially malarial fevers."

The *prognosis* is not good ; in the majority of cases the patients return to hospital till they are invalided out of the service. The author found the difficulty which might be expected in tracing the men thus invalided. However, he obtained records of 30 cases of men discharged from the

Netley Hospital, and his impression from these returns was that in many cases the soldier's heart ends in valvular disease.

We have then to deal with two kinds of irritable heart, the functional and the organic; and upon this distinction our prognosis in the individual case must chiefly depend.

Physical Signs.—To the age-in which we live nothing is sacred; not even the methods of physical examination of the heart. Not only has the time-honoured "absolute dulness" been blown away, but all and any means of discovering the dimensions of the chambers of the heart, unless it be to guess roughly at the grossest, are in jeopardy of modern scepticism. The orthodiagraphists are now declaring that, whereas physicians have been relying upon tactile and percussive methods even for refinements of appreciation of the relative sizes of the several chambers, and in particular during the stress of effort, as a matter of fact all these calculations rest upon delusion. From their pictures they assure us that during effort, even severe effort, the heart, right and left side alike, grows not bigger but actually less! And it must be admitted that their assertions, if not beyond controversy, are at any rate very carefully recorded. De la Camp's essay is a model of careful observation and sober argument. . Dietlen and Moritz follow him as carefully; and essay after essay appears to assure us that the labouring heart suffers not dilatation, not hypertrophy, but a positive diminution. Right ventricle not distended; left ventricle diminished, is their verdict. What are we to think of ourselves, we who have been demonstrating to our pupils year in and year out how to detect half centimetres of increase to right or to left, and so on, when after all the heart we thought we were following was secretly moving perhaps in the very opposite direction! These disturbing criticisms are not yet directed altogether against what we may infer about the heart in disease; so far they discredit only our appreciations of the organ under physical stress; still if those of us who have been demonstrating enlargements of the right ventricle in over-exertion are deceived, the inferences in disease, which are often no larger and are calculated on the same procedure, must come under a like disapproval. It will probably turn out, however, that orthodiagraphy is no more infallible than the pleximeter; yet if this method and the recent and important observations on the various movements of the diaphragm (Wenckebach, Keith, Halls Dally) teach a little reserve to certain schools which have pretended to carry heart-mapping to impossible and even absurd refinements, it will be a useful discipline.

My opinion, for what it is worth, I have given—that orthodiagraphic pictures are too fugacious for standard or comparative purposes. Moreover, it is conventionally assumed that the heart is a uniform body, like a liver or kidney; whereas it is in incessant flux of form, not only between its pulses but also as incessantly adapting itself to the fluctuations of the tides of the active body. It has no constant diameter, not even for each position of pulse. Roy and Adami, in cardiometrical experiments on animals (*e.g.* by compression of the aorta), proved that the heart could

expand with impunity to three or four times its ordinary capacities, and at the end of systole be as large as it had been at the end of diastole. Bruck, who has studied the orthodiagraphic shadows carefully, agrees that they are too fleeting for exact interpretation; and he expresses his surprise at the great differences in them, even in healthy young men, under the same conditions. Deep inspiration; he finds, sets the heart up more vertically; then, as the effort proceeds, each diastole is less, even in the right heart, for a phase; then, as pressures alter, the heart takes its full average size, or in a number of cases ("in einer Anzahl von Fällen") the heart is distended beyond the mean, especially to the right; and this his colleagues verified. Thus in Valsalva's experiment, and in lifting weights, he often noted more or less "acute dilatation." But he admits that the experiments were very difficult, and that he had to discard a large number of his notes. According to the views I have already expressed, in such conditions the blood is piled up on the right side, and the left ventricle contracts either on a correspondingly less volume, or, if the arterial pressures be high, the residual blood is more. Von Criegern states that at first the auricles fill, and no more enlargement may take place; or the ventricles may distend and return to their normal mean; but if the stresses be higher, or the ventricles less vigorous, there is a general and persistent distension. And even Kraus and de la Camp admit that in the panting which follows the Valsalva effort the heart is distended to the right. Zuntz and Schumberg, no children in physical methods, in their soldiers emburdened with 31 kilos, found the right ventricle dilated ("March-dilatation of the right heart"). I may repeat that the Valsalva effort represents only the initial phase of ordinary athletic exertion. Kraft of Görbersdorf reports that the heart may diminish on effort, but enlarges twelve hours later. Dietlen and Moritz admit that in their observations when the heart shrank in size the blood-pressure had fallen ("Blutdruck gesunken war"), and the heart-rate was much accelerated. Schieffer's experimental records with orthodiagraphy are very interesting; and, so far as this method is to be trusted, seem to prove that the heart preserves a constant diameter in trained men. He has observed the apparent shrinking, but in defective training he noted chiefly a considerable enlargement ("eine erhebliche Grössenzunahme"). Romberg says wisely that by physical examination alone it may often be very difficult, in the variations of lungs, diaphragm, relation to thoracic wall, etc., to map out the heart's dimensions; yet notwithstanding some approximate and even definite information is to be had; both as regards distended and recovering phases, though such changes cannot be expressed in terms of efficiency. Moreover, he says orthodiagraphy in the hands of ordinary physicians is very misleading, and that even in skilled observers the personal equations are large. I may add that the ordinary cardiogram is quite as deceptive; what we shall get out of Einthoven's electro-cardiogram time will shew. It promises well. Otto Weiss of Königsberg has published a method for objectively representing and comparing cardiac tones and murmurs,

which it is impossible to describe in this place. Some curious points have come out already, and his results will be watched with much interest. Dr. Grödel of Nauheim is perfecting a kinematographic machine for these ray shadows, which is to make sharp pictures at the rate of 24 per second. We await these results also with interest. In continuous exercise it occurs to me that the loss of water may account for some shrinking of contraction-volume, with corresponding saving of cardiac work, though it could hardly become visible. The body-weight is known to fall in long runners by 4 to 6 lb. Trained men do not drink much during exertion.

It was necessary to give some sketch of these defects of our methods before any discussion of the data of physical examination could be possible. Now taking our methods as they are, can we decide if the prevalent opinion be true that the heart of the athlete is hypertrophied? During exertion it is doing much more work, of this there is no doubt; but does it do more work in the twenty-four hours? Dr. Michell's records shew that there are two sides to this question. If for no inconsiderable part of this period the pressures and rates range lower than in the untrained and unexercised man, may not the sum of work be little altered? Still it may be replied that as the heart is submitted to much heavier demands, it must have a larger reserve. That in a sound hypertrophy the reserve is not more, or is even less, is a gratuitous assertion founded solely on dubious experiences of diseased hearts. On the other hand, we agreed that tone and reserve may be two sides of one shield; so that given corresponding tone the bigger the heart (within the limits of the other parts) the more the reserve. There are many facts in favour of this view. There is the general law of Hirsch that the cardiac muscle stands in direct relation to the skeletal. Külbs, working idle dogs against similar dogs at hard work, and Grober and Bollinger, who dissected racehorses, greyhounds, and wild hares, and compared them with hutch animals, found strong evidence that in the active animal the heart is much more muscular. I had for many years a terrier which ran with my bicycle, with marvellous ease and fleetness, and died at a ripe age of no obvious disease. His heart, for which I had no standard, was, in the opinion of my laboratory colleagues, who knew my dog, and were well practised in dogs' hearts, very much larger than his size and weight would have indicated. At the Addenbrooke's Hospital, two or three years ago, I chanced on a necropsy in a healthy, muscular, and athletic young man who had been killed by accident. We were all struck with the muscularity of his heart; though I confess we did not take the measurements on Müller's method. Furthermore, we were struck with the muscular volume of the right ventricle, a point to which I will return. The valves, lungs, and kidneys, etc., were all healthy. In young men, this thickening is all good muscle, but in older men, as Dehio pointed out, and Stadler has carefully verified, it is partly compacted of connective fibre, whereby, I may repeat, the elastic resistance is increased, peradventure conservatively; but, as the elastic limits are much narrower, strain is more easily reached, and having

occurred is less likely to heal. I think that the predominance of clinical opinion among physicians who have been occupied with athletes (Zuntz, Collier, S. West, Michell, Blake and Larrabee, Brunton, Stengel, and others) is that in them the heart is hypertrophied ; an opinion founded not merely on the quicksand of percussion, but on palpation, the kind of lift to the finger, the quality of the sounds, and on a few post-mortem records. It is not likely that any such moderate degree of increase should reveal itself to percussion, even if it were less modified by variables. Hensen rather forcibly argues that as the systolic pressure on exertion never exceeds 200 mm. at most, for with even less than this a healthy periphery would open out to moderate it (in Bright's disease the heart may contend, and for a time successfully, with systolic pressures approaching 300, and this for the twenty-four hours' round), an increase of contraction-volume would not exceed 60 c.c., and this would mean only an increased diameter of 2 cm., for the detection of which, it must be admitted, delicate methods would be required. By increase of rate, oxygenation can be doubled without increase of heart-volume, and trebled without overcharging the heart. Let us suppose in effort the oxygenations to be quintupled, and the output doubled, this would amount to about 1 cm. increase in diameter ; *i.e.* only $12\frac{1}{2}$ per cent.

Some of the observers quoted mistrust this hypertrophy as a morbid indication ; if so, it must be lest it lead in later years to high-pressure lesions in the valves, aorta, and coronaries ; a fear not unfounded, but one would hope not often realised. I must confess, however, that it is difficult to avoid the opinion that the continuance of incessant and competitive exercises, after say the age of thirty, may bring about such ultimate deteriorations, and in some cases may have issued in cardiac impairment ("strain") in early middle life (40-45). But the incidental causes of cardiac deterioration are so many, that no positive opinions in this matter can be pronounced (*vide* p. 430).

Once more, many of the observers I have named, and others also, have noted the enlargement, if any, to be on the right side ; or on both sides. In the necropsy referred to on p. 240, the right ventricle, in the opinion of all present, was at least as much hypertrophied as the left. Dr. W. Collier notes right-side enlargement. The general interpretation is that this right-sided increase is of the nature of yield ; a dilatation, not a hypertrophy. I will not repeat what I have said concerning large right-sided dilatations under heavy stress, but would rather draw attention to static increases towards the right, with lifting beat in the epigastrium, and, so far as we may rely on it, some percussion shift to the right. The most thorough study of this point I find in an essay by Grober, who examined hearts of animals by the precise method of Müller, and found, "to his surprise," the hypertrophy, after long periods of exertion, to be chiefly in the right ventricle ; as if the high pressures fell more on the pulmonary circuit. It is unnecessary for me to repeat the arguments by which I have always been disposed to support this conclusion ; but here I may add that my own examinations testify that in active rowing and football

men the deep dulness frequently transgresses the right sternal line, and there is a little heaving in the epigastrium. I have accepted such men for life insurance.

The phenomena of auscultation are so fully dealt with in other articles and in Dr. Michell's paragraphs, that I must not attempt to discuss more than a few special points. In estimating the state of the ventricles as much weight at least must be given to the quality and balance of the sounds as to percussion. My own attention for many years has been given to the duplex character of the systole at the apex, which is noted also by Dr. Michell (p. 201). In debilitated states this compound first sound soon becomes single, and shorter and sharper. Perhaps I am without other testimony to support me when I add that in some cases a so-called reduplication of the first sound consists in a division of the duplex tone; and farther, that such a "reduplication" is often an incipient murmur—a sign of relaxing mitral. At one of our M.B. examinations I had a friendly argument with my colleagues on such a case; they declared for "reduplication," while I asserted that the first element was a murmur. I lost sight of that case; but I have often heard this alleged "reduplication," as things grew worse, develop into a murmur; while on the other hand, as things grew better, the murmur would dwindle back into the "reduplication," and then disappear.

This brings me to the systolic murmurs heard in the hearts of over-wrought athletes. Authors on this subject are not careful always to separate, and to dismiss as of no practical importance, the systolic murmur at the base which depends, in some persons, on dilatation of the pulmonary artery with a shift against the chest-wall; in others on a permanent state of approximation to it, so that the murmur, if not persistent, at any rate may be easily brought out by a slight exertion. If it appears in a young man who was certainly known to have been without it, it must be watched, with other points, in the behaviour of the right side, and of the whole heart; but when habitual to the person it is usually negligible. I should not have thought this point—often emphasised by Broadbent and others—worth mentioning here, had not a gentleman been sent to me in 1909 in whom this murmur had twice stopped a life insurance, and led to a serious restriction of useful and wholesome activity. A later medical adviser, being properly sceptical as to these adverse opinions, sent him to me. The murmur during rest was sometimes absent, sometimes very faintly audible. It was wholly under the influence of the respiration, and under forced expiration roared loudly enough, but we made light of it, whatever its roaring. This murmur at the left base is not a feature peculiar to "athletes"; it is not very uncommon, at any rate on expiratory effort, in ordinary men. In a particular case the diagnosis will lie between a somewhat eccentric aortic systolic murmur, and pulmonary stenosis, probably congenital : no very insoluble problem. A murmur in this area determined by respiration would suggest either functional disorder or a harmless peculiarity of conformation. Of far graver import, or at any rate of graver warning, are the systolic murmurs heard at the apex.

Now in this article we stop short of static disease; but it is well known that a murmur at the apex, not to be distinguished from that of mitral regurgitation, may become audible in over-stress. Does such a murmur signify this regurgitation? and if so, does it indicate static disease? For my own part, I have no doubt that such a murmur may signify actual regurgitation; and accordingly I think at present that it has such a meaning in all cases. Both Dr. Michell and I, in cases before us, have traced such a murmur into the axillary region, and behind under the scapula; and this not once only but many a time. For instance, T. A. B. C., aet. twenty, I saw with Dr. Michell in December 1903. He had had cardiac fatigue at various dates, at school, etc., but got right again, so far as he knew; footballed, etc. On this breakdown we found the heart well out to the right and to the left. The beat did not suggest hypertrophy; it was short and sharp and thin. There was some arrhythmia, and a short murmur was heard in all the conductions of a mitral murmur. The gastric pouch was largely distended (general atony). No increase of the right auricle was apparent; nor were veins obvious in the neck. Dr. Michell said the murmur, when he was at his worst, was longer. A year later the heart had receded to normal size, and all symptoms were improved; but before the murmur ceased it passed through the period of apparent re-duplication, of which I have just spoken, the sound which I have called the larval mitral murmur. Mitral murmurs in over-stressed hearts are recorded by too many observers to be very rare. Stengel, out of nine hard football players, found it in three. It is often transient, and only to be found if sought for at the moment of cessation of effort. It persisted till recovery in Dr. Finny's case (p. 232); then no exercises would bring it back. In too many papers the precise distributions of the systolic murmur are not described. Blake and Larrabee, in nine "Marathon" runners, found in three a systolic murmur, which varied with the breathing; and in three more one which was apical, suggestive of mitral insufficiency. Williams found no murmur in two winners whom he examined, but found one in all the rest who trailed in. Dr. Williamson in "Marathon" runners noted a similar experience. In my Bishop's Stortford case (p. 234) a systolic murmur denoted a permanent and ultimately fatal mitral lesion. Dr. Lambert in his schoolboys detected a mitral murmur in only one, and he never recovered completely. Some physicians believe that a murmur of mitral relaxation may appear transiently during vagus inhibition. In the curable forms—and in by far the majority of cases it passes away after a longer or shorter period of disability, or even without any ill symptoms at all—I would refer it to a temporary atony of a sphincter part of the mitral machinery. I make this obvious remark because I observe this condition is called again into question. Kraus and de la Camp, and others, however, still accept this interpretation. Dr. West hesitates to admit that the apex murmur is one of mitral insufficiency, as it is not carried into the appropriate areas; he attributes it to intra-ventricular conditions. Often it is not so carried, it is true, but often again it is audible, or becomes audible towards or in the axilla, or appears

in the subscapular area also. No sharp line can be drawn between these cases, often indeed various stages of the same case. An intra-ventricular origin would be explainable, if at all, on Roy and Rolleston's hypothesis of irregular action of the musculi papillares, whose rhythm may fall out of concert, and so over-ride a cardiac contraction, or even drop out for a beat. Records of the jugular pulse immediately after severe exertion are much wanted.

Albuminuria.—As experience of this symptom in young men, and especially in athletic young men, increases, less and less importance attaches to it. The mere quantity of albumin is no great matter ; it is often abundant. A few hyaline casts are not of serious importance. After severe exercise it is said that even a few granular casts are to be found ; though I think they should give cause for suspicion and pre-caution. Dr. Michell and I, after much consideration, allowed a fine oar in excellent apparent health to row in the University boat, in spite of large quantities of albumin. We did not feel much fear of injury to himself, but feared lest he should disappoint the crew in his training ; he proved, however, as good a man as any of his comrades. I need not say that, before albumin is disregarded, the patient must be closely examined in all respects. If the heart be at all enlarged by exercise, some further vigilance may be necessary. Individual differences in this direction are as great as in respect of other functions ; but if the urine be tested immediately after a considerable effort, albumin, often in large quantity, will be found in many if not in most persons. In "fatigue cases" I regard it somewhat differently ; namely, as an evidence of "auto-toxicosis." I ought to add that many physicians, whose opinion is of weight, take a more serious view of albuminuria, and suspend the severer exercises so long as it lasts ; they admit, however, as probable, what I am sure is true, that in many men it passes undiscovered. Blake and Larrabee in the 24-mile runners found albumin after the race invariably.

Diagnosis and **Prognosis** may conveniently be taken together. In the first place we have to decide if the particular case be one of overwrought heart or of some other cardiac disorder ; and again, if more than one cause be at work. In a girl, nervousness or anaemia may be the chief evil ; or excess of tea-drinking. Then there are the lanky, long-chested lads with weak thoracic muscles and short antero-posterior diameter and wide intercostal spaces ; these youths often smoke precociously, or fall into secret vice. In such persons the heart often appears to us, and to them feels, too big and irritable. Stokes drew attention to such cases. A survey must, of course, be made of all other conditions of body, for even if there be some over-stress of the heart, a revision of other bodily or mental habits or vices may be a part of the cure. When we are satisfied on the incidental conditions of the case, and are still of opinion that the heart has been overwrought, we shall regard the age of the patient, the possibility of some previous or intercurrent infection, even of a bad cold, of overgrowth for age, of slenderness of build, and so forth. Thus we

shall come closer to an appreciation of the heart itself. And we shall determine, as well as we can, if the ailment complained of consist in a rise of pressures on the venous side, at the gate of the lungs, with cyanosis and more or less of mountain-sickness of a passing kind ; or in cardiac fatigue and atony affecting the whole of the organ, even to the degree of mitral insufficiency ; or finally, if really some strain has taken place. This, however, in the absence of infection, and in young men, we shall regard as least probable. Besides the examination of the heart, we shall inquire for any loss of appetite, sleeplessness, unreadiness for intellectual occupation, dyspnoea on slight effort, abnormal rise in pulse-rate on rising from the chair, and so forth. The difference between the pulses in the vertical and horizontal position is ordinarily about 15 ; and a reduction towards some such variation from twice or thrice the amount is a hopeful sign.

If we decide that giddiness and lividity, if any, are due only to a temporary overcharging of the very distensible reservoirs on the pulmonary side, and that the symptoms are only those of mal-aeration, and not specifically of cardiac failure, we shall give a very favourable prognosis ; but tell the patient not to do it again. Boys should be carefully graded in the more prolonged games or sports. If the case be one of fatigue with atony in a young person the prognosis is not so favourable, though still good ; if the degree of the fatigue be not excessive, and the general condition of the patient sound, recovery without elaborate methods of treatment or long inaction may be promised. If the case be of an older man, or one whose heart may have been deteriorated by some infection, the case may be one of strain ; the fibre of the heart, already weakened, may have been stretched beyond the limits of its elasticity. The prognosis is then grave; in the worst cases life may be imperilled; certainly recovery or substantial amendment will be long in coming. The majority of such patients usually recover so far as to take up again the ordinary activities of a tranquil life ; others, although they survive, never regain a capable heart, and have meticulously to pursue a sedentary occupation.

Treatment.—We cannot hope to treat the cases discussed in this article efficiently so long as our notions of them remain confused and indiscriminate. As long as the name " heart-strain " is used loosely, so long will therapeutical means be as loosely considered and applied. Yet for no cases are discretion and sagacity more important. To lay a boy or young man aside from the useful and cheerful current of his education and his sports is, when a necessity, a harassing and unhappy necessity, both for life and character. If we are compelled to turn a cheery manly boy into the twilight of wistfulness and introspection, perhaps into morbid whims and insidious decadences, we must be sure of the necessity, and clear about the economy of our methods and the accuracy of our aim. Notwithstanding, as an old student of this subject, and one, therefore, whose experience is long and should be mature, I may be forgiven if I argue again that in too many of these cases we act with confused ideas, wasteful methods, and indefinite aims. I have endeavoured to shew that "heart-

strain" in boys and young men, so far from being a frequent event, is infrequent, and apart from some forerunning infection is indeed rare; yet in social, and even in medical gossip, we are hearing of it continually. Now for this blundering I do not hold the family physician nor even the consultant so responsible as the foolish parent. It seems nowadays to be customary for the layman, frequently for the mother, perhaps at a tea-table council, to make the diagnosis; whereupon the patient is carried off to the appropriate specialist. Now the specialist, spinning round his inner circle, is little perturbed by ideas outside it, and perchance some new system or a spa has come into fashion; and as the youth has some sort of cardiac disturbance, the specialism and the system are as indispensable to him as the patient is to them.

If, on the contrary, by an extraordinary effort the healthy myocardium, or some chamber of it, or some tract in a chamber, be strained; or if by some less searching exertion this consequence ensue in a heart previously enfeebled, or in the lapse of years reduced in its elastic limits, we have a very grave state of things to face, and one which, whether in youth or age, must involve painful sacrifices of time and activity. Unless the strain be catastrophic—a cardiac landslide, so to speak—by gradual re-accommodation of function and therapeutical economy the heart's labours may be so mitigated as to reduce pressure and output to the lowest practicable levels, and thus to give the over-stretched fibres time to creep back, as far as may be, towards their primary molecular constitution. Such a creeping back no doubt does occur; but how far the muscle will return towards the normal in particular fibres, or how comprehensively in a group of strained fibres, we cannot know until in the individual time and experience give us the information. And even then we can discover it only tentatively, and in the course of certain disappointments. So grave are the issues dependent upon such tentatives, so enhanced is the price of recovery by their failures, that the boldest physician will choose to err on the side of tardiness and indecision. The swiftness and decision of his judgment must appear not in this state of affairs, but in his primary diagnosis, when he has to lay down his plans on a comprehensive view of the relations of the various elements of the individual problem; if strain, how much, and how far complicated by the more transient conditions of fatigue and atony?—nice discriminations on which we have already dwelt sufficiently. If there be any evidence of a previous infection or ill-health, if the patient be over the age of thirty-five, if the stress was very excessive, or if he be over forty-five and the effort relatively considerable, the patient must be regarded for some weeks as suspect, until these conditions can be disentangled. If on these observations it be decided that strain is really a component part of the illness, every arrangement for physical, and as far as possible for mental tranquillity, must be thought out. A poor man may go into hospital; a well-to-do man may make his hospital at home. The patient, for the first weeks or months, at any rate, must never be without an attendant. A patient left to himself, and as yet unaccustomed to meticulous rules, will surely

by some impulse, some incautious foraging in his chamber, occupation in toilette, or fidget in business, hinder or set back his amendment. Moreover, skilful massage, at first very gentle, wholly passive, and never vigorous or fatiguing, is very useful, almost from the beginning. The diet, occupations, and amusements thus systematically thought out must be written down distinctly, and upon those proper lines which will be found in other articles of this work, on heart diseases or aneurysm; and so long as there is any reason to predicate strain, they must be no laxer than in those other conditions. In cases of strain with fatigue there is very often atony of the stomach also; the viscus is windy, and the digestion slow. If this be the case, the complication must be provided for in the diet, which must be simple in quality, moderate in quantity, and digestible in form (*vide* Vol. III. p. 549).

For the strain itself, although many drugs will prove to be incidentally useful, I am not satisfied that any has a specific value. An associated vasomotor atony may seem to call for ergot, strychnine, and suchlike; but it is generally wise to refrain from mixing our poisons in the dark; such vascular laxity indeed, for all we know, may be a relief to a halting heart. To administer digitalis, again, in the acuter stages, may be likewise a stab in the dark, and however discreetly used may do more harm than good. Drug treatment, in my opinion, therefore, is to be confined to such agents as may be useful in trimming the bodily machinery, that it may run so sweetly as to give the heart the least possible trouble. In convalescence more specific medicines, such even as digitalis in cardiac atony, may be tentatively introduced as may seem desirable to the physician in regular attendance, who knows pretty exactly what the behaviour of the heart is and has been. Dr. Michell's use of diuretin has been noted on p. 205. It is very important to keep the peripheral circulation active, for which purpose, besides the massage, warm baths of not more than 100° F. may be used, and the body protected against chills.

Unfortunately, as we have seen, after strain in persons in middle or later life the cardiac functions, do what we may, are never wholly restored; and the rest of the life is hampered, crippled, or even cut short. In such cases I have not found, nor as an onlooker observed, any advantage in Nauheim or Oertel methods, unless perhaps in the convalescence of those favourable cases which do fairly well under almost any competent medical management. In these they will be undesirable until they become unnecessary. On the other hand, more than once in strain cases I have seen harm from them.

Thus it is, then, concerning strain. Our treatment must be vigilant, sagacious, opportunist, empirical, rather than systematic, elaborate, or specific. Nature, time, and economy will be our chief medicines.

To turn, in the next place, to cardiac fatigue and cardiac atony, we have seen how difficult it may be to distribute to each of these allied factors, or to general bodily weariness, their several parts in the particular case. Yet precision of treatment depends upon some such

computations. The acute damming back of venous blood in the enormously distensible vascular approaches to the lungs, alarming as the cyanosis, panting, and prostration may be, is rarely, nay—taken alone and in the healthy young heart—is never dangerous.   The effects pass off in a few hours, or at worst within three or four days.   The patient, if the stress be extreme, falls, and in falling does something for his own recovery.   If oxygen be at hand—and it should be at hand during the greater public sports—it will be very helpful.   But after being pulled up in this way, a young and elastic man only too often, and I must admit usually with impunity, is very soon found at his games again.   Still this is foolish ; there may have been more than the crowding up of the blood ; there may be an element of cardiac fatigue also, which should have and will demand the more time and skill for its relief as the claim is for a while ignored.

If cardiac fatigue be added to right-side distension, this slackness will be perceptible on the left side also (p. 203), in which case the patient must be invalided.   Only too often where no medical precautions have been taken, and the patient has obeyed his own impulse to rush again into sports, he finds himself again and again incapacitated, and may in the end have a very heavy reckoning to pay.   A heart thus bullied is subjected to fatigue poisons, and to increasing residual blood in the slackened ventricles, until strain, in the proper sense of the word, may be set up, and the more tedious treatment we have just discussed cannot be evaded.   But at present we are considering something short of this.   If with right-side distension, neuro-muscular weariness, and general and cardiac atony our task is, comparatively speaking, easy, with cardiac fatigue it is more difficult ; though far indeed from the difficulty of strain. It is, I think, for the element of cardiac fatigue, as, of course, for general fatigue, that Dr. Michell has found diuretin useful.   The method of treatment, however, will be after the manner of that of strain ; but shorter in duration and less rigorous.   In a young person free from infection, progress should be satisfactory, and recovery apparent in a few weeks—I say apparent, because, after cardiac fatigue, which is a more specific effect than mere general fatigue in which the heart partakes only in the degree of all other functions, the disablement is more intimate.   A week or two will suffice for discrimination, when the plans for a brief, or a somewhat protracted, "lay-up" can be prescribed.   In mere neuro-muscular fatigue exercise may soon be pursued again, say in four or six weeks ; but—unless the occasion of failure were obviously an accidental indiscretion or stress—the patient's natural capacity and course of training will have to be carefully reconsidered.   In cardiac fatigue we shall beware lest the case fall into the category of "irritable heart," signified by fretful action and ineffectual pulse.   Some of these cases are definitely due to cardiac strain; others to vicious habits, as excesses in alcohol and tobacco ; still "irritable heart" may follow cardiac fatigue (vide p. 235).   For such tumultuous phases the cautious and temporary prescription of aconite during absolute recumbency is useful.   For more

continuous use, cactus or the tincture of *Prunus virginiana* has seemed to me to be serviceable. In case of nervousness or sleeplessness, the bromide of strontium may be used regularly for a few days, or in single doses. Occasionally a mild hypnotic may be required. Tobacco must be absolutely interdicted in all these cases, and tea and coffee used very cautiously. But, as such palpitations may be of eccentric origin, and depend upon the dyspepsia, or upon the tedium and vexation of enforced inaction, it is more legitimate and less hazardous to make attempts—say in one month or two, as the case may be—to test the powers of recovery. It is in these cases that the methods of Ling, under a competent attendant, may be very useful. Baths also, even those which by lower temperature or higher saline content constrict the peripheral vessels, will, if the languid heart can respond to the call, be beneficial by re-educating vasomotor tone ; and from these means the convalescent may cautiously proceed to the douche, and the rest of the hydropathic apparatus. Electricity in all forms does more harm than good, unless the inframammary aching and tenderness persist, when a gentle stroking with the faradic brush is often efficacious.

If general atony be a predominant feature, as betrayed by gastric distension, and by low vasomotor tone as evidenced by flushings, dermatography, etc., the above measures may be supplemented by such tonic drugs as iron, strychnine, arsenic, bitters, as the indications may be. Strychnine should be given thrice a day on alternate days.

In all these cases alcohol must be excluded, or practically excluded ; a few spoonfuls of sound claret diluted with water may be permitted to the weakness of the flesh ; but in case of low vasomotor tone even this little concession had better be withheld. Lager beer, harmless enough in health, is for the invalid too flatulent and copious ; and may perhaps compete with the toxic substances for the paths of excretion. For the same reason milk is to be preferred to meat extracts, and other foods alleged to be rich in purines. In all cases alterative doses of mercury should be given occasionally, and fractional doses of podophyllin or euonymin more regularly.

After heart-strain, unless in comparatively mild cases in young men or boys, severe bodily exercise is at an end, probably for life. After cardiac fatigue the resumption of any such exercises must be deferred until all ordinary tests have proved satisfactory ; and in no case should competitive exercises be thought of for twelve months at least. But after a period of mere neuro-muscular exhaustion, with nothing worse than temporary cardiac atony, recovery may be rapid ; though even then the patient must be dissuaded from the more active games until the whole question of his capacity, natural and trained, can be reconsidered, and some broad rules of common sense laid down for his guidance, at any rate till the accident becomes quite a thing of the past. But for really sound, capable, and well-trained young men, who have been no more than over-tired and fagged, even common sense in such matters may become a nuisance if it damps the spirit of manliness and courage. For this

reason I would deprecate too much encroachment by the physician on the freedom of games and sports. Every boy, on admission to a school, should be put through a thorough physical examination by the physician responsible to the school. No certificates or outside opinions should be accepted unless, of course, in an advisory sense. But even in this entrance examination the boy's attention should not be drawn to his heart, but rather to his bearing and chest capacity ; and the whole examination should be, and appear to be, of an anthropological and not of a grandmotherly kind. If it be repeated at a later date this aspect of it should be preserved ; the boy should not think that it is repeated with any idea of restricting his energies. Yet it is very important that boys should be " graded " for their sports, I think by age rather than by measurements ; though, of course, both conditions must be considered. And the games also should be graded, especially in duration ; shorter bursts for the younger boys, longer endurance for the older. Switzerland is too big for schoolboys. Between the ages of fifteen and twenty years a great gulf is fixed. If, as too often in small schools, a plucky little boy be allowed to compete with his elders, he will run till he drops rather than be left behind. Now, although I have deprecated much physical examining of boys, yet a physician of a school should have no more boys to watch than he can know well by sight, as a shepherd his sheep ; so that if he sees a boy looking slack—as a boy soon does—or anaemic, or loitering, he will quietly take note of him, and try to find out quietly about his appetite, his hours of sleep, his work in class, and so forth, and act accordingly. He will also learn what he can of his moral bent, lest the boy be one that the school would be better without. If, on the other hand, the boy is overworked, either in school or in the playground, or is out of health, the physician will be able to act in time. Above all things, if there be a run of any epidemic in the school, even if it be but an epidemic catarrh, he should see that no boy shall engage in the severer games till he is thoroughly well again.

On the practice of training, so continually mentioned or implied in this article, and on the diet, clothing, and other conditions of bodily activity in health, the reader must be referred to special treatises. Training cannot be regarded as efficient which is taken up only in a desultory kind of way. On the other hand, harm is done by piling on additional exercises. Training should be slow ; to train for a month before an event is insufficient. Too many baths are injurious ; the cold morning tub is sufficient. Most young men, like old ones, eat too much. A varied diet of ordinary foods is better than fads. Tobacco and alcohol are baneful.

I cannot conclude this section without the formal opinion, which I expressed in the first edition, an opinion founded on thirty years of close observation of heart-stress, that the importance of muscular effort as a factor in cardiac injury has been much exaggerated. In the sound adult organism the effects of physical stress upon the heart are promptly counteracted by equilibrating machinery, and especially by large expan-

sion of muscular and pulmonary areas. Such a statement as that made some time ago by the editor of a leading medical journal, "that the violent strains of hard exercise bode in the end the certainty of premature decrepitude," and that "the heart can only perform a certain total measure of work," so that "whether this be done by a rapid or a slow process determines the length of days in which it is done," seems to me, both on clinical and physiological evidence, to be unjustifiable. The curse of school and university athletics is publicity and the patronage of the vulgar press. Headmasters should resist all tendencies to remove the games from the precincts of the schools.

The story of strain in the aortic area of the heart will find its place in a later article.

<div style="text-align:right">CLIFFORD ALLBUTT.</div>

REFERENCES

1. ALBRECHT. *Der Herzmuskel*, Berlin, 1903.—2. ALLBUTT, CLIFFORD. "Strain of the Heart," *St. George's Hosp. Rep.*, 1870 (1871), v. 23.—3. ANDERSON, H. B. "Strain as a Factor in Cardio-aortic Disease," *Brit. Med. Journ.*, 1905, ii. 840.—3*a*. ARNSPERGER. *Die Röntgenuntersuchung d. Brustorgane*, 1909.— 3*b*. BENCE. *Ztschr. f. klin. Med.*, Berlin, 1905-6, lviii. 203.—3*c*. BERGMANN u. PLESCH. "Schlagrolumen des Herzens," *Kongr. f. inn. Med.*, Wiesbaden, 1909.—4. BLAKE and LARRABEE. *Boston Med. and Surg. Journ.*, 1903, cxlviii. 195.—5. BROADBENT, Sir W. *Lancet*, 1897, ii. 1260.—6. BROWN, CARNEGIE. "Degeneration of the Myocardium in Hot Climates," *Brit. Med. Journ.*, 1906, i. 1462.—7. BRUCE, J. MITCHELL. "Lettsomian Lectures," *Lancet*, London, 1901, i.—8. BRUCK. "Blutdruck bei plötzlichen starken Anstrengungen," *Deutsch. Arch. f. klin. Med.*, 1907, xci.—9. BRUNTON, LAUDER. *Clin. Journ.*, London, 1909, xxxiii. 380.—10. BRUNTON and TUNNICLIFFE. *Journ. Physiol.*, Cambridge, 1894, xvii. 5.—11. CAMP, de la, "Exper. Studien über d. acut. Herzdilatation," *Ztschr. f. klin. Med.*, 1903, li. 1904.—12. CHRIST. "Muskelarbeit und Herzthätigkeit," *Deutsch. Arch. f. klin. Med.*, 1897, lix.—13. COLLIER, W. "Functional Albuminuria in Athletes," *Trans. Med. Soc.*, London, 1907, xxx. 75.—14. *Idem. Clin. Journ.*, London, 1909, xxxiii. 378.—15. CRIEGERN, v. Quoted by Bruck.—16. DA COSTA, J. M. "Over-strain of the Heart," *Amer. Journ. Med. Sc.*, Phila., 1871.—17. *Idem. Strain of the Heart* (Toner Lect. No. III.), Washington, Aug. 1874.—18. *Idem.* "Cardiac Asthenia," *Am. Journ. Med. Sc.*, Phila., 1894, cvii. 361.—19. DALLY, J. F. HALLS. Thesis for M.D., Cambridge.—20. DIETLEN und MORITZ. (Orthodiagraphic Observations on the Heart in Work), *München. med. Wchnschr.*, 1908.—21. EDGECOMBE and BAIN, *Lancet*, London, 1899, i. 22. FEILCHENFELD, *Deutsche med. Wchnschr.*, 1908.—23. FRAENTZEL. "Hypertrophy from Over-strain," *Virch. Arch.*, 1857.—24. GORDON. "Observations on a Club-swinger," *Edin. Med. Journ.*, 1907.—25. GOSSAGE. "On some Aspects of Dilatation of the Heart," *Med.-Chir. Trans.*, London, 1907, xc. 1.—26. *Idem.* "The Tone of Cardiac Muscle," *Proc. Roy. Soc. Med.*, London, 1908, I. (Med. Sect.) 254.—27. GRÄUPNER. *Deutsche med. Wchnschr.*, 1906, xxxii. 1028.—28. GREBNER und GRÜNBAUM. *Beziehungen d. Muskelarbeit z. Blutdruck* (Gaertner's Method, C.A.), Berlin, 1899.—29. GROBER. (Cardiac Hypertrophy from Exercise), *Pflüger's Arch.*, 1908, cxxv. 507.—30. HALDANE and LORRAIN SMITH. "The Percentage Oxygen Capacity, Total Oxygen Capacity, and Total Mass of the Blood in Man," *Journ. Physiol.*, London, 1899-1900, xxv. 5.—31. HALLION et COMTE. *Compt. rend. Soc. biol.*, Paris, 1896, xlviii. 903.—32. HENSON. *Deutsch. Arch. f. klin. Med.*, 1907, xci.—33. HESS. *Med. Gesellsch. in Göttingen*, Sitzung 1903.—34. HERZ. (On the Soldier's Heart), *Centralbl. f. innere Med.*, 1894, xv. 212.—35. *Idem. Wien. med. Wchnschr.*, 1903, xliii. 1761.—36. HILL, L. "Blood-pressure in Exercise," Schäfer's *Textbook of Physiology*, 1900, vol. ii.—37. *Idem. Recent Advances in Physiology*, 1906.—38. HILL and FLACK. *Journ. Physiol.*, Cambridge, 1909, xxxviii. ; *Brit. Med. Journ.*, 1909, i. 927.—39. JANOWSKI. *Ztschr. f. klin. Med.*, 1907. — 40. JONES, E. LLOYD. *Lancet*, London, 1908, i. — 41. KAUFF-MANN. "Influence des mouvements musculaires physiologiques sur la circulation

artérielle," *Arch. de physiol. norm. et path.*, Paris, sér. 5, iv. 493.—42. KLEMPERER. *Kong. f. inn. Med.*, Wiesbaden, 1907.—43. KORNFELD. "Einfluss psychische und geistige Arbeit auf dem Blutdruck," *Wien. med. Bl.*, 1899.—44. KRAUS. (Orthodiagnosis of Heart in Exertion), *Deutsche med. Wchnschr.*, 1905, xxxi. 90.—45. KRAFT. *Balneol. Kongr. Berlin*, 1908.—46. KREHL. *Pathologische Physiologie*, Leipzig, 1907. —47. KÜLBS. *Arch. exper. Path. und Pharmak.*, 1906, lv. 288.—48. LAMBERT. "Strain of the Heart in Growing Boys," *Med. Chron.*, Manchester, 1904-5, xli. 278. —49. LYDSTON. *Amer. Med.*, 1903.—50. M'WILLIAM. *Proc. Roy. Soc.*, 1893.—51. M'CARTHY, J. M'D. *Functional Disease of the Heart in Soldiers*, Thesis for degree, Camb., 1898.—52. MACKENZIE, JAMES. *Disease of the Heart*, Oxford, 1908.—53. MACNAUGHTON. "Chorister's Heart," *Lancet*, London, 1905, ii. 1136.—54. MAREY. *Circulation du sang*, 1881.—55. MASING. "Verhalten d. Blutdrucks d. jungen u. bejahrten Menschen bei Muskelarbeit" (Dehio's Method, C.A.), *Deutsch. Arch. f. klin. Med.*, 1902.—56. MARTIUS. *Klin. ther. Wchnschr.*, 1899.—57. MUNRO. *Boston City Hosp. Rep.*, 1897.—58. MORGAN. *University Oars*, 1869.—59. MORITZ. "Blutdruck bei Körperarbeit ges. u. Herzkranker," *Deutsch. Arch. f. klin. Med.*, 1903. —60. MOSSO, A. *Life of Man in the High Alps*, 1898 (Transl. from 2nd ed.).—61. MYERS, A. B. *On the Frequency and Causes of Heart Disease in Soldiers*, London, 1870. —62. OLIVER, G. *Pulse-gauging*, 1895.—62a. PAWLOW. *Arch. f. Anat. u. Physiol.*, 1889.—63. PEACOCK. Croonian Lectures; and various Contributions, 1851-65.—64. PLESCH. *Deutsche med. Wchnschr.*, Feb. 11, 1909.—65. POYNTON. "Cardiac Overstrain in the Young," *Brit. Med. Journ.*, 1899, ii. 474.—66. PRINCE, MORTON. "Transient Dilatation of the Heart," *Am. Journ. Med. Sc.*, Phila., 1901, cxxi. 188.— 67. ROLLESTON, H. D. "On the Endocardial Pressure-Curve," *Journ. Physiol.*, London and Cambridge, 1887, viii. 235.—68. ROMBERG. *Deutsche med. Wchnschr.*, 1908, xxiv. 2009.—69. ROY and ADAMI. "Strain of the Heart," *Brit. Med. Journ.*, 1888, ii.—70. *Idem.* *Phil. Trans.*, 1892, 199.—71. SCHIEFFER. (On Exertion and Size of Heart), second paper in *Deutsch. Arch. f. klin. Med.*, 1906-8.—72. SEITZ, JOH. "Lehre v. d. Ueberanstrengung des Herzens," *Deutsch. Arch. klin. Med.*, 1872.— 72a. SELIG. *Baln. Versammlung*, Breslau, 1908.—72b. *Idem.* *Prag. med. Wchnschr.*, 1905. —73. SMITH. *Veterinary Physiology*, 1907, 3rd ed. —74. STADLER. (Nature of Cardiac Hypertrophy), *Deutsch. Arch. f. klin. Med.*, 1907, xci.—75. STÄHELIN. "Muskelarbeit und Herzthätigkeit," *Deutsch. Arch. f. klin. Med.*, 1897, lix.—76. STENGEL. *Trans. Assoc. Amer. Physicians*, Phila., 1899, xiv. 331.—77. STEWART. *Journ. Physiol.*, Cambridge, 1898.—78. STRASBURGER. *Ztschr. f. klin. Med.* lxxxi.—79. STURSBERG. "Blutdruck nach Körperarbeit," *Deutsch. Arch. f. klin. Med.*, 1907, xc. 548.—80. TANGL und ZUNTZ. "Ueber die Einwirkung d. Muskelarbeit auf dem Blutdruck," *Pflüger's Arch.*, 1898, lxx. 544.—81. THURN, W. *Die Entstehung v. Krankheiten als directe Folge anstrengender Märsche*, Berlin, 1872.— 82. TIEDEMANN. Funktion d. Herzens, *Deutsch. Arch. f. klin. Med.*, 1907, li. 331.—83. TRAUTWEILER. "Mechanik und Physiol. d. Bergsteigens," *Jahrbuch S.A.C.*, 1883-84. —83a. TYSON. (Soldier's Heart), *Clin. Journ.*, London, 1906, xxviii. 205.—84. WALLER. *Physiology: and Various Papers.*—85. WEBER. HERMANN. "Hygiene of Climbing," *Lancet*, London, 1893, ii. —86. WEISS, O. *Pflügers Arch.*, 1908, cxxiii. 341. — 87. WEST, S. *Clin. Journ.*, London, 1909, xxxiii. 382.—88. WILLIAMS. "The Heart of Runners," *Boston Med. and Surg. Journ.*, 1899, cxli. 245.—89. WILLIAMS and ARNOLD. "The Effects of Violent and Prolonged Exertion on the Heart," *Phila. Med. Journ.*, 1899.—90. WILLIAMSON, O. K. "Arterial Blood-pressure Records before and after Exercise," *Brit. Med. Journ.*, 1909, i. 530.—91. WILSON. "Disordered Heart in Young Soldiers," *Army Med. Rep.*, 1896.—92. ZUNTZ. "Die Ernährung d. Herzens," *Deutsche med. Wchnschr.*, 1892, xviii. 109. — 93. ZUNTZ, LOEWY, u. AND. *Höhenklima und Bergwanderungen in ihrer Wirkung a. d. Menschen.*, Berlin, 1905.—94. ZUNTZ und SCHUMBERG. *Studien zur Physiol. des Märsches*, 1901.

C. A.

INJURIES BY ELECTRIC CURRENTS OF HIGH PRESSURE

By Sir Thomas Oliver, M.D., LL.D., D.Sc., F.R.C.P.

Since electricity has come to be so widely and increasingly used as an illuminating agent and for motive power, accidents of varying severity have been frequent. It is desirable, therefore, that we should be cognisant of the effects of high electrical currents upon the human body. We know that there is considerable danger attendant upon the generation of electricity, and we look to the expert electrician to adopt measures to prevent accidents. Since 1902, the year in which death by electricity employed in industrial undertakings and for motive purposes became notifiable to the Home Office, there have occurred the following fatal cases:—7 in 1902; 4 in 1903; 6 in 1904; 7 in 1905; 10 in 1906; and 11 cases in 1907. When we add to these the many lesser accidents that frequently occur, we recognise the need for careful precaution wherever electricity is being generated and distributed. Many of the accidents have been due to inadvertent contact with exposed parts of highly charged metal not properly insulated. The consequences of the current thus passed through the body vary with the amount of current entering, the insulated position of the individual at the time, and the kind of contact. Such conditions, for example, as standing on wet earth, the wearing of damp boots, and a moist skin tend to increase the effects of an electrical current. The danger, therefore, is not one simply of high potential, but of current plus the conditions under which it has been received. The word voltage used in this article is synonymous with "pressure" as used by the Board of Trade, and with the "electromotive force" of the textbooks.

It is difficult to say what voltage is fatal to man. Speaking in terms of voltage, Dr. W. S. Hedley says that 1000 to 2000 volts will kill. In America, where electricity was adopted as the official means of destroying criminals, a current of 1500 volts has been regarded as capable of causing death; but there are many cases on record of persons having been exposed to higher voltages without fatal consequences, and, on the other hand, contact with lower pressures has caused death. Of the two kinds of electric current—the "continuous" and "alternating"—opinions differ as to which is the more dangerous to the human body. It is more generally believed that the alternating is the more fatal. From 1902 to 1908 three fatalities only, due to the continuous current at 250 volts or less, have been reported, whereas, as a consequence of having received alternating currents, thirty deaths have been notified during the same period. These figures alone suggest that the alternating current is more dangerous than the continuous. On the relative danger to life of the continuous and alternating currents, the Report of the Board of Trade

states that alternating currents are twice as dangerous as the continuous. As electricity is too difficult a subject for a non-expert to handle, only those points are here discussed which bear upon the medical aspect of the subject, points with which medical practitioners should be familiar, as at any time they may be called to persons injured by high electric currents.

As an illustration of the uncertain effects which follow contact with a live wire without a fatal result, I saw, in September 1908, a workman aged nineteen years, who had received a shock of 6000 volts, alternating current, without fatal result. His right hand was almost burned through, it was deeply charred and sloughy. There were several large angry-looking blisters on the lower part of the forearm, and blisters on the soles of the feet. The patient's boots were untouched. This youth, who had been working on the three-phase system, had first touched one wire and then another without any result, but on touching the third wire he suddenly felt his head swim, fell, and became unconscious. He had been standing on a concrete floor at the time of the accident, and must have been pretty well insulated, otherwise the shock would have been immediately fatal.

A person may be seriously injured either by direct personal contact with a highly charged piece of metal, through the medium of damp clothes, or through an iron tool in his hand by which accidental contact is made with the live metal. As an illustration I may mention the fatal accident to a youth at St. Peter's, Newcastle-on-Tyne, in January 1897. Carrying an iron ladder through the factory he accidentally brought the top of the ladder into contact with the terminals of an arc lamp. He was killed instantaneously. In regard to arc lighting, it may be mentioned that whilst each arc light requires an electrical pressure of only from 40 to 50 volts, the lamps are usually arranged in a series and are supplied by the same current. A workman who is himself insulated may touch either terminal of an arc light without receiving any injury ; but should his insulation be defective, if he stand on moist earth for example, he may receive, as did the youth at St. Peter's, a fatal shock, since the electrical pressure between the ends of the cable is the sum of the pressure of all the lamps in series in the circuit (3).

We have no positive proof that one individual is more susceptible to electric shock than another. It is, as already stated, rather a question of the amount of current and whether it wholly enters the body. Where contact with currents of high potential has not been followed by disastrous results, it is more than probable that at the time of contact the skin was dry, in which state it is a bad conductor and offers considerable resistance to the penetration of the current. As might be expected, the electrical current produces very varying effects upon the human body. The electrolytic and physiological effects are proportional to the quantity of electricity passed. Where the voltage is low and the contact fairly good the muscles are thrown into a state of tetanic rigidity

which makes it impossible for the individual to relax his grasp of any charged metal he may have seized, nor can he be released until the circuit is broken. The effects of electric currents are experienced when they enter and when they leave the body. It is sufficient for us to remember that effects are produced at the moment of the entrance into and exit of currents from the body, and that these, therefore, are periods of danger. Dr. Hedley, in supporting the opinion that the quantity of electricity passed determines the amount of electrolytic action and physiological effect, considers that more pain is felt the higher the electromotive force, even when the current is the same. One element entering into the causation of pain is the local action of the accumulated products at the point of contact consequent upon electrolytic decompositions, and the relative resistances between the electrodes and the different layers of the skin. The individual through whose body there is passing an electric current of not too high potential generally experiences pain, but some of this must be due to the extreme contraction of his muscles quite apart from the influence of any products of electrolysis. If there be no immediate loss of consciousness, terror may cause him to faint. The memory of this plays no small part in the subsequent development of nervous symptoms. Once liberated, the patient, as a rule, is soon well again, but there are instances on record in which for many months after exposure to the current there was complaint of ill-defined pains and headache which recurred with electrically disturbed conditions of the atmosphere, and of a form of persistent nervousness which was rather the result of the mental than of the physical shock.

Another consequence of the exposure to high electric currents is burning. The portion of the surface of the body which has accidentally been brought into contact with the charged metal may become black and charred, the peculiarity of such a wound being that it is sometimes deep and apt to slough, and that whereas the burned part is insensitive to pain the surrounding tissues are extremely sensitive. Should the skin be moist at the time of contact the burn is much more severe. If a current sufficient to produce this severe local burning pass through the body, fatal results are the more probable; but if the current merely pass locally, as from the hand to the wrist, for instance, the damage will probably be local only.

I saw at Wallsend a good illustration of the severe local effects of electricity; a man who, in crossing a railway, inadvertently touched with his wet boot one of the live rails on the North Eastern Railway Company's circuit; he was observed by a signalman to be suddenly thrown down, fortunately away from the live rail. It was with difficulty that he was rescued. For some time after the accident he was violently convulsed, and for several hours he remained in a state of unconsciousness. His boots were uninjured, but on removing them the toes and the front of each foot were found to have been severely burned. When I saw the patient two days after the accident he was suffering considerable pain from intense inflammation around the injuries. The skin

of the feet was in places angry-looking, and the wounds were disposed to slough. The bones of the big toes and metatarsi were exposed. It is characteristic of all electrical injuries that the wounds heal slowly, and those of this patient were no exception to the rule, for they sloughed, and it was several weeks before any attempt at healing took place.

Although the local injuries caused by electricity are usually on the surface, and are of the nature of burns, they are occasionally deeply seated, although there is no external sign to indicate the damage done to the concealed parts. Thus, the injuries of this kind may be so severe in character as to require, a few days later, removal of the limb in consequence of deep sloughing, and after a further interval of a few days another limb may have to be amputated for similar reasons.

As electrical production and distribution now come under the Factory Act, injured workmen have claimed, and been successful in obtaining, compensation under the Act of 1906.

When the pressure has been high, the contact good, and conditions of resistance slight, the patient may at once be rendered unconscious, or be suddenly killed. Thus stricken by a powerful current a man suddenly falls, or he is thrown a distance of several feet before falling. A peculiar cry is involuntarily uttered, especially when the contact is broken, which, in electrical generating stations, for example, at once attracts workmen to the spot where their comrade is lying pale or slightly cyanosed, pulseless, apparently dead, and with mucus escaping from his mouth and nose ; now and then a feeble and gasping respiration is observed, but he lies helpless, his pupils keep dilating, and unless artificial respiration is at once resorted to, and sometimes even then, death is inevitable. There is something appalling in the extreme suddenness and severity of the shock in these cases, towards which the unexpectedness of the accident possibly contributes.

Cause of Death.—In conjunction with Dr. R. A. Bolam I undertook a series of experiments in the Physiological Laboratory of the Newcastle College of Medicine, using the constant current, upon anaesthetised dogs, with the view of ascertaining the cause of death by electric shock, and of testing the means of resuscitation. Two opinions are held by the profession : (i.) that death in such circumstances is due to respiratory arrest ; (ii.) that it is consequent upon sudden cessation of the heart's beat. By placing dogs under the influence of ether we were able to take a tracing of the arterial pressure and respiratory movements, and thereby to record the effects of high electric currents passed into the body. Immediately on making contact the animal is thrown into an attitude of opisthotonos, its muscles become extremely rigid, and as a consequence the lever recording respiratory movement is suddenly and violently thrown up, whilst the other, which traces the arterial pressure and heart-beats, suddenly rises owing to general arterial constriction, and, falling shortly afterwards, oscillates rapidly but within a narrower range. On breaking the current the respiration becomes deeper and quicker than before the shock, and in the course of a few seconds the breathing and the beat of

the heart return to the normal. When the current proved fatal there was the same initial respiratory and general muscular spasm, and a sudden rise of arterial pressure followed by an immediate fall ; one or two quivering oscillations of the lever marked the arterial tracing, and then all at once a further and complete fall of the lever followed, indicating that the heart had ceased to beat. Respiration deep and spontaneous may continue for several seconds, or even for a few minutes after the heart has ceased to beat. The experiments invariably shewed that in electric shock the death was cardiac and not respiratory. Other steps were taken to confirm this opinion, notably by listening to the heart of the animal with the stethoscope as the current entered. When the current was insufficient to kill the dog the heart's beat was momentarily delayed and then quickened, the cardiac sounds being well maintained ; but when, on the other hand, a current of higher potential was employed, the sounds of the heart would cease, immediately or very shortly after contact. Respiration deep and rhythmic might continue, but if no treatment were adopted the cardiac sounds would not return ; increasing pallor would gradually steal over the whole surface of the body, the pupils meanwhile dilating, and mucus being forcibly driven from mouth and nares. By exposing the heart of other anaesthetised dogs, and inserting a cannula into the trachea so as to carry on artificial respiration, we had ocular demonstration that it was the heart which was primarily arrested in death from electric shock, and not the breathing. Dr. A. M. Bleile, Professor of Physiology, Ohio State University, in a paper read before the American Institute of Electrical Engineers, Niagara Falls, N.Y., June 27, 1895, states that " death in electric shock is really due to the fact that the current produces a contraction of the arteries through an influence on the nervous system, and that this constriction of the arteries throws in such a mechanical impediment to the flow of the blood as the heart is unable to overcome, and that where drugs are given to counteract this effect, much larger doses of electricity can be borne." As to the constricted state of the arteries, we found, with Bleile, that if nitrite of amyl were inhaled by an animal before the electrical experiment much stronger currents could be borne. Our results and those of Dr. Lewis Jones are opposed to those of d'Arsonval, who attributes death to asphyxia.

In the main, death is the result of the action of the electric current upon the heart, even when the electrodes have been applied to the head, and this occurs, too, after the influence of the vagus has been abolished by the administration of atropine. It is not exactly known whether the heart-muscle is paralysed by the current, killed by the molecular changes produced by the current, or brought to a standstill as a result of the enormous resistance of the peripheral arteries to which I have referred. There is no definite ratio between the strength of the current and the size and the weight of the animal. A current passed on one occasion for several seconds without bad effect will on another occasion kill the animal in two seconds. J. L. Prévost and F. Battelli found that, with

alternating currents of feeble tension, fibrillary tremor of the heart was readily induced, and that with a tension of 20 to 40 volts the heart became paralysed, but that respiration continued.   With high alternating currents of 240 to 600 volts the beat of the heart and respiration were in dogs simultaneously arrested.   The effect of continuous currents was found to be practically the same as that of alternating currents, with the following difference.   Voltages of less than 50 did not produce fibrillary arrest of the heart's action when continuous currents were employed, but with alternating currents 10 volts sufficed to produce fibrillary tremor, followed by stoppage of the beat of the heart.

It is difficult to say what is the amount of electrical pressure required to kill a person.   H. F. Weber, constituting himself a measuring instrument, undertook, by experiments upon himself, to solve, if possible, this question, so far as the pressure in electric railways with overhead wires is concerned.   In one set of experiments the hands were wetted, and in the other dry.   Using alternating currents, and with wet hands, Weber found that with 30 volts the fingers, hands, wrists, forearms, and arms were almost paralysed, and that considerable pain was experienced.   By using great determination the wires he had caught could be released. With 40 volts the fingers, hands, and arms were instantaneously paralysed, the pain was almost unbearable, and the wires could hardly be released.   With 50 volts the wires could not be let go.   After 50 volts, when the skin is wet and the contact good, Weber is of the opinion that electrical pressures are dangerous, since the wires cannot be released. When the wires were caught by dry hands they could be released up to 90 volts, but the pain with this voltage was so severe as to cause the experimenter to scream involuntarily.   Weber concludes that "a simultaneous touching of both of the poles of an alternating current is dangerous as soon as the pressure exceeds 100 volts, and since it is impossible to set one's self free, the case must be regarded as fatal whenever immediate help is not at hand."   It is unnecessary to add that when a person is properly insulated he can be charged to much higher pressures than these without incurring great danger.

A young labourer was recently admitted into the Newcastle-upon-Tyne Royal Victoria Infirmary under my colleague Mr. Angus, suffering from burns and shock consequent upon accidental contact with a high-frequency current of 20,000 volts.   When I saw him two hours after the accident, he was unconscious, extremely restless, constantly throwing himself about in bed, and had widely dilated pupils.   There were several burns on the body, including a large circular burn on the back of the head with exposed bone.   He died seven hours after the accident. The necropsy made forty hours after death shewed a haemorrhage into the occipital lobe of the brain, and numerous small haemorrhages under the visceral layer of the pericardium ; the liver, kidneys, and portions of the lungs were deeply congested.

Whilst electricity is capable of producing the most serious consequences, even to the extent of destroying life, it can yet be utilised as a

curative agent in disease. We are but in the infancy of its utility in the form of high-frequency currents. It has recently been shewn that one of the effects which electrical currents are capable of inducing is electrical narcosis or sleep. To Stéphane Leduc we are indebted for what is known of *sommeil électrique*, a form of narcosis analogous to that which follows the inhalation of chloroform. The current used by Leduc is an intermittent one of low tension and constant in direction. He points out that vertigo in man and epileptiform convulsions in animals can be induced by electrical stimulation of the brain, also complete general anaesthesia. On cessation of the current the animal immediately awakes, and does not shew any sign of suffering.

Morbid Anatomy.—There is usually well-marked rigidity of the muscles. The skin may or may not shew signs of burning or of eschars ; it may be pale or livid. The abdominal viscera and large veins are usually deeply congested. The heart is usually flaccid : sometimes the right side is flaccid, and the left is hard and tense. The right auricle and ventricle are considerably distended and are filled with dark fluid blood ; the left auricle is generally moderately distended and contains fluid blood, whilst the left ventricle is firm and almost empty. The lungs present nothing abnormal ; they may be slightly congested or shew scattered ecchymoses, particularly if artificial respiration has been attempted. The brain and spinal cord are congested, but are otherwise normal. I have seen it stated at a coroner's inquest, the diagnosis being based upon the assertion and the corresponding verdict of the jury returned, that in death from electricity the blood is fluid and not coagulated after death. This statement is too sweeping and not quite correct. In most cases, it is true, the blood is found fluid after death, but in some of our experiments we found coagula in the right side of the heart, and occasionally some of the large veins were blocked by dense dark clot—particularly when the autopsy was made twenty-four to thirty hours after death. It is maintained that on spectroscopic examination the oxyhaemoglobin of the blood is reduced. If a strong solution of blood is examined, only one broad band may be observed in the spectrum, and it appears at first sight as if this were due to reduced haemoglobin ; but when the spectrum is very carefully scrutinised, and particularly, too, when the solution of blood is further weakened by the addition of water, two distinct bands of oxyhaemoglobin can be clearly discerned. It would appear, therefore, that the blood contains both oxyhaemoglobin and reduced haemoglobin. The blood on microscopical examination shews marked crenation of the erythrocytes. The pupils were invariably found to be widely dilated immediately after death. Portions of the brain and medulla oblongata of the young labourer killed after 20,000 volts (*vide* p. 258) were examined by Drs. F. W. Mott and Schuster, to whom I am indebted for a description of the microscopical appearances. There were several minute haemorrhages in the brain-tissue due to ruptured vessels in the cortex and subjacent white matter. The blood in the vessels and extravasated into the tissues shewed evidence of haemolysis. The ganglion-cells of

the cortex presented chromolytic changes, whilst others stained by the polychrome-eosin method had apparently undergone coagulative necrosis. Chromolytic changes of a pronounced degree were present in the large and small cells of the medulla oblongata. In many of the cells the chromophil substance was either absent or much diminished. Possibly some of these changes may have been due to the interval, forty hours, between death and the necropsy.

Treatment.—Persons who have received only a slight shock, and who have not been rendered unconscious, do not require any special treatment. The effects almost immediately pass away, and should any nervous symptoms remain they must be treated on general principles. For any burns or wounds ordinary surgical remedies will avail. It is to the treatment of persons who have been exposed to high electrical currents, and who are apparently dead, that the following remarks apply. D'Arsonval, believing the mode of death to be akin to asphyxia, recommended artificial respiration, and of all modes of treatment, quite irrespective of whether the death has proceeded from failure of the respiratory centre or of the heart, I know of no treatment more likely to be beneficial than artificial respiration, systematically carried out by Sylvester's method, and continued for half an hour or longer. Dr. Bolam and I twice succeeded in resuscitating a dog whose heart had ceased beating, once for thirteen minutes and on the second occasion for eight. The heart, which was exposed to view, had become rapidly distended so as to bulge out the pericardium, and had become perfectly motionless after having passed through a stage of fibrillary tremor. But by persisting in artificial respiration, aided by the occasional spontaneous inspirations which from time to time occurred, and the rhythmic traction of the tongue, the contents of the right side of the heart were gradually aspirated into and through the lungs, and auricular beats were re-established, at first irregularly and feebly ; gradually, however, they became stronger and passed over into the ventricle, so that after thirteen minutes, during which the heart was apparently irresponsive, we had the satisfaction of seeing the normal beat of the organ restored, the pulmonary and systemic circulation reestablished, and life return. Leduc points out that when an animal has been apparently killed by electricity, and the heart has ceased beating, the animal can usually be restored to life by applying the same current to the body, for since this produces immediate contraction of the muscles of the body, including those of inspiration, artificial respiration is thus aided, and can be proceeded with. Too often, however, the sufferer is killed outright, and all attempts at resuscitation fail. Rescuers on approaching the injured must beware lest the current be not broken.

THOMAS OLIVER.

REFERENCES

1. D'ARSONVAL. *Compt. rend. Acad. d. sc. Paris*, 1887 - 94. — 2. BLEILE. *Electrical Rev.*, Aug. 9, 1895 ; and other papers in the same journal, Dec. 1894, Jan. 1895.—3. Dangerous Trades Committee of the Home Office, *2nd Interim Report*, 1897. —4. HEDLEY, W. S. "Currents from the Main"; also *Lancet*, 1891, ii. 1892,

i. 69, 794.—5. Jones, H. Lewis. "The Lethal Effects of Electric Currents," *Brit. Med. Journ.*, 1895, i. 468.—6. Leduc, S. "Sommeil électrique," *Presse méd.*, Paris, 1907, xv. 129.—7. Lucas, R. C. "Accidental Electrocution causing extensive Gangrene of all the Extremities," *Trans. Clin. Soc.*, London, 1905, xxxviii. 86.—8. Ministère des Travaux Public, *Circulaire du* 19 *août*, 1895, Paris.—9. Mommerque. *Contrôle des installations électriques*, Paris, 1896.—10. Oliver, T. *Diseases of Occupation*, art. "Electricity."—11. Oliver and Bolam. "Death by Electric Shock," *Brit. Med. Journ.*, 1898, i. 132.—12. Prévost et Battelli. *Journ. physiol. et de path. gén.*, Paris, 1900, ii. 40.—13. Report of Board of Trade, 1889.—14. Tatum. *Electrical World*, May 10, 1890.—15. Weber, H. F. "What Pressure is dangerous on Electric Railways with Overhead Trolly Wires," translated in *Nature*, 1900.

T. O.

ACUTE SIMPLE ENDOCARDITIS

Synonyms.—*Benign, Papillary, Verrucose, Rheumatic Endocarditis.*

By the late Prof. J. Dreschfeld, M.D., F.R.C.P.
Revised by Thomas M'Crae, M.D., F.R.C.P.

Definition and Classification.—By endocarditis we mean inflammation of the endocardium or lining membrane of the heart. The inflammation affects principally and often exclusively the valve segments of the endo-cardium (valvular endocarditis), but other parts of the endocardium may be affected also (mural endocarditis). Both clinically and pathologically we distinguish between acute and chronic endocarditis. The acute form is again divided into benign or simple and malignant or infective endocarditis. The term benign is not a good one ; it may be regarded as such in comparison with the malignant form, but regarded by itself is far from benign. Of the chronic form, likewise, we dis-tinguish two kinds—one which is the result of acute endocarditis, and the other the fibroid or sclerotic form, which results from arteriosclerosis or atheroma.

In the article on Infective Endocarditis (Vol. I. p. 905) the difficulty of separating simple from infective endocarditis was considered. In both forms micro-organisms have been found in the affected valves, though only in the infective form do they play an essential part, so far as symptoms are concerned. Whilst the two kinds have many features in common, in others they differ ; and as the difference is often essential it is advisable to consider the two kinds separately.

In this article we shall consider simple acute endocarditis ; chronic endocarditis, giving rise to the majority of the so-called valvular affections of the heart, is dealt with elsewhere.

Causation.—It cannot be emphasised too strongly that endocarditis is always due to some infection, and in one sense may be regarded as

always due to a previous general infection, not necessarily, however, with general features. By far the largest number of cases occur with (*a*) acute rheumatic fever, hence by some authors the name acute rheumatic endocarditis is given to the disease. Its frequency in rheumatic fever is differently estimated by different authors, and this is readily to be understood, for in many cases the symptoms of endocarditis may be so slight as to escape detection ; or again persons recovering from acute rheumatic fever may shew signs simulating those of endocarditis ; these may be due to myocarditis or some functional derangement of the heart. The most trustworthy observations on this subject are those in which a large number of cases of rheumatic fever have been kept under observation, and the after-history watched for some time. Sibson analysed 325 cases of acute rheumatic fever observed during fifteen years at St. Mary's Hospital, and found that in 79 there was no endocarditis ; in 63 endocarditis was threatened ; in 13 endocarditis was probable ; in 107 endocarditis was present without pericarditis ; in 54 there was endo-pericarditis; in 6 there was pericarditis without endocarditis ; in 3 there was pericarditis with doubtful endocarditis. In a series of 300 cases of rheumatic fever studied in the Johns Hopkins Hospital, 35 per cent had endocarditis, and in 23 per cent the condition was doubtful, the patient being discharged with a murmur but no other signs of organic disease (15). The mean of the numbers given by various writers is between 20 and 30 per cent. As important points which have been made out regarding the relation of rheumatic fever and endocarditis we may note : (i.) That, in connexion with rheumatism, endocarditis occurs more frequently in children than in adults ; its incidence has been estimated at 61·3 per cent (C. West), about 66 per cent (Fuller), and even as high as 80 per cent (Cadet de Gassicourt). Dr. Poynton, in an analysis of 150 fatal cases of rheumatism in patients under twelve years of age, found that 149 had a valvular lesion. In the Johns Hopkins Hospital series the incidence of endocarditis, when the first attack was before the age of twenty years, was 45 per cent, more than double that in those over twenty years at the time of the first attack (20 per cent). (ii.) The first attack of rheumatic fever is more often followed by endocarditis than the subsequent attacks. (iii.) Endocarditis may occur with mild or severe attacks of rheumatic fever. It must be remembered that the degree of arthritis is no indication of the severity of the attack, especially in children. (iv.) The physical signs of endocarditis usually appear early in the attack of rheumatism. Sibson in about one-fourth of his cases noticed the presence of a systolic bruit, which he looked upon as characteristic of endocarditis, at the end of the first week of the rheumatic fever, and in two-thirds at the end of the second week. Sometimes, however, the signs of endocarditis appear much later, though probably in many of these cases the endocardial affection had existed some time before it gave rise to physical signs. The endocardial affection may precede the arthritic manifestations by several days. (v.) The endocarditis dependent on rheumatic fever most frequently attacks the mitral valve ; the aortic valve less frequently, and

the right side of the heart in very exceptional cases only. In the Johns Hopkins Hospital series the mitral valve was affected in 95 per cent and the aortic in 23 per cent, but in 5 per cent only was the aortic valve exclusively affected. Among 535 cases in St. Thomas's Hospital (25) the mitral was attacked in 97 per cent and the aortic in 12 per cent (the aortic alone in only 3 per cent).

(b) *Chorea.*—Endocarditis is frequently met with in persons who have had chorea ; and in fatal cases of chorea inflammatory deposits on the valves are almost invariably found. Thus, Sturges (26) collected statistics of 80 fatal cases, and in only 5 of these were the heart-valves normal. Reymond's figures bear out the same rule. As regards the frequency of endocarditis in chorea, authors differ considerably ; and, as the endocarditis may not reveal itself until years after, the exact proportion is not easily made out. Prof. Osler states that of 554 cases of chorea at the Infirmary for Diseases of the Nervous System, Philadelphia, 170 presented heart murmurs ; of these, in 149 the murmur was apical, in 21 basic. Of 449 cases reported to the Committee on Collective Investigation of the British Medical Association, 113 had heart murmurs ; how many of these were functional and how many organic it is impossible to estimate. More trustworthy results are obtained if the subsequent history of persons having had chorea is taken, an estimate which has been made by several observers. Sir Stephen Mackenzie examined 33 patients at periods varying from one to five years after the attack of chorea, and noted signs of undoubted heart disease in 60·6 per cent ; Dr. Donkin in 40 per cent ; Prof. Osler out of 140 cases found the heart normal in 51, in 17 there was disturbance which might reasonably be looked upon as functional, and in 72 cases (51·4 per cent) there were signs of organic heart lesion ; it may be noted that only in 25 of these 72 cases was there a history of acute arthritis. W. S. Thayer found that among 689 cases of chorea, 190 (27·6 per cent) had definite signs of previous endocarditis, and murmurs were present in 45 others. Concerning the relation of chorea to endocarditis, the report of the Collective Investigation Committee of the British Medical Association gives of a total of 439 cases of chorea, 97 with a rheumatic history (about 22 per cent). There are many points which suggest that chorea, rheumatic fever, and endocarditis are three terms of one and the same pathological series. In this connexion the presence, in fatal cases of chorea, of the *Micrococcus rheumaticus* in the pia mater and brain, which shewed various pathological changes, is of great importance.

(c) Various *local infections* must be given an important place in the etiology of endocarditis. Suppuration about the nose and nasal sinuses may be the source, but the first place must be given to tonsillitis, which is probably only secondary to rheumatic fever in frequency as the source of infection. The organisms obtained in cultures from the tonsils—usually some variety of streptococci—have repeatedly been shewn to cause endocarditis when injected into the circulation of animals. Frequently the usual signs of tonsillitis are not present, but when the

tonsils are removed, foci of suppuration are found in the deeper parts. Intestinal infections connected with ulceration may also be a source. Sometimes the focus of infection is in the genito-urinary tract, for example, pyelitis or cystitis, pelvic infections in women, and prostatic infection in males.

(*d*) Acute endocarditis may be associated with the acute *zymotic fevers*. Among these scarlet fever occupies the first place.   Among 2000 cases at the City Hospital, Boston, M'Collom found 13 cases of endocarditis. The onset of endocarditis is often preceded by pains and slight swelling of a joint.   The arthritic symptoms are probably due to the scarlet fever toxin, and the endocarditis may be the result of the action of the same toxin on the endocardium.   In the other acute fevers, such as enteric fever (6 in 1500 cases, the Johns Hopkins Hospital), measles, influenza, small-pox, or diphtheria, endocarditis is rare.   Pneumonia is more often associated with infective endocarditis ; and the same is the case with erysipelas, with puerperal and pyogenetic diseases generally, and with gonorrhoea.

(*e*) In cases of *acute and chronic tuberculosis* we meet with endocarditis occasionally.   Are we to look upon such cases as belonging to the infective type of endocarditis, or do they belong to the benign form, the tubercle bacillus acting as a remoter cause ?   It must be noted that in a few cases the tubercle bacillus has been found in the valve-deposits (2) ; and in some cases of acute miliary tuberculosis vegetations of recent origin have been observed on the heart-valves.   It is probable that two groups of cases can be separated : (i.) those in which endocarditis arises during the course of tuberculosis, but is not due to the tubercle bacillus, and (ii.) those which are directly associated with the tubercle bacillus. In some instances endocarditis may precede the tuberculosis.   Marshall found that acute endocarditis was present in about 5 per cent of phthisical patients.   The proof in any given case that an endocarditis is due to the tubercle bacillus is extremely difficult, and the accepted cases are few.   Of its occasional occurrence there can be no doubt.

(*f*) *Syphilis* attacks the myocardium and the endocardium ; in the former it causes endarteritis and periarteritis with tracts of fibrous tissue in the midst of the myocardium, or it may lead to granular deposits.   In the latter case valvular disease may result from arteriosclerosis, of which syphilis is one of the remoter causes ; that acute endocarditis is ever due to the *Treponema pallidum* is very doubtful.

(*g*) Of other diseases in which endocarditis occurs we may mention gout, chronic nephritis, diabetes, and the erythema group.   Several cases of gout are on record (3, 8), in which endocarditic processes shewing the presence of urate of sodium crystals were found.

In chronic nephritis we often find chronic valvular heart affection from arteriosclerosis, yet occasionally it may be associated with acute endocarditis (7, 22).   In these cases it is probably due to an infection which is often terminal.   In the erythema group the infection may be associated with that of rheumatic fever.

(*h*) *Trauma.*—Several cases have been recorded in which the signs of endocarditis followed a blow or fall on the chest. These, however, should not be regarded as causing endocarditis in the strict sense of the term unless an infection be added. Rosenbach and others have shewn that in the absence of infective organisms, traumatic lesions of the valves are not followed by endocarditis.

(*i*) Endocarditis without any apparent cause, and occurring as an idiopathic disease, has been described by some authors. This, however, cannot be considered as probable, and these must be regarded as instances of infection in which the source was not evident. Cases ascribed to cold are probably due really to a rheumatic or tonsillar infection.

Other etiological factors relate chiefly to *age*. Endocarditis occurs most frequently between the ages of fifteen and forty ; it is rare in old people, in whom valvular lesions are mostly due to a sclerotic process ; it is not rare in children, as observed by C. West, who noticed it 71 times in 122 cases of heart disease (see also Vol. II. Part I. p. 650). In very young children, however, the affection is rare ; in them pericarditis is more often found than endocarditis.

Fetal endocarditis is by no means a rare affection, and may occur with or without congenital anomalies of the heart. The right side of the heart is generally affected in the fetus, but there is always a tendency for the site of any anomaly to be attacked. Apart from the anomalies which dispose to right-sided endocarditis, other factors are in play which determine its frequency as compared with its rarity in extra-uterine life. Such factors are the thickness of the right ventricle, the increased pressure to which it is exposed, and the absence of pulmonary respiration, which causes such a difference between the blood of the right and left sides of the heart after birth, especially as regards the amount of oxygen. Klebs, who was one of the first to attribute all forms of endocarditis to micro-organisms, gives another explanation ; namely, the direct infection of the right side of the heart through the blood coming from the placenta. Fetal endocarditis has rarely been recognised before birth, and may be undetected for years after birth. The fetal right-sided endocarditis affects principally the pulmonary valves—often when there is already obstruction or stricture ; occasionally the tricuspid valve only : similarly, left-sided fetal endocarditis more frequently affects the aortic valve with or without contraction of the lumen of the aorta, and the mitral valve only occasionally.

Finally, endocarditis may be secondary, being an extension of an affection either of the myocardium or of the aorta.

Pathological Anatomy.—Endocarditis affects principally the valves of the heart, hence the name valvulitis ; and, except in the intra-uterine form, it is almost always confined to the valves of the left side : here, again, it affects the mitral more frequently than the aortic valve (the tricuspid valve, however, is occasionally also affected in combination with stenosis of the mitral valve). On the mitral valve it affects the auricular surface, and here again principally the portions of the valve which are

in close apposition when the valve closes; when it affects the aortic valve it is found on the ventricular surface round the corpora Arantii. How often the condition is due to the deposit of organisms from the blood as compared with embolism is difficult to say, although from the absence of vessels in the aortic and pulmonary valves the latter does not seem probable in them. That the left side is much more often affected than the right side is due to several causes, but principally to the higher blood-pressure and the difference in the oxygenation of the blood; the first factor leads more easily to abrasion of the endocardium, and other changes favouring the deposits of inflammatory material or thrombi, and by the latter the action of the micro-organisms is thought to be favoured although no great importance can be given to this. That the mitral valve is more frequently affected than the aortic may be due, as Sibson (*loc. cit.* p. 458) pointed out, to the fact that the mitral flaps press against each other when the valve is shut with much greater tension and force than the cusps of the aortic valve.

It must be noted, however, that other parts of the endocardium, especially the chordae tendineae, are implicated in the process. An endocarditis affecting chiefly other portions of the endocardium, to the exclusion of the valve, has been described by Neuwerck; it is more or less chronic, and leads not only to superficial cicatrices, but also to subendocardial and myocardial inflammation (Rosenbach).

Appearance of the Affected Valves.—In the early stages of endocarditis, which we have but rarely the opportunity of seeing, except in some cases of fatal chorea, the endocardium at the affected parts is slightly swollen, and of the rosy tint of increased vascularity. When the layer of endothelium degenerates, the underlying material comes in contact with the blood-stream and fibrin is deposited in irregular masses, forming the vegetations which are of very varying size and prevent closure of the valves or obstruct the blood-stream. In these vegetations organisms may be found, usually in larger numbers in the superficial portions. The degree of reaction varies greatly, and may be very slight in the endocarditis found in those dead of some chronic disease. Organisms are not always found, especially in more chronic forms, and toxins may have caused degeneration in the endothelium. At a more advanced stage vegetations are found which are usually sessile but sometimes pedunculated, forming a string or garland of small beads, on the auricular surface of the mitral and ventricular surfaces of the aortic valves; not on the free edges of the valves, but at some slight distance from the border, corresponding to the lines of the maximum contact of the valve segments when the valve is closed. They vary greatly in size. When the chordae tendineae are involved, the endocardium covering appears opaque and slightly raised; and in rare cases may be the seat of small vegetations.

Subsequently these vegetations gradually become absorbed, but how often without leaving permanent changes behind it is difficult to say; this is probably rare. The common result is a process of fibroid change which

varies greatly in extent. Too much stress cannot be laid on the conclusion that the gravity of this form of endocarditis lies not in the presence of vegetations — unless in the rare instances when a part is dislodged with resulting embolism—but in the slow fibrotic changes which are inaugurated and which tend to advance steadily. These changes result in various deformities of the valves. The segments may be thickened and altered or become adherent to each other. As a result the valves may no longer close the opening, or the gradual contraction may result in a narrowing of the orifice. Not infrequently these processes affect the chordae tendineae and papillary muscles with consequent thickening and shortening. This is sometimes more serious than the changes in the valves themselves. Later the valves may have an almost cartilaginous appearance, and calcareous deposits may occur.

Histologically the affection shews changes of an inflammatory nature in the endocardium, and deposit of fibrin in the form of thrombi, both white and mixed, from the blood. Sections of a small vegetation in a very early stage shew proliferation of the endothelial layer, increase of the branched cells in the subendothelial tissue, and infiltration of the layers of the endocardium with leucocytes, fibrin, and serum between the trabeculae, and a deposit of fibrin on the free surface of the endothelium. In the severe cases the myocardium shews indurative changes, which may be looked upon as due to an extension of the inflammatory process ; the fibrous septa and the lymphatic spaces being chiefly involved ; even the muscular fibres may shew changes partly due to compression, and partly to myocarditis.

From acute endocarditis must be distinguished : (*a*) Patches of red coloration of the valves, which are sometimes seen in persons who have died of an acute infectious disease. These patches are simply due to blood imbibition. (*b*) Certain deposits on the auriculo-ventricular valves, which are the remains of an embryonic condition of the valves, as pointed out by the Bernays ; and in these Luschka has demonstrated pigment particles due to old haemorrhages (Rosenbach, *loc. cit.* p. 156). (*c*) A form of endocarditis probably due to sudden disturbance of intracardiac pressure, whereby the endocardium is injured. We have here small, close, hard vegetations, firmly fixed and without adherent fibrin (Dickinson).

Symptoms.—Acute endocarditis is sometimes easy, at other times most difficult to recognise. It must be remembered that the features may be due more to associated myocarditis than to the endocarditis itself, for we are probably always safe in considering that acute endocarditis implies coincident myocarditis. In mural endocarditis there may not be any symptoms or signs to suggest the condition.

The onset is usually insidious, and there may be no symptoms from the endocarditis itself to draw attention to the heart. This is especially true in children. A rigor at the onset is rare. The subjective symptoms vary considerably with the age of the patient, the primary disease, the presence of complications such as pericarditis, and the effects of previous attacks of endocarditis. The physical signs may sometimes be absent, or

appear only when the acute process has passed into a chronic state. In
some cases symptoms are absent, and it is only perhaps when the patient
is brought under our notice in an attack of hemiplegia due to embolism
that endocarditis may be detected.

If we take the acute rheumatic as the most common form of endo-
carditis, we find in many cases no subjective symptoms to lead us to
suspect an endocardial affection ; the febrile symptoms, such as tempera-
ture, pulse, respiration, do not differ from those in cases of rheumatic
fever without endocarditis ; and it is only by the physical examination of
the heart that the existence of endocarditis is detected, although often
the persistence of the fever after the subsidence of arthritis may suggest
endocarditis. In a second group of cases the patient, who has generally
been affected with the rheumatic fever for a week or more, has suddenly
a rise of temperature without any fresh pains ; or he complains of
oppression, uneasiness, or pain over the region of the heart and palpita-
tion ; the pulse becomes small and quick, and the heart's action tumultu-
ous : in other cases, of subacute course, dyspnoea on exertion is the
only symptom complained of, yet physical examination of the chest
reveals the existence of an endocardial murmur. In children, when peri-
carditis complicates endocarditis, which it frequently does (the carditis
of Sturges), the symptoms are more pronounced and fairly character-
istic ; the breathing, with the alae nasi dilated, is hurried and laboured,
and there is great orthopnoea ; the child has an anxious look and is
somewhat cyanotic, sleep is very much disturbed, and there is generally
marked delirium. The pulse in these cases is very quick, small and
compressible, and there may be persistent vomiting. It must be noticed
that in children the joint affection in rheumatic fever may be so slight as
to be easily overlooked (see art. on "Acute Rheumatism of Childhood,"
Vol. II. Part I. p. 650), and it may happen that the only noteworthy
feature is a rise of temperature with profuse sweating, which may go on
for some time. The daily examination of the heart at first shews
nothing abnormal, but in a short time the physical signs of endocarditis
present themselves. In other cases in children, as in chorea, little
general disturbance is noticed. In general, fever is the most important
single feature.

Physical Signs.—These are sometimes very well marked, and admit
of no other explanation ; at other times they are indefinite. The
frequency of associated myocarditis, with perhaps some dilatation, must
be kept in mind. The signs of slight or moderate dilatation may
predominate.

On general inspection there may be slight cyanosis and marked visible
pulsation may be seen in the neck and above the clavicles. The visible
precordial impulse may be widespread and shew a peculiar wavy quality.
In some cases it does not seem to be uniform in all parts, and in others
the local impulse at the apex may appear vigorous. On palpation little
may be felt, and the discrepancy between the visible and palpable impulse
may be striking. Sometimes the shock of both sounds is felt.

On percussion it is usually only if there be dilatation that an increase of the area of dulness is made out. Occasionally an increase in dulness may be due to pericardial effusion; if so, the pulse is feeble, the apex-beat is not so well felt as usual, and the area of dulness has the characteristic outline of pericardial effusion. It is important as early as possible to make accurate record of the extent of cardiac dulness as an aid to the later recognition of dilatation or pericardial effusion.

The most important physical signs of valvular endocarditis are noticed on auscultation. As the mitral valve is most frequently affected, and as the fibrinous deposit is apt to prevent the complete closure of the valve, the signs of mitral regurgitation are presented; that is, a systolic murmur heard best at the apex, and conveyed towards the axilla and also towards the sternum. In a good many cases of rheumatic endocarditis, which afterwards lapse into chronic valvular disease, a systolic murmur, soft and blowing in character, is first noticed at the apex, but as a rule is better heard over the lowest portion of the sternum close to its junction with the left costal cartilages. The appearance of a systolic murmur is preceded for days by an impurity and prolongation of the first heart-sound, usually best heard at the apex, which is in itself suggestive of endocarditis. Prolongation of the first sound is the first whisper of an "approaching murmur" (Sibson, *loc. cit.* p. 493). This is probably due to the soft gelatinous deposit, which alters the first sound while the valves are still smooth and elastic. According to Sibson, we may notice occasionally, besides the mitral bruit, a tricuspid systolic murmur also; but this is not heard at the very beginning of the endocarditis. As regards acute endocarditis in children, Sturges (27) gives as the earliest physical indication : "Tumultuous, quickened, and uneven heart's action and sounds, that are changeful from day to day, especially the first; sounds reduplicated, at and above the apex (not at the base); a temporary tricuspid murmur; marked accent commencing the first sound, whether mitral or tricuspid." Occasionally, however, even in children, a loud systolic murmur may rapidly appear; this is sometimes only heard when the child lies down; in the erect position it becomes fainter and may even disappear.

In cases observed by the reviser of this article, the most rapid appearance of a murmur which was permanent occurred in less than two days. For the first twelve hours there was reduplication at the apex, then a murmurish quality became evident, which in twenty-four hours changed into a definite murmur with and following the first sound.

In acute febrile affections, such as the acute zymotic diseases, and in rheumatic fever, a systolic murmur is often heard under conditions other than that of endocarditis; therefore, when we hear such a murmur we must not conclude at once that there is endocarditis. The murmur may be due to changes in the heart-muscle, or to a change in the blood (haemic bruit). Although it is not always easy or even possible to distinguish these conditions, certain signs will help us. The pulse in myocardial affections is often quick, small, and irregular; and there are marked

dyspnoea and vertigo. The haemic quality is noticed when there is well-marked anaemia; it is heard not only over the mitral, but often also over the pulmonary and aortic areas, and is accompanied by venous murmurs in the neck, while the pulse may be dicrotic. Besides the mitral murmur, especially if the heart-muscle is weak and early dilatation of the right ventricle comes on, we may note reduplication of the second sound, accentuation of the pulmonary second sound, and sometimes also a tricuspid systolic murmur.

If the endocarditis affects the aortic valves we may find no special physical signs if the vegetations are very small; at other times we get evidence of aortic regurgitation, a diastolic bruit heard best at the midsternum; and, if there be much regurgitation, we get other indications of aortic incompetence. The reviser of this article has observed cases in which aortic insufficiency due to endocarditis appeared under observation; it was noted that the diastolic murmur was variable at first, being heard one day and gone the next, to reappear later. The collapsing character of the pulse was variable, in some being present both when the murmur was heard and when it was absent; in others it did not appear until the murmur was permanent.

The physical signs denoting stenosis of either mitral or aortic valve are very rarely to be noticed, as the narrowing results from a contraction of the valves which generally takes place as the endocarditis becomes chronic. Sansom stated that in some cases he had observed reduplication of one or other of the heart-sounds as an early sign of endocarditis; and in these cases the endocarditis was followed by stenosis rather than by regurgitation. In a patient with rheumatic endocarditis in whom the development of the signs of both mitral stenosis and insufficiency was watched, the interval from clear heart-sounds to definite signs of mitral stenosis was three months (T. M‘C.).

In the rare cases of right-sided endocarditis the signs are those of tricuspid or pulmonary regurgitation.

As regards the rate of the heart, it is worthy of note that endocarditis may be present and the pulse-rate continue normal.

Complications.—Leaving out of consideration the rare cases—which, however, mostly belong to infective endocarditis—in which there is rupture of the inflamed valve or of the chordae tendineae, the most frequent complications of endocarditis are *pericarditis* and *myocarditis*. Sibson in 161 cases of acute endocarditis noticed that pericarditis was present in 34 cases, and in children the proportion is even larger. Changes in the myocardium may be regarded as almost always accompanying endocarditis. We may suspect it if the heart's action becomes weaker, or if there are any physical signs of acute dilatation of the heart; the pulse becomes quicker, weaker, and often irregular, the apex-beat weaker, and the murmur less distinct; there is also marked dyspnoea, and the patient complains of tightness and oppression, and occasionally of pain and palpitation.

Pleurisy and *pneumonia* are occasional complications, especially of rheumatic endocarditis.

Embolic infarcts occur more frequently in infective endocarditis, and in chronic valvular diseases, than in the acute rheumatic endocarditis. In some rare cases (10) the endocarditis propagated to the aorta may produce acute aortitis, a complication which is difficult to diagnose; it produces severe paroxysmal pain behind the sternum with radiation towards the shoulder, dyspnoea, and perhaps a diastolic aortic murmur.

Course and termination are most variable; in some cases the symptoms may disappear, and the patient completely recover; in others the patient apparently recovers, but for some time looks very anaemic, and the physical signs never disappear. Or the patient may enjoy excellent health and not be aware that he has any valvular lesion till many years afterwards, when the first symptoms of want of compensation of the heart-defect make themselves felt; the length of time before these symptoms come on depends on many factors, such as the extent of the lesion, the condition of the heart-muscle, the occupation of the patient, or intercurrent diseases. In other but fortunately very rare cases, in which the valvular lesion is very severe or the myocardium very much enfeebled, the symptoms denoting failure of compensation (dyspnoea, quick, weak, or irregular pulse, oedema or dropsical effusion) may come on early after the onset of the disease. When pericarditis complicates the endocarditis the patient may be years without any serious trouble, till compensation begins to fail.

The duration of an attack of simple endocarditis is most variable. In some patients the symptoms may continue for months with more or less irregular fever. Death may take place during the acute stage from the presence of complications such as pericardial effusion, myocarditis, pneumonia, embolism, or in some rare cases from hyperpyrexia; or some added infection may convert the rheumatic into infective endocarditis.

In children, in whom the physical signs are usually well pronounced, and pericarditis often present, all the signs may completely subside, and a restoration to complete health take place; in most cases, however, the child apparently recovers and may enjoy good health for many years in spite of the presence of the signs of valvular disease; yet eventually, either without apparent cause or on the appearance of some incidental disease, he manifests the subjective and objective signs of valvular disease. In some few cases belonging to the group called active carditis by Sturges (27), death takes place from the associated pericarditis or from pulmonary oedema, embolic pneumonia, or cerebral embolism. The cause of death in some cases of chorea with endocarditis is often very obscure, and not due directly to the endocarditis.

Diagnosis.—From what has been said of the symptoms it will be clear that the diagnosis, though easy in some cases, is occasionally impossible; in many cases, indeed, the endocarditis can only be suspected. When no murmur is heard over the precordial region we can only suspect endocarditis when, say in a case of acute rheumatic fever, there are signs of disturbed cardiac function, the heart-sounds become veiled

and impure, and the patient complains of palpitation or oppression. When a murmur is heard over the region of the heart we have to distinguish between an exocardial and endocardial murmur, and if endocardial whether it is due to endocarditis.

The exocardial murmur, which is occasionally difficult to discriminate, especially in children, is a *pericardial friction*-sound; but the character, the rhythm, the situation, the variability of the murmur, the direction in which it is propagated, and some other points will help us to distinguish pericarditis from endocarditis. Thus in pericarditis pain and oppression are often noticed, and a double murmur is heard which does not take the place of the heart-sounds, nor is the double murmur synchronous with them; the murmur may have the character of a hard or soft friction-sound; it is heard usually over the right ventricle, though it may be audible with less intensity near the apex; it appears to be superficial, is localised over a small area, is not propagated either to the axilla or along the sternum, and is variable within short periods of time. Occasionally the rub may be felt when the hand is placed over the precordial region. If there be much effusion the signs of this will be found. An exocardial murmur may be pleuro-pericardial. As a rule there is no difficulty in distinguishing this from pericardial friction and from an endocardial murmur, generally the rub extends towards the left beyond the limits of the heart. Another exocardial murmur, cardio-respiratory, may sometimes be heard above the apex-beat towards the left; it varies in intensity, is usually systolic, with inspiration, and varies with change in position of the patient.

Having eliminated the exocardial murmurs, we have yet to determine whether the murmur be due to endocarditis or some other cause; and, if due to endocarditis, whether recent or old, benign or infective. The chief points of distinction between the haemic murmur and the murmur produced by the dilatation of the heart, and by endocarditis, have been given on pp. 269, 270. As a rule there is no difficulty in distinguishing recent from old endocarditis; we have to take into account the history of the case—whether there have been previous attacks of rheumatism or chorea, or of some of the other diseases followed sometimes by endocarditis; or whether the patient has suffered from dyspnoea on exertion or oedema of the feet. The presence of secondary changes in the heart due to chronic valvular disease, such as hypertrophy or dilatation, is of great help; but we have to bear in mind that dilatation of the right heart may come on occasionally in acute endocarditis, and that a previous attack of rheumatic endocarditis favours the recurrence of such attacks, should the patient suffer again from acute rheumatism; thus we may have an acute endocarditis implanted on an old one.

The production of murmurs from myocarditis, dilatation, fever, and anaemia has always to be excluded. Not infrequently time is necessary for this, and only the subsequent condition may determine whether or not endocarditis was present. A murmur which appears early is likely to be due to endocarditis as the other conditions are present later.

The discrimination of rheumatic or benign from infective endocarditis was considered in the article on the latter disease (Vol. I. p. 919).

Prognosis in acute endocarditis is sufficiently evident from what has been stated concerning the course and termination of the disease. Death during the acute stage is generally due either to the severity of the primary disease—be this rheumatism, chorea, or an infective fever—or to some complication, such as myocarditis, pericarditis, or pneumonia; in some rare cases symptoms of dilatation of the right side of the heart with venous stasis, shewn by dyspnoea, dropsy, and irregularity of the heart's action, may come on and lead to death. A very large majority of patients recover from the acute attack, remain well for years, but become the subjects of chronic valvular disease; and this may occur in patients in whom the murmur had disappeared for a time; lastly, in some few instances complete and permanent recovery takes place. The previous condition of the heart is of great importance. When acute endocarditis occurs in persons already affected with valvular disease the prognosis is more serious; for often the fresh endocarditis is of the infective or malignant kind; or, even without this, the fresh deposit may lead to embolism or, by increasing the weakness of the heart, hasten the downward course of the disease. The valves affected influence the outlook; aortic is more serious than mitral endocarditis, and implication of both valves graver still.

As regards the distant prognosis, the age, general health, extent of involvement, and especially the matter of prolonged rest must all be considered.

Treatment.—*Prophylaxis.*—As acute endocarditis is associated most frequently with acute rheumatism, our attention must be directed to prevent the occurrence of this malady in persons with a family or personal proclivity to the disease; such persons should wear flannel next to the skin, avoid living in damp houses and in districts where clay forms the subsoil and rheumatism abounds, and avoid as much as possible sudden changes of temperature. Careful attention should be paid to the condition of the throat. If there be recurring attacks of tonsillitis, or adenoids be present, proper and thorough treatment should be carried out. As the focus of infection is frequently deep in the tonsils, removal of them should be thorough and by dissection; merely cutting off the projecting part is not enough.

When a patient has acute rheumatic fever, can we by speedy and proper treatment prevent the occurrence of endocarditis? This question has been the subject of many discussions, especially since the introduction of the salicylates, but there is abundant evidence that the cardiac affections are not warded off by their use. Some maintain that the treatment with large doses of alkali, combined with absolute rest in bed, has a more protective effect against the cardiac complications than the salicylates or salicin; and many now use this combined treatment. So far the prophylactic treatment has had but little success; yet it is most important that every case of acute rheumatism in adults, and still more the various

modified and less pronounced forms in children, should be treated at once by rest in bed, with complete repose and appropriate medicine (alkalis and salicylates). It is only in a comparative minority that we find endocarditis developing in patients with rheumatic fever *after they have been put to bed*; this emphasizes the importance of absolute rest as a preventive measure.

Local Treatment.—Venesection, recommended by Bouillaud and his school, and extensively practised for years, need only be mentioned as of historical interest. The application of a few leeches to the precordial region, especially in young and plethoric subjects, is sometimes of value. The local application of cold in the form of the light ice-bag is the most useful local measure. It may be applied for a short time at first and the periods rapidly lengthened until it is kept on continuously. It reduces the fever, diminishes the frequency of the pulse, calms the action of the heart, and relieves such subjective symptoms as pain and oppression. It is well to apply flannel next to the skin and the ice-bag over the flannel. As a rule it is tolerated quite well and comforts the patient.

Other local remedies used are blisters, sinapisms, and tincture of iodine. Large blisters have often been recommended as derivatives, and recently Dr. Caton has spoken favourably of repeated small blisters. Blisters are often applied both in endocarditis and endo-pericarditis, with relief of some of the subjective symptoms ; but they do not appear to influence the disease very much.

General Treatment.—With the appearance of the first symptoms of endocarditis some physicians recommend the administration of larger doses of alkalis and suspension of the salicylates, which have a depressing effect on the heart ; others see no objection to a continuance of the salicylates, unless signs of failure of the heart or of myocarditis appear ; others again prefer to give salicin, which has a much less depressing effect. It is difficult to see how any drug can influence the process in the endocardium. Iodide of potassium has been given at a later stage of endocarditis to hasten the absorption of the deposits.

If the focus of infection is recognised (*e.g.* the tonsils) and cultures can be obtained, the use of vaccins may be tried. This has been done in several cases at the Johns Hopkins Hospital, but as yet without any striking results. The use of serum has not proved of great value.

More essential than the medicinal treatment is the general management of the patient. The first essential is rest, which should be absolute, both bodily and mental. The time must depend on the individual patient and should certainly be for weeks, often months, especially in children, three months being a fair average. A full explanation of the reasons for this should be given to an adult or to the parents of a child. Too much stress cannot be put on this ; a few weeks' rest may add years to the patient's life. The condition of the heart with the effect of slight exertion on the heart rate and rhythm must be taken into account when allowing the patient more liberty. Convalescence in any case must be prolonged, and if there is the slightest doubt as to the wisdom of more

exertion, it is better to defer it.   The diet should be light but nutritious, and, unless the heart shews signs of failure, it is advisable to avoid stimulants altogether.

Certain drugs may be indicated by certain symptoms and under certain conditions.   If there be much pain and restlessness small doses of morphine may safely be given.   Antipyretics are only indicated when the temperature is high and the pulse very quick.   Phenacetin (gr. iii. to v.) is preferable to antipyrin or sodium salicylate.   Digitalis is not required unless the pulse becomes quick and small, or irregular ; the tincture of digitalis or digitalin may be given when signs of cardiac failure appear.   Besides this drug we may give strychnine, ammonia, brandy, and ether under the above conditions.   When there is much dyspnoea and cyanosis, inhalations of oxygen will be found useful, especially in children.   In cases in which the pulse is quick but full, and in which the heart's action is good, digitalis had better be avoided.

For the anaemia, which often persists long after the acute symptoms have passed off, preparations of iron are given with arsenic ; the latter drug often appears to have a better effect than the iron preparations. Convalescents from acute endocarditis should be sent for some weeks into the country or to the seaside, a dry bracing climate being preferred.   Those cases of rheumatic endocarditis which assume a malignant type, which run a long and protracted course, and in which fever persists, rigors and haemorrhages appear, and further complications (septic pneumonia, embolic abscesses) arise, require the same treatment as cases of infective endocarditis, to which class indeed they belong.

<div align="right">J. Dreschfeld, 1898.
T. M'Crae, 1909.</div>

<div align="center">REFERENCES</div>

1. Caton.   " The Treatment of Acute Rheumatic Endocarditis," *Lancet*, 1895, ii. 399.—2. Cornil.   *Les Bactéries*, 1885.—3. Coupland.   *Trans. Path. Soc.*, London, 1873, xxv. 69.—4. Dickinson, W. H.   "On the Pathology of Chorea," *Med.-Chir. Trans.*, London, 1876, lix. 4.—5. Donkin.   *Diseases of Children*, London, 1893, 302.—6. Edwards.   *Lancet*, 1850, i. 673.—7. Hanot.   *Bull. Soc. anat.*, Paris, 1874.—8. Lancereaux.   *Gaz. méd. de Paris*, 1868.—9. Lees, D. B.   *Lancet*, London, 1893, ii. ; and *The Treatment of some Acute Visceral Inflammations*, 1904.—10. Leger. *Thèse de Paris*, 1877.—11. Leyden.   *Deutsche med. Wchnschr.*, 1895.—12. Litten. *Centralbl. f. klin. Med.*, 1897.—13. Mackenzie, S.   *Trans. Internat. Med. Congress*, London, 1881.—14. McCollom.   *System of Medicine* (Osler and M'Crae), 1907, ii. 352. —15. M'Crae.   *Journ. Amer. Med. Assoc.*, Chicago, 1903, xl. 211, and *Am. Med.*, Phila., 1903, vi. 221.—16. Marshall.   *Johns Hopkins Hosp. Bull.*, 1905, xvi. 303. —17. Osler.   *Chorea*, 48.—18. Poynton.   *Quart. Journ. Med.*, Oxford, 1908, i. 228.—19. Reymond.   *Dictionnaire encyclopédique des sciences médicales.*—20. Roger. *Arch. gén. de méd.*, Paris, Dec. 1866 and Jan. 1867. — 21. Rosenbach.   *Die Krankheiten des Herzens*, 1893, 160.—22. Rosenstein.   *Path. und Therapie der Herzkrankheiten*, 1893, 69.—23. Sansom.   *Lettsomian Lectures*, 18.—24. Sibson. Reynolds's *System of Medicine*, vol. iv. 461. — 25. *St. Thomas's Hosp. Rep.*, statistical tables of.—26. Sturges, O.   *Chorea*, London, 1881.—27. *Idem.   Brit. Med. Journ.*, 1894, i. 565.—28. Thayer.   *Journ. Amer. Med. Assoc.*, Chicago, 1906, xlvii. 1352.—29. Thorel.   *Ergebnisse der allgemeine Pathologie*, u.s.w., Lubarsch und Ostertag, neunter Jahrgang, I. Abt., 1903.—30. West, C.   *Diseases of Infancy and Childhood*, 7th. ed., 1884.

<div align="right">T. M'C.</div>

CONGENITAL DISEASES OF THE HEART

By Laurence Humphry, M.D., F.R.C.P.

Synopsis

Section I

Defects in the septa of the heart.
Stenosis and atresia of the pulmonary artery.
Stenosis and atresia of the aorta.
Transposition of the primary arterial trunks.
Premature closure or patency of the fetal passages.
Irregularity in the number or form of the valves.
Anomalous septa.
Misplacements of the heart.
Deficiency of the pericardium.

Section II
(*Causation*)

Fetal endocarditis.
Mal-development.
Development of normal heart.
Mode of formation of septal defects, Stenosis, Atresia, and Transposition of the Pulmonary artery and of the Aorta.

Section III
Symptoms.
Cardiac signs.
Duration of life.
Causes of death.
Treatment.

The subject of malformation of the human heart is one of great interest, and has attracted the attention of medical observers since the beginning of the last century, but in more recent years these anomalies have been subjected to a thoroughly scientific investigation. The earliest observations consist for the most part of descriptions of morbid specimens, which are scattered through various periodical publications. From time to time these have been collected together, and have formed the subject of dissertations or lectures. One of the first of these was a dissertation by Meckel in 1802, a descriptive account drawing attention to the curious resemblance presented by some of the monstrosities to the hearts of reptiles, amphibians, and crustaceans. Chapters on the subject also appear in various works by Corvisart, Laennec, Hope, and others. A special essay by Farre in 1814, and a series of lectures by Norman Chevers in 1851 on *Morbid Conditions of the Pulmonary Artery*, drew particular attention to the very frequent anomalies of this vessel. In 1855 Dorsch insisted on the importance of fetal endocarditis as a determining element in the causation of these abnormalities, a hypothesis which became too one-sided in its application.

Peacock, in 1855, was the first to issue a systematic treatise on the subject, a work which is stamped throughout with the most accurate observation. A new edition of the same work appeared in 1866, and in the preface Peacock reminds us that it is but recently that attempts have been made to reduce the different forms of irregular development to any scientific arrangement, or to explain their nature and mode of production. In the classification of malformations of the heart he is guided partly by

the period at which the development of the organ becomes arrested or perverted; partly by the degree of impediment to the circulation which such deviation occasions, and the consequent interference with the functions of the heart after birth.

In 1875, at Vienna, Rokitansky published his most important monograph on the *Defects of the Septa of the Heart*, in which he differed from the current views of the development of the septa, and insisted on the importance of studying the anomalies in connexion with the different stages of development. More recently, Moussous and Thérémin, in France, and Profs. Wardrop Griffith and A. Keith, in this country, have added considerably to our knowledge of the subject. A comprehensive study with copious statistics has been contributed by Abbott (1).

Section I. is devoted to a descriptive account of the commoner forms of malformation of the heart. In Section II. the mode of formation of the anomalies is explained as far as possible by reference to the processes of normal evolution. Section III. contains some of the more important phases in the life-history of the subjects of congenital heart disease.

SECTION I

SYNOPSIS.—*Defects in the septa of the heart — Complete absence of both auricular and ventricular septa — Defects in the auricular septum : Defect of the primary septum ; Defect of the secondary septum ; Patent foramen ovale—Defects in the ventricular septum : Complete defect ; Partial defect ; Defects in uncommon situations.*

Stenosis and atresia of the pulmonary artery—Stenosis and atresia of the aorta—Transposition of the primary arterial trunks—Premature closure or patency of the fetal passages—Irregularity in the number or form of the valves—Anomalous septa—Misplacements of the heart— Deficiency of the pericardium.

COMPLETE ABSENCE OR VERY IMPERFECT INDICATION OF THE AURICULAR AND VENTRICULAR SEPTA.—The heart consists of two cavities, an auricle and a ventricle, with a single vessel which supplies both the systemic and pulmonic circulations.

Many cases of this kind have been collected by Peacock, the specimens shewing examples of hearts in very different stages of development. One of the earliest records of this malformation was brought before the Royal Society by Wilson in 1798. The heart was contained in a sac which rested upon the surface of the liver ; the lower part of the pericardium was absent. There was a single auricle and ventricle, and one vessel which divided into two branches ; the smaller of these went to the lungs, and the other passed upwards behind the thymus gland and gave off the usual aortic vessels. There was no ductus arteriosus, and the two pulmonary veins entered the descending vena cava.

Other cases are also described, by Farre and by Forster, in which the heart retained its most rudimentary form.

Examples in which there was some division between the auricular or ventricular cavities have been not infrequently recorded ; in some the auricles are more or less divided, but there is only one orifice of communication between these and the ventricle ; in others the arterial trunk is divided into an aorta and a pulmonary artery.

DEFECTS IN THE AURICULAR SEPTUM.—**Defects of the Primary Septum.**—*Complete, or almost Complete, Defect of the Interauricular Septum.*— The auricle remains single and undivided, or there may be a slight indication of a septum in the form of a sickle-shaped membrane at the upper and hinder part. This condition is usually associated with other considerable abnormality.

Partial Defect with Open or Closed Foramen Ovale.—There may be a large defect in the lower part of the septum limited below by the upper and hinder part of the ventricular septum, while the foramen ovale is closed and may be seen above the aperture of defect ; in other cases the foramen ovale remains open. The pulmonary artery in many of these cases is wider than the normal, and the aorta may be contracted. The result of this form of defect is to leave open a free communication between the auricles and the upper part of both ventricles over the ventricular septum. A specimen of this form of defect is described by Dr. Norman Moore. The auricles were enormously dilated ; the apex was bifid like the heart of a dugong. The foramen ovale was completely closed, the septum auricularum did not meet the septum ventriculorum, and there was a large opening below it, but above the flaps of the mitral and tricuspid valves ; one part of each of these was attached to the septum ventriculorum just below this opening ; thus the auricles were in communication with one another, and each auricle with both ventricles. Prof. Wardrop Griffith (30) met with a well-marked example of this rare form of defect of the auricular septum unconnected with the fossa ovalis. There was a large aperture between the two auricles situated a quarter of an inch above and in front of the opening of the coronary sinus, apparently due to failure on the part of the primary septum to meet the endocardial cushions dividing the common auriculo-ventricular apertures.

Defect in the Secondary Septum.—The septum may be deficient either with or without remains of the primary membranous septum.

The remains of the primary membranous septum may be in the form either of a lattice-like membrane, or a pouch-like sacculation which protrudes into the auricular cavity.

In some instances a defect is found above the foramen ovale, this latter being closed or open. A few cases of this kind are described by Rokitansky.

A case is recorded by Professor Greenfield in which there was a deficiency of a great part of the upper and anterior portions, and in addition a perfectly formed but widely patent foramen ovale. The auricles were enormously enlarged and the appendices elongated, the left

coming right round to the front of the heart. When opened the auricles were found separate at the lower part only, and communicated partly with one another by an opening of nearly circular shape, about one and a half inches in diameter. The upper and a considerable part of the anterior portion of the opening was formed simply by the wall of the auricle ; at the lower and more posterior part it was bordered by the septum. The upper edge of the septum was curved and thick. No ridge whatever could be discovered indicating where the septum should be attached on the upper wall of the auricle. At half an inch below the upper edge of the septum was a patent foramen ovale. On the aspect of the posterior half of the septum towards the right auricle was an extensive irregular cribriform membrane, only attached here and there to the muscular wall. It extended from the entrance of the inferior vena cava to the aperture of the foramen ovale. The foramen ovale had the normal oblique direction and the normal funnel shape, but was of unusual length. In addition to other deviations from the normal, the pulmonary artery was greatly dilated and its wall thickened, and the aorta had only two valves, and its orifice was greatly narrowed ; beyond the valves the trunk was dilated.

Two cases are recorded by Wagstaffe with openings in the auricular septum above the foramen ovale ; in one the foramen was closed, in the other open. Cases of this kind are, however, probably rare.

Prof. Wardrop Griffith has recorded many examples of auricular defect, and in a clinical lecture on a case of infective endocarditis in a malformed heart (29), discusses fully the several developmental errors which may lead to apertures between the auricles, and suggests that coalescence of the fenestrae of the primary septum during the earlier stage in the formation of the foramen ovale may be an additional cause to those commonly described.

Patent Foramen Ovale.—Complete patency of the foramen ovale is due to failure in the development of the membrane of the fossa ovalis, and is a very common condition. It may exist without any other cardiac anomaly, and may give rise to no special symptoms. In the majority of cases it is associated with pulmonary stenosis, defective ventricular septum, or other malformation.

Small canals or perforations between the membranes and muscular partitions are not uncommon, and an oblique valvular opening is frequently to be found at the margin of the fossa ovalis where the membrane has failed to unite to the ring. In infants who have survived their birth only by two or three months the opening is normally in the form of a slit ; but it may persist through life, and is of no clinical significance.

Redundancy of the fossa ovalis has been noted by Prof. Wardrop Griffith (30) in a case in which there were no signs or symptoms of disease ; the fossa ovalis was very deep, with well-defined margins.

DEFECTS IN THE VENTRICULAR SEPTUM.—**Complete Defect.**—The heart consists of three cavities ; the auricles are divided by a more or less

complete septum, and there are generally two auriculo-ventricular orifices. The ventricle is either wholly undivided, or there may be a slight indication of a rudimentary septum at the lowest part of the cavity. The common arterial trunk is usually divided into an aorta and a pulmonary artery.

In the cases, described by Peacock, of complete defect of the ventricular septum, the aorta and pulmonary artery were more or less abnormal, being either stenosed or transposed ; although in one instance the position was natural and the orifices somewhat dilated.

Rokitansky states that complete absence of the ventricular septum is always associated with some form of anomaly of the large arterial trunks.

A specimen of this malformation was removed by myself from a girl aged sixteen, who died of pulmonary phthisis. The heart consisted of two auricles and a single ventricle, and the pulmonary artery and aorta were transposed. The septum between the two auricles was complete, but the right was nearly twice as capacious as the left. The coronary sinus opened into the right auricle, and the right auriculo-ventricular valve was tricuspid in shape ; the left auriculo-ventricular valve was somewhat irregular, the aortic cusp being puckered and contracted. The single ventricle was capacious, and presented only the merest rudiment of division in the form of a muscular projection at the posterior and inferior part. The aorta was of large size, but arose from what would be the normal position of the pulmonary artery ; the aortic valves were normal, also the openings of the coronary arteries. The pulmonary artery arose behind and slightly to the left of the aorta, the opening into the ventricle being situated between one of the segments of the tricuspid and mitral valves. The pulmonary valves were normal, but the orifice appeared somewhat smaller than usual. The ductus arteriosus was closed.

Professor A. H. Young describes a three-chambered heart removed from an adult aged thirty-five years, in whom no heart malformation had been suspected. The aorta and pulmonary artery were transposed in the usual manner, and there was hardly any trace of division between the right and left ventricles. There were two auriculo-ventricular apertures opening into the common ventricle, and there was a small aperture in the fossa ovalis.

Partial Defect of the Ventricular Septum.—Following the description given by Rokitansky, the ventricular septum may be divided into a posterior muscular septum, a membranous portion, and an anterior muscular septum, the latter being again divisible into a front and hind portion. (See pp. 296, 297.)

Defects may be seen at one or other of these sites at the base, where during fetal life the division of the cavities is last effected.

Defect in the posterior septum throws the two ventricles into free communication. A case of this kind is described by Rokitansky ; the aperture was of considerable size, and, as seen from the right ventricle

anteriorly, opened into the left ventricle, over the free edge of the rudiment of the ventricular septum ; the septum of the auricles was incomplete. The free upper edge of the rudimentary ventricular septum was sickle-shaped, and the front portion terminated above in a band which was inserted between the two arterial trunks. The pars membranacea was also defective.

Other cases of similar defect are recorded, associated with abnormal size of the right ventricle, persistent ductus arteriosus, or transposition of the right and left hearts.

Defect in the pars membranacea, or the "undefended space," is ascribed by Peacock as the cause of almost all the apertures found in the upper part of the ventricular septum, and in this he has been followed by many English writers. It is probable that in the defects of the pars membranacea Peacock included apertures which extended both in front of it and behind it. He remarks that if the interventricular septum be partially defective, the imperfection most generally occurs at the base. In this situation there exists normally, in the fully developed organ, a triangular space in which the ventricles are separated only by the endocardium and fibrous tissue on the left side, and by the lining membrane and a thin layer of muscular substance on the right. Laterally it is bounded by the attachments of the right and posterior aortic cusps, and its base is formed by the muscular substance of the septum. The dimensions of the space vary with the size of the heart, but ordinarily in the adult the sides may be estimated at about seven Paris lines, and the base is somewhat wider. When the lower part of the space is perforated, the left ventricle and origin of the aorta communicate with the sinus of the right ventricle, but if the defect be situated farther back, towards the angle of attachment of the valves, the communication may be between the left ventricle and the right auricle.

The anterior part of this opening would therefore correspond with an aperture due to defect in the hinder part of the anterior septum as described by Rokitansky.

An aperture confined to the "undefended space" would be of very small dimensions, but it may be defective in conjunction with defects of either the posterior septum or of the hinder portion of the anterior septum.

Complete Defect of the Anterior Septum.—Several instances of this condition are described and figured by Rokitansky. In these the whole of the anterior portion is deficient, throwing both the ventricles and the origin of the arterial trunks into communication.

The majority of these cases shewed in addition either transposition or some anomaly in the position of the large arterial trunks. In others there was stenosis or atresia of the pulmonary artery. The foramen ovale was usually open or only partially closed.

Defect of the Hinder Portion of the Anterior Septum.—This is a very common form of deformity, and like the rest is usually accompanied by malformation of other parts, with abnormality of the origin of the arterial trunks, or with stenosis or atresia of the pulmonary artery. An

aperture in the hinder part of the anterior septum places the two ventricles in communication, the left ventricle and origin of the aorta with the sinus of the right ventricle.

Defect of the Foremost Part of the Anterior Septum.—By this malformation the origins of the arterial trunks are placed in communication ; the condition is no doubt rare. The aperture is seated in front of the pars membranacea septi and just below the anterior segment of the aortic valve.

Dr. Sidney Coupland describes an excellent example of this defect. The heart was hypertrophied, both ventricles enlarged and the walls thickened. On laying open the conus arteriosus the upper part of the ventricular septum was seen to be perforated by a crescentic aperture, which was of sufficient size to admit a No. 12 catheter, and was seated on the posterior wall of the conus, immediately below and to the right of the posterior segment of the pulmonary valves. Viewed from the left ventricle the aperture had the following relations :—Its shape was more oblong than it appeared on the right side, and it occupied the fleshy part of the septum about a quarter of an inch from its union with the anterior wall of the ventricle. The upper margin was formed by the bulging segment of the anterior, sometimes called right aortic cusp, from above which issued the right coronary artery. The orifice was thus placed between the anterior or right and the left posterior or left valve cusp, but in closer contiguity to the former than to the latter. There was no further malformation of the heart.

In a case described by Rokitansky there was a rounded orifice in the foremost part of the anterior septum on the left side ; it was situated beneath the right aortic valve 10 mm. in front of the membranous septum : seen from the right side, it appeared in the conus 13 mm. in front of the membranous portion just below the right pulmonary valve. The apex of the heart was bifid, the aorta displaced to the right, and the position of the pulmonary valves was altered. The aorta and pulmonary artery were of normal calibre.

Other cases are recorded by Drs. Rolleston (73), Hale White, and Prof. Wardrop Griffith. In a specimen shewn by the latter the aperture was just below the anterior segment of the aortic valve, and was separated from the front of the pars membranacea by a band of muscular tissue. As seen from the right ventricle the aperture was separated from the under aspect of the anterior right pulmonary flap by an aneurysmal dilatation of the anterior segment of the aortic valve. Aneurysmal pouches are not uncommon in this position, owing, Prof. Griffith thinks, to want of support of the muscle of the conus arteriosus (35).

Defects in Uncommon Situations.—It is rare to find apertures of communication between the ventricles elsewhere than at or near the base of the septum.

Rokitansky records a case in which, with other malformation, there was a perforation near the middle of the septum. Sir Dyce Duckworth describes a specimen in which there was an aperture in the septum of the

ventricles about the junction of the middle and lower thirds ; the opening was large enough to admit a crow quill, and was situated somewhat posteriorly ; the foramen ovale was pervious. Apertures in these unusual situations do not seem to admit of any general explanation.

It would be of interest to ascertain the distribution of the auriculo-ventricular bundle in cases of absent or imperfect septa.

STENOSIS AND ATRESIA OF THE PULMONARY ARTERY.—**Stenosis of the Pulmonary Artery.**—This is the commonest form of cardiac mal-formation.

In many recorded cases stenosis of the pulmonary artery is combined with imperfection of the ventricular septum, a dilated aorta communicating freely with both ventricles. Minor variations depend on the degree of stenosis, the extent of the septal defect, and the degree of displacement and dilatation of the aorta : the foramen ovale may be either patent or closed ; the ductus arteriosus is usually closed.

In a large number of these cases there is some deviation of the septum of the ventricles, so that the origins of the aorta and pulmonary artery are misplaced ; this deviation of the septum is most frequently to the left, so that the right ventricle is of large size and the aorta arises wholly or to a great extent from that cavity. In some instances the septum of the ventricles is found to be entire while the auricular septum is defective.

Atresia or obliteration of the pulmonary artery is a far rarer condition than the preceding. Several cases, collected from various sources, are quoted by Peacock ; and he records two cases which came under his own notice. An important distinction in these two cases is that in the first the ventricular septum was incomplete, while in the second it was fully formed. In the first there was obliteration of the orifice and trunk of the pulmonary artery ; the aorta arising chiefly from the right ventricle and giving off the pulmonary branches through the ductus arteriosus. The right auricle was large and its walls thick, and the foramen ovale was not completely closed by the valve, but would allow the blood to flow from the distended right auricle into the left. The cavity of the right ventricle was of very large size, and consisted almost entirely of the sinus ; the infundibular portion was reduced to a mere chink, and was entirely closed at the usual point of origin of the pulmonary artery, the trunk of which formed an impervious cord as far as its union with the ductus arteriosus ; the septum of the ventricles was imperfect at the base ; the wall of the right ventricle was extremely thick, and the left auricle and ventricle were very small in relation to the right. The aorta arose chiefly from the right ventricle, and was of large capacity so far as the point at which it gave off the ductus arteriosus, through which the supply of blood was transmitted to the lungs.

The second specimen was removed from a child which died nine days after birth. The heart was of unusual form, being broader from side to side than from above downwards. The left ventricle constituted the largest part of the organ. The two auricles communicated freely through

the patent foramen ovale. The cavity of the right ventricle was of very
small size, the outlet from the ventricle by the pulmonary artery being
entirely closed by the union of the valves at the origin of this vessel.
The pulmonary vessel was pervious down to the valves. The ductus
arteriosus was of the usual size, and passed into the aorta, forming a com-
munication between the branches of the pulmonary artery and that vessel.
The septum of the ventricles was entire. The cavity of the left ventricle
was of large size, and was separated from the left auricle by the usual
valves. The ascending aorta was large and the ordinary branches arose
at the arch. After the entrance of the ductus arteriosus the aorta
diminished considerably in capacity. The course of the blood in this
case must have been from the right auricle into the left auricle, thence
into the left ventricle and aorta, and from that vessel to the lungs by the
ductus arteriosus. The right ventricle, being thrown out of use, had
atrophied ; while the left, having to maintain both the systemic and
pulmonary circulations, was unusually capacious and hypertrophied.

A remarkable instance of this condition is recorded by Hare. It
was removed from a child aged nine months, who died cyanotic. The
right auricle was enlarged, and had only a very small communication
with the left through an opening in the foramen ovale, one-sixteenth of
an inch in breadth and one-tenth of an inch in length. On cutting into
the right ventricle it was found that the columnae carneae were fused
almost into one, and the cavity would only hold a moderate-sized pea.
The ventricular septum was perfect. The orifice of the pulmonary artery
was closed, but its trunk was in communication with the ductus arteriosus
and divided into the usual branches. The left ventricle was hyper-
trophied, and gave origin to the aorta. The unusually small opening
between the right and left auricles, the only communication between the
two sides of the heart, was remarkable in this case.

Rare cases of atresia of the pulmonary artery with transposition
of the viscera have been described by J. M'Crae and others.

In all cases of atresia the possibility of the circulation being carried
on, and life maintained, depends upon the open condition of either the
interventricular septum or the foramen ovale, and on the patency of the
ductus arteriosus.

There are some important differences in the site of the constriction,
partial or complete, of the pulmonary artery, and the nature of the con-
striction varies also.

The following forms may be recognised :—

Stenosis and atresia of the trunk of the artery.

Stenosis at the conus arteriosus.

Stenosis of the valves with or without narrowing of the trunk of the
vessel, and with dilatation of the pulmonary artery.

Stenosis of the Trunk.—The trunk and canal of the artery may be
contracted or obliterated for a greater or less extent in its course, or even
converted into a fibrous cord. The cause of this contraction is no doubt
due, in the majority of instances, to irregularity in the development or

division of the common arterial trunk, and is usually associated with other developmental defects. **Atresia** occurs whenever the deviation of the septum of the bulb is so considerable that the septum, the convexity of which is directed towards the pulmonary artery, becomes actually applied to the wall of the vessel and fuses with it as far down as its mouth. The cause of the unequal division is probably due to imperfect development of the fifth branchial arch.

Stenosis at the Conus Arteriosus.—The conus or infundibular portion of the ventricle is usually ill developed, and there is a constriction between it and the sinus of the ventricle. The degree of stenosis may be extreme, the orifice being only of sufficient size to admit a small probe. The condition is usually associated with much thickening of the endocardium and surrounding muscular tissue, with increase of the fibrous tissue ; these results being in many cases due to the impediment of the passage of the blood of some duration.

Dr. Keith (46) regards the majority of cases of pulmonary stenosis as due to defects in development of the bulbus cordis (*vide* p. 297).

Stenosis at the Valves.—When the constriction is at the valves, their free edges or adjacent parts are adherent, forming a curtain, and leaving an aperture of varying size and shape for the passage of the blood.

The valves themselves are usually irregular in number, size, or form.

The pulmonary artery is usually found to be more or less diminished in calibre throughout ; but this is not invariably the case, for in some specimens dilatation occurs in the calibre of the vessel on the distal side of the obstruction. A specimen of this latter condition of the pulmonary artery is described by Peacock. The heart weighed about nine ounces : the anterior surface was almost entirely composed of the right ventricle, which was greatly dilated and hypertrophied. The pulmonary orifice was very much constricted from disease of the valves ; the three curtains were blended together so as to form a kind of diaphragm which extended across the orifice, and protruded forwards in the course of the vessel, and was perforated in the centre by a small rounded aperture. The trunk of the pulmonary artery was of somewhat large size, and its coats were thick. The fetal passages were completely impervious. The case was an uncommon one, for with extensive disease of the valves of the pulmonary artery the heart was otherwise well formed. It must be concluded that the degree of obstruction at the pulmonic orifice must at the time of birth have been only slight. With regard to the dilatation of the trunk of the pulmonary artery combined with the stenosis, Peacock remarks that this is generally the case where the septum of the ventricles is entire, but where the septum is deficient and the stenosis at or near the orifice, the trunk of the artery is usually small and its walls thin. In a remarkable case reported by Holmes, the dilated pulmonary artery imitated an aneurysm, and the case was published as an example of distal ligature of the left carotid artery for the relief of aortic aneurysm. The symptoms were relieved after the operation ; years after the patient died of pulmonary tuberculosis and the aorta was shewn to be healthy.

In cases of obliteration of the pulmonary artery the blood is usually transmitted to the lungs from the aorta through the ductus arteriosus; more rarely from the left subclavian artery or from other branches from the descending aorta.

ATRESIA AND STENOSIS OF THE AORTA.—This may occur either alone or associated with other deformities. A case is recorded by Mr. Shattock of atresia of the aortic aperture in an infant from adhesion of the valves. The ascending aorta was much diminished in calibre, and arose from the left ventricle, the cavity of which was almost obliterated and could only hold a pea. The right side of the heart was large and the ductus arteriosus was patent (75).

Peacock mentions a case of obliteration of the aortic orifice, reported by Romberg, in a child who lived four days and was cyanosed. The right ventricle was dilated and hypertrophied, and the pulmonary artery was large. The left auricle and ventricle were very small, and there was not a trace of the aortic orifice. The foramen ovale was largely open, and the supply of blood to the aorta was conveyed from the pulmonary artery by the ductus arteriosus.

Similar specimens have been exhibited by Canton and by Hare. In these cases of atresia with complete ventricular septum, the left ventricle becomes abortive, and is almost entirely thrown out of the circulation, and they may be well compared with similar cases of atresia of the pulmonary artery in which the right ventricle becomes abortive.

Rauchfuss has collected twenty-four cases of stenosis and atresia of the aorta, with perfect ventricular septum; it appears that atresia of this orifice is less rare than a similar condition of the pulmonary artery.

Stenosis occasionally affects the left conus arteriosus, but not so frequently as the right.

Coarctation of the Aorta.—A narrowing of a part of the aorta at the ductus arteriosus is sometimes found. It is noteworthy that in the normal fetus the aorta is considerably reduced in size after giving off the large vessels, that it often presents a marked constriction at the part corresponding to the attachment of the remains of the ductus arteriosus, and that this constriction or isthmus is succeeded by a fusiform dilatation, *the aortic spindle of His.* This narrowing is rare, and complete occlusion of the aorta is very rare. The lesion is more common in the male than in the female. An abstract of 18 cases is recorded by Dr. J. Fawcett, in one only of which the aorta was completely obliterated; in Peacock's series, however, 10 out of 40 shewed complete occlusion. Dickinson and Fenton in 105 cases found 14 of complete occlusion. Of 198 cases collected by Abbott, 69 were in the newly born and 129 in patients more than one year old.

The site of the stricture is at or near the junction of the ductus arteriosus with the aorta, and the duct may be either patent or closed. The aorta may be dilated and thickened on the proximal side of the lesion and on the distal side may rapidly dilate and then narrow again,

so that the descending aorta is small and narrow throughout. Dr. Trevor has communicated to me the details of a case in a boy aged 12 years who died suddenly from rupture of the dilated arch of the aorta. The aortic valves usually shew inflammatory changes. The anastomotic connexions are chiefly maintained through some of the branches of the subclavian artery and branches arising from the distal side of the stricture of the aorta. The heart is usually enlarged, and other malformations are frequently present in the heart and elsewhere. Some observations are also made in this connexion by Mr. Shattock (76) in considering a specimen of congenital atresia of the oesophagus.

A curious case of aortic stenosis, with other defects, is recorded by Prof. Greenfield. The heart was greatly enlarged, especially the right ventricle ; the two auricles communicated freely ; the septum of the ventricles was entire. The left ventricle was somewhat hypertrophied and dilated : the aortic valve consisted of two cusps, anterior and posterior, the anterior being formed by the fusion of two. The aortic orifice was greatly narrowed, and the aorta shewed marked dilatation commencing a little beyond the valve. The ductus arteriosus was closed, and beyond its point of junction the aorta became narrowed, and then again returned to its normal size ; the pulmonary artery was dilated.

Hypoplasia of the aorta with smallness of the heart was described by Virchow in 1856 in connexion with chlorosis : more recently Beneke made elaborate measurements of the vessel at different periods of life, and found that after puberty the arteries rapidly enlarged, and the heart acquired a great increase of force. Suter, on the other hand, as the result of careful observations, fails to find any relation between the "narrow aorta" and anaemia, and concludes that the size of the aorta varies with age and sex, and that measurements made in the cadaver cannot accurately represent its size in the living subject.

For other irregularities of the aorta and vessels the reader is referred to works on Teratological Anatomy.

TRANSPOSITION OR MALPOSITION OF THE AORTA AND PULMONARY ARTERY.—Many different varieties of malposition present themselves, from complete transposition to slight aberration from the normal relative position of these vessels.

The condition of the cardiac cavities associated with complete transposition may be perfectly normal, but more constantly shews extensive derangement. In rare cases the ventricles also are transposed, and the other vessels more or less irregular. In nearly all cases the foramen ovale is found pervious to a greater or less extent, and generally the ductus arteriosus is also open. The ventricular septum may be defective, absent, or entire.

Two remarkable cases of anomaly in position of the large arterial trunks have been placed on record by Prof. Wardrop Griffith.

In one there was transposition of the thoracic and abdominal viscera in addition to malformation of the heart and vessels (35). The child lived

about four and a half months, was cyanosed, and the signs of transposition were noted during life. The necropsy revealed, first, a transposition of the thoracic and abdominal viscera ; and, secondly, a series of abnormalities in the vascular arrangements. The latter were as follows :—The heart was transposed, its apex pointing to the right, and the systemic auricle was on the left side, whilst the vestigial fold of Marshall was made out on the right. The left auricle, which was remarkably displaced, received above a left superior vena cava, and below another large vessel. The right auricle was smaller than the left, and received the pulmonary veins. The auricles opened into a common ventricle which constituted by far the greater part of the heart, as seen from the front. Passing from the left side of the base of this ventricle was the aorta ; while just to the right of this was a very slight flattened elevation exactly in the position where one would, making allowance for the transposition, have expected to find the pulmonary artery. The cavity of the ventricle was large and irregular, and imperfectly divided into two by a septum, which started below and to the left of the apex, but was incomplete above. The right ventricle formed the whole of the apex, but was much smaller than the left. The aorta arose from the upper and left side of the left ventricle, passed upwards, arched over the root of the right lung, and then descended to the right of the vertebral column. The aorta was the only vessel leading out of the ventricles, and the main stem of the pulmonary artery was represented by a fibrous cord, closely adherent to the aorta, which could be traced down to the flattened elevation of the ventricle before mentioned. The two pulmonary arteries received their blood-supply by a patent ductus arteriosus, and the lungs were further supplied with blood by the greatly enlarged bronchial arteries. The position of the left auricle was especially noteworthy in this case, having been, as it were, dislocated behind the aorta and rudimentary pulmonary artery. Prof. W. Griffith remarks that it is difficult to avoid the conviction that it may, by pressure, have prevented the development of the proximal part of the right fifth branchial arch, and thus led to an almost total absence of the main stem of the pulmonary artery.

In another specimen, described by the same author (28), there was lateral and antero-posterior transposition of the aorta and pulmonary artery. The heart was somewhat enlarged, the ventricular part being especially bulky. The two auricles were normal in most respects, but the foramen ovale was widely patent—the deficiency being above and in front of the valve, which was also defective at its upper and anterior part. On opening the ventricular cavities they were found to communicate freely with one another by a large aperture at the upper part of the septum, limited below by a smooth crescentic-rounded margin. The posterior boundary of the opening was continued up as a thin fibrous membrane, and blended with the upper part of the septal flap of the right auriculo-ventricular valve, which is separated from the orifice of one of the vessels arising from the ventricular cavity. There was thus an absence of the anterior part of the septum which is developed from the aortic bulb septum, while

the posterior part, derived, according to His, from a septum medium, was normally developed; the ventricles were not transposed. From the upper and anterior part of this common ventricle arose a vessel which arched backwards over the root of the right lung, and was continued down the back of the chest. It gave off the coronary arteries and vessels to the head and upper extremities; from behind this aorta arose another vessel from the ventricular cavity, which gave off the branches to the lungs and then joined the arch of the other large vessel. The second vessel, therefore, appeared to have the mixed characters of the aorta and pulmonary artery. The valves of this vessel formed a bicuspidate cone projecting into the lumen.

In a case of Dr. F. H. Thiele the vessels were transposed, but the ventricular septum was complete except for a minute aperture in the undefended space, and the ductus arteriosus was closed.

An unusual form of transposition of the primary vessels was found in a case by Dr. Hess. It was removed from a child eight hours old, who died with coma and convulsions. The heart was quadrangular in shape, the auricles were completely separated, and both auricles opened into the left ventricle. The left ventricle was very large, and at the upper and posterior part gave origin to the pulmonary artery. The right ventricle was a small rudimentary cavity from which the aorta arose, and which communicated with the left ventricle by a crescentic opening ten lines in circumference; apparently the sinus and infundibular portion of the right ventricle were divided by a septum; from the latter the aorta was given off, while the sinus was united with the left ventricle, from which the pulmonary artery arose.

Other forms of malposition are recorded, though far less frequently, in which the two vessels arise from the left ventricle, while the right ventricle is merely a rudimentary cavity, and has communication with the left through an aperture in the septum.

PREMATURE CLOSURE, AND PATENCY OF THE FETAL PASSAGES.— **Premature Closure of the Foramen Ovale.**—The condition is extremely rare; there are only three cases recorded by Peacock; in one the child lived thirty hours and was cyanosed, the right ventricle and pulmonary artery were extraordinarily developed, and there was no trace of the foramen ovale. In the other two cases, which were similar as to the obliteration of the foramen ovale, the right cavities were greatly enlarged, but the left were on the other hand very small.

Patent foramen ovale. See defects in the auricular septum, p. 279.

Premature Closure of the Ductus Arteriosus.—The duct may become abortive at different periods of fetal life, judging from the fact that in some malformed hearts no remains of it can be found. In such cases the pulmonary artery is usually narrow and ill-developed, owing to the small quantity of blood which circulates to the lungs in fetal life. The obliteration of the duct is probably due to imperfect development of that portion of the branchial arch, and may be one of the causes of pulmonary

stenosis. Other deformities usually coexist or supervene as the result of the premature closure of the duct.

Persistency of the ductus arteriosus is the result of failure of the normal involution which usually takes place before the fourteenth day. The vessel may be widely patent or narrow, and in the majority of cases the orifice of the pulmonary artery is stenosed or other anomalies are present. The right ventricle is hypertrophied, and the trunk of the pulmonary artery may be dilated. In a few instances the duct has remained patent without other anomalies. (See p. 305.)

IRREGULARITIES IN THE NUMBER AND FORM OF THE VALVES.—Slight defects in the semilunar valves are of comparative frequency and do not cause any symptoms; they may be due to malformation or to fetal endocarditis. The number may be reduced or increased.

Bicuspid Semilunar Valves.—This commonest form of anomaly, in which there are only two segments, affects both the pulmonary artery and the aorta. One segment is sometimes normal in size, the other, frequently the larger, appears to be the result of the union of two segments, shewing often an indication of the division between them; or the two may be of nearly equal size.

There may be only one curtain, with an indication of its division into three segments; it becomes stretched or protrudes in a funnel shape in the course of the vessel. Rarely there are two large segments with a small rudimentary one interposed.

The bicuspid form of valve has a great tendency to undergo sclerotic change, and to result in regurgitation. In the aorta it has been noted that the segments united are not infrequently those opposite the coronary orifices. In many the result is due to malformation, but endocarditis may account for some of those formed in later life, the partition between the two segments having been destroyed. When the pulmonary valve is anomalous there is usually found some other malformation, such as septal defect.

Redundancy in the number of segments more frequently affects the pulmonary artery than the aorta. The chief forms are (i.) three of nearly equal size, with a smaller one interposed between two others; (ii.) four segments of nearly equal size; and (iii.) three or four segments of nearly equal size with one or two smaller curtains interposed, and imperfectly separated from those adjoining.

The valvular anomalies due to mal-development take place at the time that the aortic bulb is transformed into aorta and pulmonary artery. Where the number of segments is deficient there is probably suppression of one of the endothelial cushions. On the other hand, when there is redundancy of the segments, one rudiment gives rise to two or more segments. This most commonly happens in the case of the external rudiment, the last to appear.

The Auriculo-Ventricular Orifices and Valves. — Prof. Symington relates a case in which, in addition to incomplete septa, there was only

one auriculo-ventricular opening with a bicuspid valve and a single arterial orifice, the aortic. The segments of the tricuspid or of the mitral valve are sometimes found united together in the form of a membranous curtain with a central triangular or circular aperture. Congenital stenosis of the tricuspid or mitral valve is probably extremely rare, and some reputed cases may have been in reality post-natal. Dr. T. Fisher, who describes congenital mitral stenosis in a male infant of 15 months, is only able to refer to four other cases. The two apertures may be affected in the same heart, and with a history of long-standing cyanosis in a young person, and in the absence of rheumatic attacks, it may possibly be of congenital origin, but such cases are open to criticism. The united and malformed cusps are very liable to become the seat of disease, and the stenosis is increased by chronic thickening of the united valve segments. (*Vide* also p. 330.)

A peculiar malformation of the tricuspid valve was found by Prof. Wardrop Griffith (25), in which the septal flap was quite isolated from the others, whilst the anterior segment was partly obscured by muscular bands passing from the uppermost part of the ventricle and adherent to the valve.

ANOMALOUS SEPTA.—The majority of cases in which supernumerary cavities in the heart are described are really due to the existence of an anomalous septum. This is most commonly found in the interior of the right ventricle, and at a site where there is normally a strong muscular band indicating the division between the sinus and the infundibular portion of the right ventricle. In well-marked cases there is a distinct resemblance to the right systemic and pulmonic ventricles of the turtle.

There is usually an aperture of communication between the middle and right ventricles, but the right ventricle has no direct connexion with the auricle. Two cases are recorded by Sir Stephen Mackenzie, in which there were, in addition to many other abnormalities, apparently three ventricles ; he remarks that the infundibulum of the right ventricle was shut off from the sinus by means of an imperfect, partly muscular septum, an exaggeration of the division of the muscular columns to which the folds of the tricuspid valve are attached.

Septa or fibrous bands are more rarely found in the auricles. Dr. Rolleston (72), Prof. Wardrop Griffith, and Prof. Sidney Martin record such anomalies occurring in the left auricle. Dr. Fowler describes a similar instance, in which there was a band attached to the septal wall and continuous with the membrane forming the fossa ovalis. He regarded this band as an overgrowth of the valve closing the foramen ovale, which had become directed by the blood-stream towards the outer wall of the auricle, and had become adherent there. Prof. Wardrop Griffith (31) suggests as a possible explanation of the band that there had been a failure in the complete amalgamation of the part of the auricle formed from the pulmonary veins and that derived from the left-hand division of the common auricle of the embryonic heart.

Moderator bands are occasionally found in the interior of the ventricles,

consisting of muscular fibres surrounded by endocardium. They not infrequently arise from the septum, and are attached to the wall of the ventricle, and in a specimen shewn by Prof. Wardrop Griffith (34) the band passed through the mitral orifice and was attached to the margin of the valve of the foramen ovale. In a case recorded by Sir William Turner the inner surface of the ventricles was almost uniformly smooth, owing to a deficiency of the columnae carneae. Prof. Wardrop Griffith (34) in discussing this band regards it as a remarkably constant structure accidentally cut in opening the heart, and suggests that it may serve a highly specialised function similar to that of the bundle of His.

GENERAL ANOMALIES.—Some of these occur in monsters which are stillborn.

External Misplacements.—*Ectopia Cordis.*—*Clefts of the thoracic wall* and fissure of the sternum may be present, so that the heart is covered only by membrane and integument, and protrudes; in other cases there is no apparent defect of the thoracic wall. There is commonly some other malformation present, such as protrusion of the abdominal viscera.

Three varieties are usually described: ectopia cervicalis, pectoralis, and abdominalis. In the first the heart is placed in the neck, in close connexion with the ramus of the jaw. In the second form there may or may not be a fissure of the parietes of the chest. In the abdominal form the organ lies below the diaphragm, and is sometimes protruded so as to form a tumour externally. In one well-noted case the heart was found to occupy the position of the right kidney, and the vessels arising from it passed through the opening in the diaphragm into the thorax.

Internal Misplacements.—*Dextro-cardia.*—Transposition of the heart is generally associated with transposition of the viscera. A few cases have been observed in which the transposition affected the heart only.

Two hypotheses have been proposed for the explanation of this anomaly. Dr. Fraser suggests that the transposition may be due to the subject having been one of twins which were developed from a single ovum, and in which dichotomy was complete. Von Baer has found that in a few instances the embryo lies with its left side directed towards the yolk, whereas the right side is normally in this position.

Meso-cardia.—The organ occupies a central position in the thorax similar to that which obtained at the earlier periods of fetal life. It usually presents anomalies in structure as well.

Bifid Apex.—Occasionally there is an indication of a fissure at the apex of the heart, following the course of the interventricular septum, and more or less dividing the apex into two, giving a resemblance to the heart of the dugong.

Deficiency of the Pericardium.—Complete absence of the pericardium is very rare except in association with ectopia cordis, or other serious anomaly. Partial defect is sometimes observed, and the only remnant of the pericardium may be found in the form of a sickle-shaped fold attached to the diaphragm which forms an incomplete sac for the heart. A specimen

was described by Bristowe, in which there was a rudiment of the pericardium at the upper part and right side of the heart. In another case, recorded by Dr. Boxall, the pericardial sac was incomplete, and death was caused by dislocation of the heart during a severe attack of vomiting. Dr. Keith (47) describes cases of partial deficiency of the pericardium; the deficiency is always on the left side, and the phrenic nerve has a distinct relation to the opening, which he regards as a patent pleuro-pericardial foramen.

SECTION II

CAUSATION

SYNOPSIS.— *Fetal endocarditis—Mal-development—Embryonic heart—Mode of formation of septal defects—Stenosis, Atresia, and Transposition of the Pulmonary Artery and of the Aorta.*

The cause of the various forms of cardiac abnormality is an interference with the normal processes of development at some particular stage of embryonic life. Thus, an arrest of development may occur in which the heart retains in great measure the rudimentary form of the stage at which its growth is arrested; or there may be some perversion or irregularity in development at some part by which distortion is produced, and which gives rise to secondary changes dependent on the primary defect.

In some cases in which the malformation has occurred at a very early date, as, for instance, where the heart consists of only two cavities, it may be impossible to detect the primary deviation from the normal. In many, however, in which the heart has been more fully developed, it is often possible to detect the primary defect, or, at any rate, to trace the sequence of events by which the secondary changes have been induced. The arrest of development has been attributed by some to maternal impressions during pregnancy, but in many cases the date of the impression does not coincide with the period of fetal life at which the arrest must have taken place.

Fetal Endocarditis has by some writers been credited with a large share in the production of different forms of cardiac malformation, probably to a far greater extent than is justified by the evidence, and many deny that it is a factor in the causation of any cases.

An attack of rheumatic fever in the mother during pregnancy, or a tendency to rheumatism in the parents, may be a cause of fetal endocarditis; but in most instances no such history can be obtained.

The chief form of inflammation of the fetal endocardium is of the sclerotic kind; the warty form is of far less frequency, although it is seen occasionally affecting the edges of the adherent and stenosed pulmonary or aortic valves. Minute projections may be found on the

auriculo-ventricular valve of newly-born children ; these have been mistaken for vegetations. They consist of nodules of translucent or firm connective tissue which usually disappear in the course of time. In others the edges of the valves, more often the mitral, are the seat of haematomas, caused by small spherical blood-extravasations projecting from the free end of the valve, and probably due to the rupture of intravalvular blood-vessels. They seem to arise either before or shortly after birth, and very soon shrink away ; occasionally they are found in connexion with a stenosed valve. In the sclerotic form the cusps are thickened and contracted, and the edges often united to those adjoining ; the chordae tendineae become thickened, and the valvular orifice much diminished in size. It is often impossible to tell whether the endo-carditis is of fetal origin, or has at a later period become engrafted upon an already deformed valve. According to Rauchfuss, fetal endo-carditis is only more common on the right side of the heart when in association with malformation, otherwise the left heart is as frequently affected.

Perversion of Development.—Interruption to the normal course of development is the cause of the greater number of cardiac malformations. This is in great measure indicated by the nature of the defect, the early period of fetal life at which the first deviation must have occurred, and by other circumstances which tend to shew that if any endocarditis is present it has been engrafted upon an already deformed valve or orifice. This view is strengthened by the observation that in a considerable num-ber of instances developmental errors are present in other parts of the body. Dr. Archibald Garrod (20) has collected a series of eighteen such cases, the associated abnormalities being of various kinds. In five of the eighteen cases fetal endocarditis was clearly present, but in three of these there were other abnormalities which were obviously not secondary to the inflammation ; in two the associated defects were of a minor kind, and fetal endocarditis sufficed to explain all the appearances. Dr. Keith (46), also in the examination of twenty-three fetuses with various kinds of malfor-mations, found in addition the heart malformed in fourteen, strong evidence against the hypothesis of fetal endocarditis in these cases. But even if malformation be regarded as the primary cause, we still remain in ignorance of the nature of the force which disturbs the natural process of evolution.

Before attempting to discuss the mode of formation of the various specimens of malformation described in Section I., it will be necessary to refer to the development of the embryonic heart. A full account would be out of the scope of this article, and attention will only be drawn to those events which help to elucidate the pathology of the malformed specimens.

Development of the Heart.—The heart is originally developed out of two lateral tubes of mesoblast, symmetrical and distinct, which coalesce, soon after the thirteenth day, to form a single longitudinal tube, which is slightly twisted upon itself. This single tube has double walls, the inner endothelial, the outer mesoblastic or muscular ; it is continuous in front

with the two primitive aortae, and posteriorly with the veins. During the third week slight constrictions become evident, which mark off the several divisions from one another. The anterior of these is the aortic bulb, the middle thicker part is the ventricular portion, and the posterior forms the auricular segment.

This tube, then, becomes bent upon itself in such a way that the venous or auricular portion comes to lie partly dorsal to, and partly behind the ventricular portion, the latter being continued forward as the bulbus arteriosus. Between the primary undivided auricle and ventricle a constriction occurs which elongates into a short flattened canal, the auricular canal, which is bounded by two lips, an upper and a lower. These lips become thickened by the formation of endocardial cushions which grow across the canal in such a way as to divide it into two passages, the right and left auriculo-ventricular orifices.

The internal division of the heart into right and left sides is effected by three septa or partitions, which appear within the cavity of the heart, and which arise perfectly independently of one another ; namely, the interauricular septum, the interventricular septum, and the septum of the truncus arteriosus.

The Interauricular Septum.— The division of the auricle precedes that of the ventricles and of the bulb. The history of the process as given by His, Lindes, and Born differs in some important respects. According to Lindes and Born, when the auricles develop they expand upwards, and a partition remains between them at the upper part, the septum primum, or septum superius. This septum increases with the continued growth of the auricles, and becomes thickened along its lower edge, and finally separates the two auricles, except under its lower edge, where the two cavities still communicate. This communication is not, as has been previously maintained, the foramen ovale, inasmuch as the septum continues to grow downwards to the auricular canal, and, by uniting with the partition in the canal, closes permanently the primary communication. According to Lindes, before the primary septum has quite reached the roof of the ventricles, certain small apertures may be noticed in it. These gradually increase in number, converting the septum into a lattice-like membrane through which the blood streams from right to left, causing the septum to bulge to the left.

The parietal portion only of the septum remains imperforate, forming a muscular frame which is especially well developed anteriorly. Finally, there is one large aperture left in the septum at its apex and anterior part, the true foramen ovale. A new septum also appears above the foramen ovale and to the right of the insertion of the primary septum, and its edge forms part of the boundary of the foramen ovale. In a human embryo 25 mm. long, the auricular septum contains numerous perforations, and in a fetus of three or four months the septum appears as a cribriform membrane supported on a muscular frame. During the fourth month the foramen ovale becomes partially closed by a fold which acts as a valve and allows the blood to pass from the right to the left

auricle, but prevents its passage in the reverse direction.   The final
closure of the foramen ovale does not take place until some time after
birth, and is one of the last events ; it is at first effected merely by the
close apposition of the valve, which projects into the left auricle, to the
margin of the aperture by the pressure of the increased quantity of blood
returning by the pulmonary veins ; at a later stage the edge of the valve
gradually coalesces with the margin of the opening, but the union often
remains incomplete for some months.

The Ventricular Septum and Division of the Truncus Arteriosus. —
The ventricular cavity becomes partially divided towards the close of
the fourth week by a fold, the septum inferius, which rises from its
dorsal and posterior wall, and the position of which is indicated externally
by a slight groove on the surface of the heart.

The formation of the aortic septum is effected by two longitudinal
ridge-like thickenings of the endothelial lining which arise from opposite
sides at the junction of the fifth branchial arch ; these encroach on the
lumen, reducing it to a slit, and then meet so as to divide the lumen into
two completely separate passages.

The septum appears first at the distal end of the truncus, and
gradually extends backwards towards the ventricles.   The septum first
appears towards the end of the fourth week, and is well advanced before
the end of the fifth week ; it has a slightly spiral course, so that the two
tubes into which it divides the truncus arteriosus are respectively dorsal
and ventral at the proximal end next to the ventricles, and right and
left at the distal end of the truncus.   Of the two tubes the one which
lies dorsally at its proximal end and on the right side distally is the
systemic trunk, the other which is ventral proximally and on the left
side distally is the pulmonary trunk ; and the same relations are retained
throughout life by the ascending aorta and the root of the pulmonary
artery.

The *truncus arteriosus* originally arises from the right-hand corner of
the ventricular cavity, and the two trunks into which it splits retain for
a time the same relations.   In other words, at a time when the inter-
ventricular septum is already partially formed, both the systemic and
pulmonary trunks arise from the right ventricle, and the left ventricle
has for a time no outlet except through the right ventricle.   The com-
pletion of the interventricular septum has to be effected in such a way
that while the pulmonary trunk is left in connexion with the right
ventricle, the systemic trunk shall be cut off from this cavity and placed
in communication with the left ventricle.   The formation of the inter-
ventricular septum is consequently somewhat complicated.   The greater
part of the septum is formed from the septum inferius, but it is completed
above, partly by the endocardial cushion at the lower edge of the inter-
auricular septum, the septum intermedium of His, and partly by the
prolongation of the aortic septum, which divides the truncus arteriosus
into systemic and pulmonary trunks.

The aortic septum grows tail-wards beyond the truncus arteriosus, so

as to project a certain distance into the ventricular cavity ; it then fuses with the free lower edge of the interauricular septum in such a way as to cut off the systemic trunk from the right ventricle, and to place it in communication with the left ventricle ; while finally the septum inferius extends so as to meet and fuse with the interauricular septum, and so completes the separation of the ventricles from each other.

Dr. Keith's description (44, 45) differs from that usually given of the development of the mammalian heart ; he considers that in addition to the sinus venosus, the auricular and ventricular divisions, there is a fourth part, namely, the bulbus cordis, and that this latter in the course of evolution has been separated from the left ventricle and aorta and completely incorporated in the right ventricle as the infundibulum of that chamber. He regards the submergence of the bulbus as a critical phase in the development of the heart during which many of the malformations are apt to occur.

Auricular Septal Defects.—From the study of the specimens of defect of the auricular septum in connexion with its development it will be apparent that apertures may exist either at the foramen ovale or in other parts of the septum. In the latter case, those which exist at the lowest part of the septum are probably due chiefly to failure of union of the primary membranous septum with the upper part of the ventricular septum and with the partition in the auricular canal, thus leaving a free communication between the two auricles and between the latter and the ventricles.

In some cases the septum may be entirely absent, the auricular cavities remaining undivided. When the growth of the secondary septum is defective there is frequently to be seen a lattice-like membrane between the two auricles which imperfectly divides them, and is due to the persistence of a portion of the provisional membranous septum which stretches across the persistent muscular frame. If absent or largely defective, it may give rise to an aperture at the upper and front part of the auricular septum ; and the completely formed foramen ovale, either closed or patent, may be found below. In other cases the persistent membrane becomes sacculated, and protrudes in a pouch-like form towards the interior of the auricle.

Defects in the Ventricular Septum.—*Normal Arrangement of Septa.*— The septum ventriculorum is divided into a posterior muscular septum, a pars membranacea, and an anterior septum, the latter being again separated into a posterior and an anterior portion ; the importance of this division is well insisted upon by Rokitansky in his classification of septal defects in the ventricle.

In the higher mammalia the normal arrangement of the septa in the fully developed heart is as follows : the cross-section of the ventricle is that of a crescent, the pulmonary artery being at the anterior extremity of the infundibular portion of the ventricle, while the posterior horn is occupied by the auriculo-ventricular orifice above the sinus of the ventricle. The internal wall is composed of two more or less distinct

parts.   The anterior is formed of oblique bundles passing from above downwards and slightly from behind forwards.   These bundles arise superiorly to the left of the pulmonary artery and pass to the superior half of the anterior margin of the ventricle.   They correspond to the false septum of reptiles.   Amongst the larger number of mammals the posterior border of this septum forms a very evident projection, or else sends obliquely a fleshy tongue or band to the external wall which accentuates this distinction.   This septum is interposed between the pulmonary artery and aorta.   The radiating fibres of the rest of the ventricle are placed between the two auriculo-ventricular orifices and the two ventricular cavities.   The external wall is covered with fleshy columns arising from the pulmonary orifice, and running obliquely from before backwards and downwards, which establish a limit between the general ventricular cavity or sinus and the infundibulum.   At the junction of these two columns with the posterior border of the septum is occasionally seen a white fibrous line or cicatrix.   If this spot is perforated by a needle the aorta is penetrated below the right sigmoid cusp.

It is supposed by Sabatier that this cicatrix is the vestige of an orifice from the right ventricle, representing the opening from this ventricle into the left aorta which is present in reptilia.   This anterior portion of the ventricular septum is muscular in structure, but immediately posterior to this it will be found thinner and membranous in character ; this *pars membranacea septi* or undefended space is more obvious in the heart of an infant than in an adult.   Along the upper line of this thinner portion is attached the internal flap of the tricuspid valve.   It corresponds to the upper border of the middle portion of the interventricular septum, and behind this again the septum is thicker and muscular in structure.   The bundle of His rises from the auriculo-ventricular node and passes over the auriculo-ventricular septum below the central fibrous body, and under the septal cusp of the tricuspid valve ; it divides into two branches, one passing into the right ventricle and moderator band, the other into the left ventricle.

Reference to the specimens of defect before described shews that apertures in the posterior portion of the septum, in the pars membranacea, or in the posterior part of the anterior septum, will place the two ventricles in communication ; while a defect in the front portion of the anterior septum will cause an aperture of communication between the two arterial trunks.   The latter defect is much rarer than the other kinds ; the aperture is situated below and in front of the right aortic cusp, and perforates the conus arteriosus just below the mouth of the pulmonary artery, and involves the fleshy part of the septum.   Dr. Keith (46, 48) and others regard this anomaly as being due to a persistence of the primitive ostium bulbi, namely, of the aperture of communication between the primitive ventricle and bulbus cordis.

In many cases where there is a defect at the pars membranacea or at the hinder part of the anterior septum, or an aperture extending into both of these regions, there is a primary defect in the development of the

arterial trunks, and the vessels are either misplaced or one of them is stenosed. Frequently there is evidence of endocarditis surrounding the aperture, and the endocardium is roughened or thickened.

Cases are recorded in which the pars membranacea has been found sacculated and bulging into the cavity of the ventricle, forming the so-called aneurysms of the undefended space, and due in a few instances to congenital weakness at the spot. In some, no doubt, endocarditis has an important share in their formation, and they are due to disease in after-life.

Stenosis and Atresia of the Pulmonary Artery. — When stenosis occurs at an early period of fetal life, towards the end of the second month or early in the third month, when the ventricular septum is well developed but not closed, and the auricular septum is forming, the right ventricle, unable effectually to discharge its contents through the narrow pulmonary artery, becomes over-filled, but is able to relieve itself by outflow over the still unclosed base of the interventricular septum, a measure which is sufficient in itself to prevent the complete closure of the septum. The right auricle in the same way, distended by the backward pressure, finds relief into the left auricle, and thus the normal course of the circulation is materially impeded. When the stenosis is considerable and interferes at a still earlier period with the emptying of the right ventricle, the growing septum becomes pushed over more and more to the left by the distension of the right side, and so prevents the proper connexion of the aorta with the left ventricle ; and in addition a constant flow of blood is established from the right ventricle into the aorta, so drawing the aortic orifice still farther to the right, and producing a widening of this aperture and also of the ascending trunk of this vessel. To such an extent may this displacement of the aorta be carried that this vessel has origin entirely from the sinus of the right ventricle, the left ventricle being left as a small supplementary sac with a communication into the right ventricle. This is in the main the explanation given by W. Hunter, and accepted by the late Dr. Peacock. It is held by some authors that the same series of events might be produced by an irregularity in the division of the bulb, in which the septum descended so as to form a wide aorta at the expense of the pulmonary artery, the aorta being naturally situated farther to the right in the earlier period of fetal life.

Dr. Keith gives a different explanation of the mode of formation of these cases, based on his views of the part played by the bulbus cordis in the development of the heart. He regards the majority of cases commonly described as pulmonary stenosis as due to an arrest of development of the bulbus resulting in various degrees of stenosis. Those cases in which there is a division in the right ventricle are due to a want of complete fusion between the infundibulum and body of the right ventricle, a constriction remaining between them.

The hypertrophy of the right ventricle in these cases is the obvious result of the large share it has to take in carrying on the systemic

circulation through the aorta. When the defect in the interventricular septum is considerable, or the communication of the right ventricle with the aorta very free, the septum of the auricles is more likely to be complete than where the reverse obtains, owing to the less degree of disturbance of the circulation through the auricles.

In atresia or complete obliteration of the canal of the pulmonary artery the obstruction may be either due to adhesion of valve segments, an impervious orifice, or obliteration of the trunk of the vessel as far as the ductus arteriosus. The primary defect may occur in early fetal life before the ventricular system is completed, or later, when the cavities have been separated. In the former case, as in stenosis, the right ventricle retains its communication with the aortic orifice, and is the main agent in carrying on the systemic circulation, while the left ventricle remains small and atrophies. When the obliteration of the pulmonary artery occurs after the completion of the ventricular system, the right ventricle becomes almost abolished and the right auriculo-ventricular aperture diminished in size. The left ventricle, on the other hand, becomes enlarged, and its walls much hypertrophied, as it has to carry on both the systemic and pulmonary circulations.

In almost all these cases the blood is carried to the lungs by the pervious ductus arteriosus. The foramen ovale is occasionally closed when the ventricular septum is imperfect, but is necessarily open when this septum is complete. Of thirty-four cases collected by Peacock, in eight only was the ventricular septum completed, and all these latter died a few months after birth.

In all cases of atresia of the pulmonary artery the possibility of the circulation being carried on depends upon the open condition of either the interventricular or the interauricular septum, and the patency of the ductus arteriosus.

Atresia, like stenosis, is probably due to an abnormal division of the bulbus arteriosus. Atresia occurs whenever the deviation of the septum of the bulb from the normal arrangement is so considerable that the septum, whose convexity is directed towards the pulmonary artery, becomes actually applied to the wall of that vessel and fuses with it as far down as its mouth.

Stenosis and Atresia of the Aorta.—When the constriction occurs before the completion of the ventricular septum, the narrowing of the aorta must occasion the blood to accumulate in excessive amount in the right ventricle, since both aorta and pulmonary artery communicate originally with this cavity. This repletion of the right ventricle must cause a corresponding repletion of the right auricle, and a distension and enlargement of the passage of communication between the two auricles. If, however, development has proceeded as far as closure of the passage through the ventricular septum, and limitation of the aorta on the side of the right ventricle, the condition of repletion would be confined to the cavities of the left heart, and would occasion enlargement in them also.

In atresia of the aorta the left ventricle becomes abortive and is almost entirely thrown out of the circulation, as happens in the case of the right ventricle in atresia of the pulmonary artery.

Transposition or Malposition of the Aorta and Pulmonary Artery. —The condition of the cardiac cavities associated with transposition may be perfectly normal, but more commonly shews extensive derangement.

The explanation of these deformities must be found in connexion with an abnormal division of the bulbus arteriosus, and the development of the complete septum between the arterial trunks.

The torsion of the axis which takes place during the first seven weeks has a very important bearing ; for any departure from the normal, or a failure in bringing the arterial bulb into due relation with the anterior segment of the interventricular septum, is the direct agent in the causation of malposition or transposition of the great arterial trunks. It is probably during the sixth, seventh, or eighth week that these abnormalities first begin. The union of the forked septum, which grows down the arterial bulb from above, with the upper and fore part of the interventricular septum determines the exact relation of the opening of the two arterial trunks to one another, and the slightest deviation will derange the relation. It should be observed also that the bulbus arteriosus originally communicates with the right ventricle, and that it becomes divided into an anterior pulmonary artery and a posterior aorta, at which stage both the large arterial vessels belong to the right ventricle.

The left ventricle would be quite destitute of way of issue, did not the ventricular septum remain permanently open as the aortic orifice. At this period the left ventricle pours its blood into the right, whence mixed blood is driven into both arterial trunks.

Section III

Symptoms and Physical Signs.—A child suffering from congenital malformation of the heart is weakly, difficult to rear, and generally presents at birth, or soon after, signs of derangement of the circulatory system. Lividity, of a bluish-violet tint, affecting especially the face, hands, feet, and the visible mucous membrane, is apparent.

The respiration is often laboured, and paroxysms of difficult breathing may occur from time to time. These are apt to be exaggerated by screaming, struggling, suckling, or exposure to cold air. The extremities are cold and the terminal phalanges of the hands and feet may be clubbed.

From observations made by Farre and Peacock the bodily temperature is not lower than normal, but Henoch and others record considerable lowering of the surface temperature, although normal in the rectum.

Convulsions and cerebral seizures are frequent and often fatal. In a case observed by myself the child was liable to attacks of prolonged unconsciousness. These usually occurred once or twice in the week after

a meal, lasted for several hours, and recovery took place without any ill effect; the attack was accompanied by much increase of the cyanosis.

Paroxysms of dyspnoea, in which the cyanosis is greatly intensified, are described by D'Espine and Mallet and also by Variot. Convulsive seizures may be induced, and the attack is often followed by severe exhaustion.

The onset of symptoms is variable; these may be obvious from the first, or there may be no evidence of anything wrong with the child until a year or more after birth, when perhaps the onset of some accidental affection unmasks the latent defect. The earliest and most definite symptom is cyanosis.

Cyanosis.—This is present in about 90 per cent of these cases, hence the origin of the name morbus caeruleus. It is most prominent in cases of transposition, and in atresia and stenosis of the pulmonary artery, whereas in defects of the auricular septum and in patent ductus arteriosus it is usually absent; it is also sometimes present in association with the trilocular heart.

The pathology of cyanosis in congenital heart disease has from early times occasioned much discussion, and divers explanations have been brought forward to account for it. The hypotheses ordinarily adduced attribute it to intermixture of the arterial and venous blood, or to extensive venous congestion. The former of these is amply negatived by the observation that in many cases of single ventricle no cyanosis has been observed; and that cyanosis may exist without any admixture of the blood-currents. The admixture hypothesis has been attributed to William Hunter by Peacock and other writers. Reference, however, to Hunter's cases of congenital malformation does not confirm this interpretation. He does not even mention the admixture of the blood as the cause of the cyanosis; but after remarking on the small quantity of blood which reached the lung in two cases of pulmonary stenosis, he says that, as the carnation tint of complexion depends on the florid colour of the blood, the dark or grey complexion in these cases corresponds particularly with the observation of the latest philosophers that the blood takes its bright hue in the lungs from respiration.

The venous congestion hypothesis, advanced by Morgagni, and ably supported by Stillé in America, has been most widely accepted, but cannot be said to cover the whole field.

It is probable that there are other factors which combine with venous stasis to produce the peculiar discoloration. The possibility of deficient aeration of the blood through the vessels going to the lungs must be taken into account. Dr. Lees regards this as the essential cause of cyanosis, and estimates that the amount of cyanosis is a direct measure of the extent to which aeration of the blood has been hindered. It must also be noted that it is mainly in cases in which obstruction to the circulation has existed before birth, or long before the full development of the circulatory system, that the cyanosis occurs. The cyanosis may be partly explained by hyperglobinaemia; that which is constant may be due to high red

blood counts ; that which only occurs on exertion may be due to deficient aeration. The condition of the integuments will materially affect the colour ; where the patient is emaciated and the skin is thin the peculiar purple or black tint is frequently observed ; on the other hand, when the body is well nourished, or the skin oedematous, the colour is of a deep rose tint and less intense.

The direction of the blood-current is of much interest in malformed hearts, and will be determined not only by the apertures in the septa and by the abnormal condition of the orifices, but mainly by the pressure of the fluid in the several cavities.

Haemorrhages.—In 1811 Nasse pointed out that the subjects of cardiac cyanosis were liable to haemorrhages, and in 1815 Meckel from consideration of 77 cases of congenital morbus cordis agreed with Nasse in considering that the haemorrhage was due to malformation of the blood. Peacock and Rauchfuss referred to the occurrence of haemorrhages in congenital heart disease, but little attention has been paid to it of late years, although Lee Dickinson in 1895 wrote a paper on the subject.

In the cyanosis of congenital heart disease there is commonly a high red count and an increase in the amount of haemoglobin. Toeniessen first observed the condition of the blood in congenital pulmonary stenosis ; in one of his cases the red cells were 7,540,000, and in another case 8,820,000. Banholtzer, in a case of pulmonary stenosis with cyanosis and clubbing, found 160 per cent of haemoglobin and 9,447,000 erythrocytes. Dr. G. A. Gibson (23) was one of the first in this country to draw attention to this condition of the blood. In 13 cases examined by Townsend the red count varied between 5,600,000 and 11,800,000. Banholtzer found the specific gravity of the blood in his case to be 1071·8 instead of 1056-1060. Dr. Lloyd Jones observes that in the newly-born child the specific gravity of the blood is very high (about 1067) ; and he has made the same observation in cases in which the foramen ovale had never closed, and in which the fetal condition of the circulation remains.

The clubbing of the digits consists in a drum-stick enlargement of the terminal phalanges of the fingers and toes, with often a claw-like appearance of the nails. It is usually later in its appearance than the cyanosis, but may be present when cyanosis is absent. It is remarkable that whilst clubbing of the fingers is common in congenital heart disease, Marie's sign-group or osteo-arthropathy has only once been recorded (Batty Shaw and Cooper). The pathogenesis of clubbing of the fingers is discussed in a special article in Vol. III. p. 67.

Cardiac Signs.—The detection of cardiac malformation by the physical examination of the heart is usually not difficult ; but a diagnosis of the exact form of anomaly must in many cases be impossible.

In some it is possible to arrive at a fairly close decision as to the existing conditions. On *percussion* the heart will usually be found enlarged, with indications of hypertrophy and dilatation of the right ventricle and auricle ; the impulse is powerful, displaced outwards, and visible over a large area, and there may be some prominence from yielding

of the parietes in the precordial region.   On *auscultation* there is commonly to be heard a loud, long, systolic murmur, which can be traced with varying intensity over the whole of the precordial region, over the back of the chest, and is conducted widely in all directions.   These may constitute all the cardiac physical signs, and it would be impossible upon these to make an exact diagnosis, inasmuch as they have been found in the most diverse forms of malformation.

Stenosis of the Pulmonary Artery.—There are certain signs which enable us to predict this anomaly with a great measure of certainty.   In many of these there is to be felt on light palpation, at about the second left interspace, a fine thrill, systolic in time ; it may be appreciable over a considerable part of the precordial area, but is most marked at the upper part ; an impulse can often be felt below the xiphoid cartilage ; on percussion the dulness extends beyond the right border of the sternum ; on auscultation a loud blowing murmur, systolic in time, is also present, and is to be heard louder at the left base than elsewhere.   With regard to the presence or absence of a thrill, Abbott (1) in an analysis of 75 " primary " cases concludes that a thrill is frequently present when the interventricular septum is entire and also when a defect of that septum coexists with a widely patent foramen ovale ; when the interauricular septum is closed and the interventricular septum is open a thrill is rare, or, if present, may be due to the septal defect.

The character of the second sound at the pulmonary cartilage is somewhat variable.   In many cases it is feeble and faint ; in a few cases which have come under my observation it has been loud and ringing.   This ringing sound has attracted the notice of other writers, but its significance has not been ascertained.   Dr. A. E. Garrod reports two cases in which this peculiarity of the second sound was observed, but there was no autopsy. Peacock regarded the accentuated sound at the base as produced by the aortic valves, this vessel being often unusually large.   On the other hand, it has been suggested that this sign indicates obstruction at the conus arteriosus.   The sign is probably not distinctive of the particular seat of obstruction, but it may be due to dilatation of the pulmonary artery immediately distal to the stenosis and a patent ductus arteriosus.

Coarctation of the aorta may in some instances be recognised during life.   In a case of complete occlusion of the aorta in a man aged twenty-nine, recorded by Dickinson and Fenton, the special signs were those of great enlargement of the heart, a faint systolic murmur heard over the whole precordial region, a ringing aortic second sound, and a diastolic murmur in the aortic area, traceable down the back ; marked pulsation in the subclavians and in the vessels of the neck, and between the left scapula and the spine.   In a case of incomplete occlusion in a man aged forty-eight, described by Prof. G. R. Murray, the heart was enlarged, and the superficial arteries could be seen pulsating in the upper intercostal spaces in front and in the interscapular regions behind ; no pulsation could be felt in the abdominal aorta or in the tibial vessels.   A systolic murmur was audible over a large area in front, and also behind

over an area extending from the seventh cervical spine above to the ninth dorsal spine below ; a heaving impulse could also be felt on the left of the three upper dorsal vertebrae.

The chief points upon which a diagnosis may be based are—enlargement of the heart, the presence of marked pulsation of the arteries of the head, neck, and arms, or evidence of anastomotic circulation between the arch and the descending aorta, and absence of pulsation in the abdominal aorta and femoral vessels. The murmurs present will vary according as the occlusion is partial or complete.

Patent Ductus Arteriosus. — No distinctive physical signs may be present when the channel remains small and narrow. On the other hand, when a free communication exists between the aorta and pulmonary artery, and constitutes the main anomaly, very interesting and characteristic physical signs are found. These have been well described by Dr. G. A. Gibson (22), and are as follows : a thrill is felt over the base of the heart with its greatest intensity in the left third intercostal space and extending for some distance outwards from the mid-sternum. It begins shortly after the apical impulse and continues almost to the next apex-beat. During its continuance the shock of the pulmonary cusps may be distinctly felt. Over this area of the heart an almost continuous rushing murmur with some variations in intensity is heard, beginning shortly after the first sound which is quite clear at its commencement and continuing almost to the beginning of the next sound. The murmur is heard best in the left third interspace, but may be heard all over the base of the heart. The second sound is accentuated and sometimes reduplicated. There may not be any cyanosis or other evidence of disturbance of the circulation. Such cases have been recorded by Dr. Turney ; and I have recently seen a case of congenital heart disease in which the physical signs closely corresponded with the signs observed by Dr. Gibson in several recorded cases, and in one of which a patent ductus arteriosus was verified at the necropsy. The blood stream is from the aorta into the pulmonary artery, and the channel in the duct is freely open. In the case verified the opening of the duct admitted a 12-14 bougie. By careful examination of the signs a patent ductus arteriosus may be distinguished from pulmonary stenosis, but a differential diagnosis will also have to be made from a communication between the aorta and pulmonary artery, · the result of aneurysm.

A precise diagnosis of imperfections in the septa is not possible. In these cases a blowing systolic murmur is commonly to be heard over the precordia, which in defect of the auricular septum may be more marked at the base than the apex.

Congenital affections of the other valves will create murmurs referable to the position of their orifices.

The diagnosis of transposition of the main vessels is impossible by cardiac physical signs. Transposition of the viscera may exist in connexion with this anomaly, and may be recognised.

As was first pointed out by Dr. A. E. Garrod (20*a*), Mongolian

imbeciles often shew evidence of cardiac malformation, and as already mentioned various congenital deformities are apt to exist in the same individual.

DIFFERENTIAL DIAGNOSIS.—There may be difficulty in deciding in some instances whether a cardiac murmur is of congenital or acquired origin.

No certain rules can be laid down, but the physician will be guided by the collateral signs, the past history of the patient, and the occurrence of any illness which would be likely to have laid the foundation of any cardiac disease. In the absence of any guidance from these records it may be noted that the murmurs of the common forms of malformation are systolic in time, that the murmur is not conducted in the manner usual in the acquired forms, and that it may have been observed in early childhood. In the more severe forms there would be evidence of much enlargement of the right ventricle, with probably some tendency to clubbing of the fingers. In the slight forms there would be no evidence of any secondary effects, or of mechanical interference with the heart's action, but it is probable that some degree of cyanosis, with a soft murmur over the pulmonary artery in the first few weeks of life, which subsequently disappears, may be due to delayed closure of a fetal passage.

DURATION OF LIFE.—There is considerable difference in the age attained in the various cases of cardiac malformation; the majority of those in whom there is any very serious defect do not survive birth more than a few days.

In some the mechanical difficulty of the circulation makes it impossible for life to be carried on for any great length of time; while in others with a considerable degree of malformation the circuit through the heart and great vessels is sufficiently free for life to be maintained for some years. Many persons with a slight degree of malformation, such as a patent foramen ovale, or a small aperture in the ventricular septum, have died at an advanced age, and have never presented any cardiac symptoms.

The duration of life in pulmonary stenosis depends partly on the degree of the obstruction, and also on the condition of the cardiac septa. The prognosis in extreme stenosis is more favourable when there is some defect in the septum, as by this means relief is afforded to the overcharged right auricle and ventricle. In atresia of the pulmonary orifice life is much more abbreviated, and will also depend mainly upon free communication between the two sides through imperfect septa. In a few cases the patients have lived for some time when the lungs derived their supply from vessels supplied by the aorta.

In transposition of the main vessels the length of life is usually not great, but in some instances the patients have survived to adult life or even longer. An open condition of the septum, or patency of the ductus arteriosus, is favourable for the prolongation of life. With complete

absence of the ventricular septum the majority die in infancy, but a few have survived to adult age and even longer. Abbott (1*a*) from an analysis of 400 cases of congenital heart disease, concludes "that the duration of life is relatively longer in uncomplicated defects of the interauricular septum, in patent ductus arteriosus, coarctation of the aorta and in pulmonary stenosis with closed interventricular septum, while in pulmonary stenosis with defect of the septa the duration of life is much shorter." The last conclusion may be valid as regards all degrees of pulmonary stenosis, but the statement would probably require modification if applied to cases of considerable stenosis.

The cause of death in a large number of infants is due to mechanical interference with the circulation. A considerable number die of convulsions, cerebral abscess, bronchitis, or pulmonary complaints. Those who live to adult age are peculiarly prone to pulmonary tuberculosis, and probably the great majority die from this complaint, or from cardiac failure. Dropsy is comparatively rare. Infective endocarditis is occasionally engrafted upon the malformed valves or stenosed orifice. (Osler, Saenger, Garrod (19).) Thrombosis in the pulmonary artery above the stenosed orifice is extremely rare ; Lee Dickinson described a case.

TREATMENT.—The treatment in congenital heart disease is mainly hygienic. The surface of the body must be carefully protected against cold, and a warm climate is desirable. Violent exertion or over-exercise is apt to produce palpitation and shortness of breath, and should be avoided.

A carefully regulated diet, especially in childhood and infancy, is of importance. Special precautions should be taken to prevent the onset of bronchial affections and convulsions, which are the commonest causes of death at an early age. The special liability to tuberculosis of those who reach adult age must not be forgotten. The treatment of any complications must be directed to the relief of the more urgent symptoms, and the remedies employed would be those which are applicable to similar conditions ensuing in the course of other heart affections.

<div align="right">LAURENCE HUMPHRY.</div>

REFERENCES

1. ABBOTT, MAUDE E. "Congenital Cardiac Disease," in *Modern Medicine* (Osler and M'Crae), 1908, iv. 323.—1*a*. *Idem.* "Statistics of Congenital Cardiac Disease," *Journ. Med. Res.*, Boston, 1908, xix. 77.—2. BANHOLZER. "Ueber das Verhalten des Blutes bei angeborener Pulmonalstenose," *Centralbl. f. inn. Med.*, Leipzig, 1894, xv. 521.—3. BENEKE, F. W. *Die anatomischen Grundlagen der Constitutionsanomalien des Menschen*, 8vo, Marburg, 1878.—4. BORN, G. "Beitrage zur Entwicklungsgeschichte des Säugethierherzens," 4 plates, *Arch. f. mikros. Anat.*, Bonn, 1889, xxxiii. 284.—5. BOXALL. "Incomplete Pericardial Sac ; escape of Heart into left Pleural Cavity," *Trans. Obstet. Soc.*, London (1886), 1887, xxviii. 209.—6. BRISTOWE, J. S. "Malformation of the Pericardium and left Pleura," *Trans. Path. Soc.*, London, 1855, vi. 109.—7. CANTON. "Congenital Obliteration of Origin of the Aorta," *Trans. Path. Soc.*, London, 1848-50, ii. 38.—8. CHEVERS, N. *Collection of Facts illustrative of Morbid Conditions of the Pulmonary Artery*, 8vo, London, 1851.—9. COUPLAND, S. "Defect in the Ventricular Septum of the Heart, probably Congenital ; unusual Site

of Aperture," *Trans. Path. Soc.,* London, 1879, xxx. 266.—10. DICKINSON, W. L. "A Case of Malformation of the Heart with Haemophilia," *Trans. Clin. Soc.,* London, 1895, xxviii. 138.—10a. *Idem.* "Thrombosis of the Pulmonary Artery in Congenital Stenosis," *Trans. Path. Soc.,* London, 1897, xlviii. 57.—11. DICKINSON and FENTON. "Case of Complete Coarctation of the Arch of the Aorta ; Necropsy," *Lancet,* London, 1900, ii. 1196.—12. DUCKWORTH, Sir D. "Notes of a Case in which there was a small Aperture in the Septum Ventriculorum near the Apex of the Heart," *Journ. Anat. and Physiol.,* Cambridge, 1877, xi. 183.—12a. D'ESPINE et MALLET. "Malformation congénitale du cœur avec cyanose paroxystique," *Rev. de méd.,* Paris, 1908, xxviii. 941.—13. FARRE, J. R. *On Malformations of the Human Heart,* 8vo, London, 1814, 2.—14. FAWCETT, J. "Coarctation of the Aorta," *Guy's Hosp. Rep.,* London, 1905, lix. i.—15. FISHER, T. "Two Cases of Congenital Disease of the left Side of the Heart," *Brit. Med. Journ.,* 1902, i. 639.—16. FORSTER. "A Heart consisting of only Two Cavities," *Trans. Path. Soc.,* London, 1846-48, i. 48.—17. FOWLER, J. K. "Membranous Band in the Left Auricle," *Trans. Path. Soc.,* London, 1882, xxxiii. 77.—18. FRASER, A. "A Case of Complete Transposition of the Thoracic and Abdominal Viscera," *Trans. Roy. Acad. Med. Ireland,* Dublin, 1894, xii. 367.—19. GARROD, A. E. "Malformation of the Aortic Valves ; Ulcerative Endocarditis ; associated Malformation of the Liver," *Trans. Path. Soc.,* London, 1897, xlviii. 42.— 20. *Idem.* "On the Association of Cardiac Malformations with other Congenital Defects," *St. Barth. Hosp. Rep.,* London, 1894, xxx. 53.—20a. *Idem.* "The Association of Congenital Heart Disease with the 'Mongolian' Form of Idiocy," *Trans. Clin. Soc.,* London, 1899, xxxii. 6.—21. GIBSON, G. A. "Clinical Lectures on Circulatory Affections," *Edin. Med. Journ.,* 1900, N.S. viii. 1.—22. *Idem.* "Clinical Lecture on Persistent Ductus Arteriosus," *Med. Press and Circ.,* London, 1906, i. 572.— 23. *Idem.* "The Condition of the Blood in the Cyanosis of Congenital Heart Disease," *Lancet,* London, 1895, i. 24.—24. GREENFIELD, W. S. "Case of Malformation of the Heart, with Large Deficiency in the Interauricular Septum, Patency of the Foramen Ovale and Stenosis of the Aortic Orifice," *Journ. Anat. and Physiol.,* London, 1890, xxiv. N.S. iv. 423.—25. GRIFFITH, T. WARDROP. "An Example of a Peculiar Malformation of the Tricuspid Valve of the Heart" (plate), *Journ. Anat. and Physiol.,* London, 1903, xxxviii. N.S. xvii. 251.—26. *Idem.* "Almost complete Absence of the Auricular Septum and other Cardiac Malformations complicated by acquired Mitral Disease," *Med. Chron.,* Manchester, 1902-3, 4th ser. iv. 385.—27. *Idem.* "Case of Combined Disease of the Mitral, Aortic, and Tricuspid Valves of the Heart, in which the Patient was under Hospital Observation for 25 Years," *Lancet,* London, 1907, ii. 1147.—28. *Idem.* "Congenital Malformation of the Heart with Lateral and Antero-Posterior Transposition of the Aorta and Pulmonary Arteries," *Proc. Anat. Soc. Gt. Britain,* Feb. 1895, p. xiv.—29. *Idem.* "Case of Infective Endocarditis involving the Pulmonary Valves and associated with Imperfection of the Interauricular Septum," *Lancet,* London, 1906, ii. 973.—30. *Idem.* "Example of a large Opening between the two Auricles of the Heart, unconnected with the Fossa Ovalis" (plate), *Journ. Anat. and Physiol.,* London, 1899, xxxiii. N.S. xiii. 261, 502.—31. *Idem.* "Heart with a Fibro-Muscular Band passing across the Cavity of the Left Auricle," *Journ. Anat. and Physiol.,* London, 1896, xxx. N.S. x. p. vi.— 32. *Idem.* "Note on a Second Example of Division of the Cavity of the left Auricle into Two Compartments by a Fibrous Band" (plate), *Ibid.,* London, 1900, xxxvii. N.S. xvii. 255.—33. *Idem.* "On Affections of the Tricuspid Valve of the Heart, with a Note on a Pedunculated Thrombus occurring in the right Auricle" (4 plates), *Edin. Med. Journ.,* 1903, N.S. xiii. 105.—34. *Idem.* "Two Examples of Moderator Band in the left Ventricle of the Heart," *Journ. Anat. and Physiol.,* London, 1900, xxxiv. N.S. xiv. p. xxxi.—35. *Idem. Ibid.* 1892, xxvi. 117.—36. HARE. "Malformation of the Heart. Complete closure of the Orifice of the Pulmonary Artery. Very small Foramen Ovale. Cyanosis," *Trans. Path. Soc.,* London, 1853, iv. 81.—37. *Idem.* "Malformation of the Heart ; Contraction of the Pulmonary Orifice, with an Opening in the Septum Ventriculorum," *Ibid.,* 1860, xi. 45.—38. *Idem.* "Malformation of the Heart ; Obstruction at the Aortic Orifice (only two Valves) ; Open Ductus Arteriosus," *Ibid.,* 1860, xi. 46.—39. HENOCH. *Congenital Cyanosis,* Transl. New Syd. Soc.—40. HESS. "Case of Malformation of the Heart, exhibited by Dr. Peacock" (plate), *Trans. Path. Soc.,* London, 1855, vi. 117.—41. HIS. *Anat. mensch. Embryonen,* Leipzig, 1880.—42. HOLMES, T. "Distal Ligature of the left Carotid Artery for Aortic Aneurysm," *Trans. Clin. Soc.,* London, ix. 114, x. 97, and (sequel) xxi. 146.—43.

HUNTER, W. "Three Cases of Mal-conformation in the Heart" (plate), *Med. Observations and Inquiries*, London, 1812, vi. 291.—44. KEITH, A. "Exhibition of Thirty Malformed Human Hearts," *Journ. Anat. and Physiol.*, London, 1905, xxxix. N.S. xix. p. xiv.—45. *Idem.* "Heart," Quain's *Anatomy*, 1908, 11th edit. i. 209.—46. *Idem.* "Malformations of the Bulbus Cordis; an Unrecognised Division of the Human Heart," *Studies in Pathol., Aberdeen Quatercent. Celebrat.*, Aberdeen, 1906, p. 55.—47. *Idem.* "Partial Deficiency of the Pericardium," *Journ. Anat. and Physiol.*, London, 1907, xli. 3rd ser. ii. 6.—48. KEITH and FLACK. "The Form and Nature of the Muscular Connections between the Primary Divisions of the Vertebrate Heart," *Ibid.*, 1907, xli. 3rd ser. ii. 172.—49. KUSSMAUL. "Ueber angeborene Enge und Verschluss der Lungen-Arterien-Bahn" (3 plates), *Ztschr. f. rationelle Med.*, Leipzig u. Heidelberg, 1866, Dritte Reihe, xxvi. 99.—50. LAENNEC. *Auscultation.* —51. LEES, D. B. "Case of Malformation of the Heart, with Transposition of the Aorta and Pulmonary Artery," *Trans. Path. Soc.*, London, 1880, xxxi. 58.—51a. LIBMAN, E. "Congenital Stenosis of the Aorta at the Isthmus," *Proc. N.Y. Path. Soc.*, 1902.—52. LINDES. *Ein Beitrag zur Entwicklungsgeschichte des Herzens*, Dissertation, Dorpat, 1865.—53. LLOYD-JONES, E. "Congenital Heart Disease," *Journ. Physiol.*, Cambridge, 1891, xii. 326.—54. M'CRAE, J. "A Case of Congenital Atresia of Pulmonary Artery, with Transposition of Viscera; a Second Case of Transposition," *Journ. Anat. and Physiol.*, London, 1906, xl. 3rd ser. i. 28.—55. MACKENZIE, Sir STEPHEN. "Two Cases of Congenital Malformation of the Heart" (plate), *Trans. Path. Soc.*, London, 1880, xxxi. 63.—56. MACLENNAN. "Case of Dexio-Cardia without displacement of other Viscera," *Brit. Med. Journ.*, 1896, ii. 1314.—57. MARSHALL, A. M. *Vertebrate Embryology*, 8vo, London, 1893.—58. MARTIN, S. "Heart with left Auricle divided by a Septum," *Journ. Anat. and Physiol.*, London, 1900, xxxiv. N.S. xiv. p. xxxi.—59. MECKEL, J. F. *De Cordis Conditionibus Abnormibus* (plate), Dissertation, Halae, 1802.—60. MINOT, C. S. *Human Embrology*, 8vo, New York, 1892.—61. MOORE, N. "Malformation of the Heart," *Trans. Path. Soc.*, London, 1881, xxxii. 39.—62. MORGAGNI. *De Sed. et Causis Morb.*, Venetiis, 1761.—63. MOUSSOUS, A. C. *Maladies congén. du cœur*, Paris, 1906.—64. MURRAY, G. R. "A Case of Incomplete Coarctation of the Aorta; Necropsy," *Practitioner*, London, 1904, lxxii. 284.—65. OSLER, W. "The Goulstonian Lectures on Malignant Endocarditis," *Brit. Med. Journ.*, 1885, i. 467, 522, 577.—66. PARKER. "Malformation of the Heart; great Contraction of the Pulmonary Orifice; Aorta arising entirely from the right Ventricle. Aperture in the Septum Ventriculorum," *Trans. Path. Soc.*, London, 1846-48, i. 51.—67. PEACOCK, T. B. *Malformations, etc., of the Human Heart, with Original Cases*, 2nd ed., 8vo, London, 1866.—68. *Idem.* "Malformation of the Heart; Large Aperture in the Septum of the Auricles; with the Foramen Ovale closed," *Trans. Path. Soc.*, London, 1878, xxix. 43.—69. PEACOCK's collection of Specimens of Malformation of the Heart in the Hunterian Museum.—70. RAUCHFUSS, C. "Die angeborenen Entwicklungsfehler und die Fötalkrankheiten des Herzens und der grossen Gefässe," Gerhardt's *Handb. der Kinderkrankheiten*, Tübingen, 1878, Bd. iv. Abth. i. 12.—71. ROKITANSKY, C. F. *Die Defecte der Scheidewände des Herzens*, fol. Wien, 1875.—72. ROLLESTON, H. D. "Band in the left Auricle of the Heart of a Boy aged 1½ Years," *Proc. Anat. Soc., Gt. Britain*, Feb. 1896, p. v.—73. *Idem.* "Communication between the Ventricles of the Heart; Congenital," *Trans. Path. Soc.*, London, 1891, xlii. 65.—74. SABATIER, A. *Études sur le cœur et la circulation centrale*, 4to, Montpellier, 1873.—74a. SAENGER. *Deutsche med. Wchnschr.*, 1889, xv. 148.—75. SHATTOCK, S. G. "Atresia of the Aortic Aperture in an Infant," *Trans. Path. Soc.*, London, 1881, xxxii. 38.—76. *Idem.* "Congenital Atresia of the Oesophagus" (plate), *Ibid.*, 1900, xli. 90. — 77. SHAW, H. BATTY and HIGHAM COOPER. "Pulmonary Hypertrophic Osteo-Arthropathy in Congenital Heart Disease," *Trans. Clin. Soc.*, London, 1907, xl. 259.—78. STILLÉ. "On Cyanosis, or Morbus Caeruleus," *Amer. Journ. Med. Sc.*, Phila., 1884, N.S. viii. 25.—79. SUTER, F. "Ueber das Verhalten des Aortenumfanges unter physiologischen und pathologischen Bedingungen," *Arch. f. exper. Path. u. Pharmak.*, Leipzig, 1897, xxxix. 289.—80. SYMINGTON. "Specimen of a Heart with Incomplete Interauricular and Interventricular Septa, one Auriculo-Ventricular Opening (left) and a Single Arterial Orifice (Aortic)," *Journ. Anat. and Physiol.*, London, 1900, xxxiv. N.S. xiv. p. xiv.—81. THÉRÉMIN. *Etudes sur les affections congénitals du cœur*, St. Petersburg, 1895.—82. THIELE, F. H. "Case of Congenital Cardiac Malformation," *Journ. Anat. and Physiol.*, London, 1903, xxxvii. N.S. xvii. p. xliv.—83. TOENIESSEN.

Ueber Blutkörperchenzählung bei gesunden und kranken Menschen, Erlangen, 1881.—84. Townsend. *Boston Med. and Surg. Journ.*, 1900, cxlii. 426.—85. Turner, Sir W. "Human Heart with Moderator Bands in the Left Auricle," *Proc. Anat. Soc., Gt. Britain*, Feb. 1893, p. xix.—85a. Turney. "Three Cases of Patent Ductus Arteriosus with Arterio-Venous Murmur," *Trans. Clin. Soc.*, London, 1907, xl. 245.—85b. Variot. "Clinique Infantile," 1908, p. 193.—86. Viner, N. "Blue Baby, Seventeen Years Old," *Montreal Med. Journ.*, 1908, xxxvii. 181.—87. Virchow. *Ueber die Chlorose und die damit zusammenhängenden Anomalien im Gefäss-Apparate, insbesondere über Endocarditis puerperalis*, 8vo, Berlin, 1872.—88. Wagstaffe, W. W. "Two Cases of Free Communication between the Auricles, by Deficiency of the Upper Part of the Septum Auricularum. From Cases aged 52 and 6 respectively. No Cyanosis" (plate), *Trans. Path. Soc.*, London, 1868, xix. 96.—89. White, W. Hale. "Case of Patent Ventricular Septum, together with an Aneurysm of the Base of the Aorta opening into the right Ventricle," *Ibid.*, 1892, xliii. 34.—90. Wilson, J. "A Description of a very Unusual Formation of the Human Heart," *Phil. Trans.*, London, 1798, lxxxviii. 346.—91. Young, A. H. "Rare Anomaly of the Human Heart. A Three-Chambered Heart in an Adult aged Thirty-Five Years," *Journ. Anat. and Physiol.*, London, 1907, xli. 3rd ser. ii. 190.

L. H.

RIGHT-SIDED VALVULAR DISEASES

By G. Newton Pitt, M.D., F.R.C.P.

Diseases of the Pulmonary Valves

PULMONARY INCOMPETENCE.—This is the rarest of the valvular lesions of the heart, only 24 cases having been noted in the post-mortem room at Guy's Hospital out of 16,000 examinations during a period of thirty-two years.

One of the earliest papers on the subject was by Whitley (1857), who reported 3 cases. Blattmann (1887) collected 14, Barié (1891) 35, and Gerhardt (1892) 6 new cases; all of which were confirmed by necropsy. Bryant in 1901 published 16 cases associated with mitral stenosis, and refers to 9 other published cases of pulmonary incompetence. To this we have been able to add 51, of which 39 are unpublished cases from the records at Guy's Hospital.[1] The total number is therefore 109, besides some 30 published clinical cases which were not verified after death.

Etiology.—These cases may be divided into five important groups: A, Infective endocarditis, 60 cases; B, Dilated pulmonary artery, 18 cases; C, Aortic aneurysm pressing on the pulmonary valves, 14 cases; D, Abnormality in the number of valves, 13 cases; E, Pulmonary stenosis, 14 cases; F, Unclassified, 6 cases.

[1] I am indebted to my friend, Dr. Alfred Salter, for the great trouble he took in examining for me the records at Guy's Hospital from 1874 to 1897, and for his collation of the notes on the cases of pulmonary and tricuspid lesions.

A. *Infective Endocarditis.*—This is by far the most frequent cause of the incompetence, being present in 60 cases.

The age-distribution is as follows :—

0-10 years	.	.	.	2 cases.		41-50 years	.	.	.	4 cases.
11-20 ,,	.	.	.	13 ,,		51-60 ,,	.	.	.	4 ,,
21-30 ,,	.	.	.	16 ,,		61-70 ,,	.	.	.	1 case.
31-40 ,,	.	.	.	12 ,,						

Etiology.—There are several associated conditions which are of especial importance in connexion with infective endocarditis of the pulmonary valve.

No obvious cause	.	.	.	21 cases.	Gonorrhoea	.	.	. . 8 cases.
Patent ventricular septum	.	10 ,,			Puerperal infection.	.	.	. 5 ,,
Congenital pulmonary stenosis		8 ,,			Pyaemia	.	.	. . 4 ,,
Patent ductus arteriosus.	.	3 ,,			Pneumonia	.	.	. . 5 ,,

In 21 of these infective endocarditis occurred in a malformed heart.
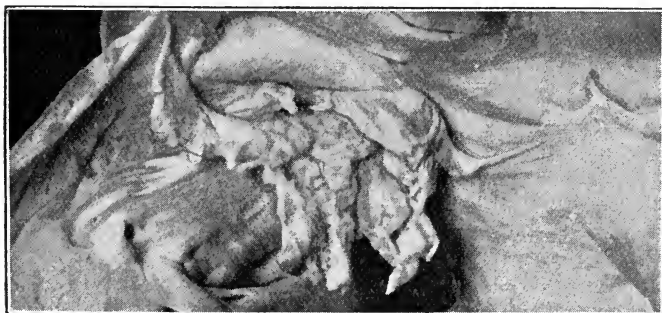
FIG. 28.—A pulmonary artery with a mass of pendulous vegetations attached to the right anterior cusp ; the greater part of the posterior valve has completely ulcerated away, and what remains of the right anterior valve has been perforated by the pressure of the infective vegetations.
    From a man aged nineteen, who had gonorrhoea four months before death, and was admitted with pyrexia, dyspnoea, and a double basic bruit.   At the inspection there were also found advanced tubal nephritis, apoplexies in the lung, and a small patch of pneumonia.

The regurgitation of the blood-stream, through an aperture in an imperfect ventricular septum, or through a patent ductus arteriosus, tends to damage the endothelium of the wall on which it impinges, and it thus forms a suitable nidus for vegetations.   The formation of vegetations opposite an aperture, and on malformed pulmonary valves, is well shewn in several museum specimens.

It is remarkable how very rarely a diastolic bruit has been noticed in cases of congenital pulmonary stenosis, even when the cusps have been fused into a rigid cone, through which, it would appear, regurgitation must have taken place.   Probably the blood does not regurgitate vigorously enough to produce a bruit through a small orifice, owing to the low pulmonary pressure.   It has not infrequently happened that a diastolic murmur has not been heard until an ulcerative destructive process has been superadded.

In cases of infective endocarditis due to gonorrhoea or puerperal septicaemia the pulmonary valve is the one which is especially liable to be implicated. In several of the former cases streptococci and not gonococci have been found in the vegetations.

In 16 out of 19 cases recorded at Guy's it is definitely stated that there was not any history of any rheumatism, and in only one patient had its occurrence been noted. This differs from left-sided lesions, in which old rheumatic heart disease has generally preceded the infective process. Still in several there was chronic thickening of the valves on the left side, rheumatic in origin.

Sometimes masses of vegetations block the orifice and prevent the closure of the valves ; in a few cases, however, the process has consisted merely in a destruction of the cusps, clearing away one or more as if it were cut off with scissors. This is well shewn in Fig. 28, in which one cusp is retroverted and carries a pendulous mass of vegetations. The greater part of the other cusps has ulcerated away, leaving only small pieces at the attached margins.

TABLE OF ASSOCIATED LESIONS IN 19 CASES

Mitral stenosis	2 cases.
Thickened mitral valve	3 ,,
Mitral valve, vegetations on	3 ,,
Aortic ,, ,, ,, ...	4 ,,
Tricuspid ,, ,, ,, ...	3 ,,
All four valves affected	4 ,,
Aneurysm on a branch of the pulmonary artery .	2 ,,
Infarcts in the lung	5 ,,

It is specially mentioned in some of the post-mortem reports of these cases that the spleen was small.

In 3 cases the pulmonary valves were also thickened ; and in 10 cases the pulmonary valves alone shewed vegetations, although in one of them the mitral valve was thickened, and in one stenosed.

Symptoms (19 cases).—Haemoptysis and dyspnoea were the symptoms chiefly noticed. In 8 cases the implication of the pulmonary valves was definitely diagnosed. This is a very large proportion when it is borne in mind that some of the cases were in surgical wards where the condition of the heart would not be systematically examined, and others were admitted shortly before death. In 5 carefully reported cases, only a systolic bruit appears to have been observed. In 3 cases it is definitely stated that no bruits could be heard. In others a diastolic, as well as a systolic, bruit was noted.

B. *Dilatation of the Pulmonary Artery.*—We have collected 18 cases of pulmonary incompetence associated with and probably due to dilatation of the artery ; in six years Bryant collected 16 (9 verified P.M.) ; some of the cases overlap, so his list is not referred to here. It has long been

recognised that aortic incompetence may take place when the first part of the aorta becomes dilated from any cause, although the valves remain healthy. It is an interesting question whether dilatation of the pulmonary artery may, in the same way, be an efficient cause of pulmonary incompetence.

Pathological Evidence.—Conditions under which Dilatation of the Pulmonary Artery takes place.—An examination of museum specimens, which are collected from the more extreme cases, would appear to shew that the associated conditions are fibrosis of the lungs, and bronchitis with emphysema; dilatation also occurs in some cases of pulmonary stenosis. This conclusion, however, is fallacious, as an examination of 5000 consecutive necropsies shews that mitral stenosis was present in 19 out of 21 cases in which dilatation of the pulmonary artery was noted, and is consequently the chief lesion to be considered. In the two remaining cases phthisis, and bronchitis with emphysema, were present.

Clinical Evidence.—Not infrequently, in advanced cases of mitral stenosis, pulsation may be observed in the second and third left spaces close to the sternum, which is now generally considered to be due to dilatation of the infundibulum and of the pulmonary artery. Bamberger, Graham Steell, Goodhart, Bryant, and other observers, have insisted that the occurrence of diastolic bruits down the left side of the sternum in cases of advanced mitral stenosis, and also in cases of dilated pulmonary artery with emphysema or fibroid lung, is not very infrequent. The bruits have been considered to indicate a certain amount of leakage through the pulmonary orifice. Notes of several cases are to be found among the Guy's clinical records, but in all, save two, there was no conclusive evidence of regurgitation at the necropsy (including Bryant's 16). It may readily be granted, however, that incompetence would probably have to continue for a long time before it need produce structural changes.

Stokes' observation, made many years ago, remains true in the main, that even when the pulmonary artery is found dilated there are generally no changes in the cusps, nor any notable symptoms during life. This last sentence must be qualified by the statement that a variable diastolic bruit may be occasionally detected. The orifice may have been sufficiently stretched during life to allow leakage, and yet appear normal after death.

Some of these basic diastolic murmurs in cases of mitral stenosis have led to a diagnosis of aortic incompetence; yet there can be little doubt that in such cases, when the aortic valves are found healthy, the murmurs have been of pulmonary origin.

Dr. Graham Steell in 1889 drew attention to the variable diastolic murmur to the left of the sternum as evidence of pulmonary incompetence and of high pressure in the artery. Similar observations have been made by Dr. G. A. Gibson, Sir J. Barr, and Sir Dyce Duckworth.

These authors have published over 20 post-mortem cases secondary to mitral stenosis. There were 3 more cases at Guy's between 1901-6; these were briefly—(i.) Man, thirty-six, mitral stenosis, aortic valve normal,

adherent pericardium, diastolic bruit at one time audible. (ii.) Man, twenty-seven, enormous heart, pulmonary artery half as large again as the aorta, mitral stenosis, pulmonary incompetence. (iii.) Man, sixteen, mitral stenosis, tricuspid and pulmonary incompetence, greatly dilated pulmonary artery. Right auricle contained 125 grams and left auricle 25 grams of clot.

1. There is therefore evidence, both clinical and pathological, that in advanced mitral stenosis the pulmonary artery may become distended.

2. In such cases a variable diastolic bruit down the left side of the sternum may be evidence of a functional incompetence of the pulmonary orifice. This is certainly of far more frequent occurrence towards the end of life than is generally recognised.

3. Almost invariably post-mortem evidence of any structural change at the orifice will be wanting.

In the 6 following cases there was both clinical and post-mortem evidence of pulmonary incompetence without any thickening of the valves :—

1. A man, aet. thirty-four, who for six months had had a cough with dyspnoea. The cardiac impulse was feeble, but there was intense and widely extended fremitus ; a loud musical diastolic bruit, not transmitted to the arteries, was audible. The pulse was small and feeble, not aortic in character. Distended jugular veins.

P.-M.—Pulmonary orifice 4 inches, aortic $3\frac{1}{4}$, tricuspid $6\frac{1}{4}$, mitral 5 inches (Stokes).

2. Man, aet. thirty-four, with dyspnoea, cyanosis, and oedema.

P.-M.—General pleuritic adhesions. Right ventricle greatly dilated and hypertrophied. The pulmonary artery was greatly dilated, and its valves were incompetent (Rokitansky).

3. Man, aet. twenty-eight, with a history of acute rheumatism five years previously. At the apex a systolic and a fainter diastolic bruit were heard ; over the tricuspid a loud systolic bruit, and a dull diastolic sound ; and in the first, second, and third left spaces there was a short systolic with a variable diastolic bruit.

P.-M.—Aneurysmal dilatation of the pulmonary artery ; valves thin, but elongated. Mitral stenosis and pulmonary incompetence (Galewski).

4. Man, fifty-two, incompetence of all valves, dilated pulmonary artery.

5. Man, thirty - five, acute on chronic rheumatism, with dilatation of tricuspid and pulmonary orifices.

6. Bronchitis and emphysema with dilatation of right-sided orifices.

In 9 other cases of pulmonary incompetence the dilatation of the artery was the main cause of the incompetence, but the valves were thickened.

In 2 cases the cusps were reduced to two, and twice there were four. One of these cases is of especial interest, as it was under the care of Dr. Addison in 1857 ; the remarkable character of the to-and-fro basic bruit led Sir Hermann Weber to diagnose the pulmonary origin of the disease.

A similar case of thick incompetent pulmonary valves with an orifice $6\frac{1}{8}$ inches in circumference, associated with advanced emphysema, has been recorded by Dr. Coupland. In the Guy's Museum there are two specimens in which the margins only of the incompetent pulmonary valves are thickened ; in one the valves are also retroverted. The imperfection at the pulmonary orifice in both cases was diagnosed. They were associated with mitral stenosis, and with a fibroid condition of lung. Prof. L. Rogers has recorded a case of incompetence due to dilatation associated with atheroma of the pulmonary artery which was diagnosed during life. The patient, a native of Calcutta, aged twenty-three, was admitted with a pulmonary diastolic and an apical systolic bruit and oedema. The necropsy shewed hydropericardium with atheroma and dilatation of the pulmonary artery, which had produced incompetence without thickening of the pulmonary valves ; the coronary veins were much dilated. He also records 10 cases of atheroma with dilatation of the pulmonary artery, occurring chiefly between the ages of twenty and forty in the natives of India, and probably due to syphilis ; in only 1 case were the pulmonary valves thickened. Palpitation, dyspnoea, and dropsy, due to tricuspid incompetence, were the main symptoms ; the right side was greatly dilated and hypertrophied, and hence the disease was often mistaken for mitral disease. Two of his cases, however, also presented mitral stenosis. The exceptional occurrence of great dilatation of the pulmonary artery with atheroma in connexion with bronchitis and emphysema was noted by Whitley many years ago, and Fagge put up specimens to illustrate the condition ; they did not associate the condition with syphilis. In 2 cases there was also infective endocarditis of the cusps.

C. *Aortic Aneurysm pressing on the Pulmonary Artery* (14 cases). —An aneurysmal pouch of the aorta immediately above the valves, especially above the left posterior cusp, may press upon the pulmonary artery, or open into it close to the valves. The inflammation which is set up, as the wall yields, causes the free margin of one or even of two pulmonary cusps to become firmly adherent to the wall of the artery, and thus to become incompetent. The adhesion of the free margin of valves to the wall of the vessel where they are stretched over an aneurysmal pouch is shewn in Fig. 29 ; on the right of the figure is seen the aperture through which the aorta had opened into the pulmonary artery. One pulmonary cusp has completely blended with the wall, and only the extreme end of the second has not been obliterated. As may be readily imagined, the incompetence in such a case is considerable.

This cause of pulmonary incompetence, which has been overlooked by most authors (Grawitz reports 2 cases), cannot really be very uncommon, as there are 8 such specimens at Guy's and 2 at St. George's Hospital, in which pulmonary cusps had adhered to the wall, and thus become useless.

The evidence of the incompetence is generally masked during life by the associated lesion of the aortic valves.

D. *Abnormality in the Number of Pulmonary Valves* (13 cases).—Eight cases with two cusps ; 4 with four cusps ; and 1 with five.

In most cases these abnormalities have been of congenital origin ; the consequent imperfect closure of the orifice probably initiated sclerotic changes in the cusps which tended to progress as the incompetence became established.

In one or two cases the regurgitant stream through a patent ven-

Fig. 29.—A pulmonary artery stretched over an aortic aneurysm ; two cusps have adhered to the wall of the vessel and the aperture, which is apparent immediately below the juncture of two valves, leads into a saccular aneurysm of the aorta, an inch across, immediately above the aortic valves, which had remained competent. The margin of the aperture is smooth, not much thickened, and is free from lymph.
From a man aged thirty-nine, who gave no history either of rheumatism or of syphilis. He had been short of breath for some years, but was only ill for three weeks.

tricular septum has impinged on normal segments and caused two to adhere together.

Dilg has collected the scattered information on the subject of abnormality in the number of cusps, and finds recorded of

Two pulmonary cusps,		64 cases.	Two aortic cusps,		23 cases.
Four	,, ,,	24 ,,	Four ,,	,,	2 ,,
Five	,, ,,	2 ,,	Five ,,	,,	1 case.

He attributes the abnormalities to (i.) fetal endocarditis ; (ii.) defective site ; (iii.) one of these, associated with endocarditis in later life. Meckel noted that when the foramen ovale was patent there were often only two pulmonary segments. When there are only two cusps one is usually abnormally large, and has arisen from the fusion of two. Normally in molluscs, osteoid fish, and reptiles, only two cusps form. In fetal chicks the anterior and inner cusps form the first, and the third cusp at a later period. Virchow, however, considered that in many cases the union of two cusps is a change belonging to a later period of life.

E. *Pulmonary Stenosis* (14 cases). — In 8 of these cases there was ulcerative endocarditis ; in 2, a dilated pulmonary artery ; and in 1, only two cusps. There remain 3 cases, not included in the previous lists, in

which a basic diastolic bruit had been noted indicating regurgitation through an orifice which was ultimately found to be congenitally stenosed. The extreme rarity of this bruit in these cases is remarkable.

. F. The 5 following, which cannot be included in the previous groups, remain to be considered. (*a*) Foramen in ventricular septum opening directly on a pulmonary valve (Wunderlich). (*b*) A gummatous infiltration of the pulmonary artery, with adhesion of a valve to an aortic aneurysm (Schwalbe). (*c*) and (*d*) Two specimens· in Guy's Museum, from patients aged thirty-eight and seventy, in whom there were only symptoms of bronchitis during life. In one, there are only two cusps, which are retroverted, one of them being adherent to the vessel wall ; in the other specimen one cusp is adherent along its free margin, and the other two are elongated and thickened. The causation of these changes is obscure. In both cases a lesion of the pulmonary orifice was diagnosed. (*e*) A man, aged twenty-seven, who had had four attacks of rheumatism ; after death the mitral orifice was stenosed, the pulmonary and aortic valves incompetent, and the pericardium adherent. One pulmonary flap was markedly retroverted (Gerhardt).

Thickened Pulmonary Valves.—Three additional cases of thickened valves may be noted :—(1) A man, aged forty-five, chronic bronchitis and emphysema ; tricuspid orifice $5\frac{1}{2}$ inches, but valves normal. The pulmonary valves are thickened, and one rather readily tends to turn over. (2 and 3) Women, aged thirty-four and twenty-seven, both had old rheumatic mitral, aortic, and tricuspid stenosis. The pulmonary valves were thickened but competent.

In the preceding groups are included 4 other cases of mitral stenosis and cardiac failure, and 4 of mitral and tricuspid incompetence, which had presented pulmonary incompetence with thickening of the valves.

While discussing the etiology attention should be drawn to the remarkable fact that in cases of the most extreme mitral stenosis, in which a similar defect is ultimately set up in the tricuspid valve, the pulmonary cusps almost invariably escape, and do not shew even a trace of thickening.

The proportion of cases with a history of acute rheumatism is small —not much over 10 per cent ; and the influence of rheumatism is far less on this than on any other valve. Besides the 3 cases referred to later in which the acute vegetations were limited to the pulmonary orifice, there was one case in which there was also infective endocarditis of the other valves.

The free margins of the cusps were more or less adherent in 18 cases, and retroverted in 4.

The statement usually made that pulmonary incompetence is a congenital defect is not accurate ; practically, a diastolic bruit is not usually audible in cases of pulmonary stenosis, unless infective endocarditis is also present. Yet, on the post-mortem table, the valves may be found united together in a truncated cone, and cannot have closed. About one-quarter of the collected cases shewed evidence of old pulmonary

stenosis, and in the same proportion there was some abnormality in the number of the valves. There is practically no evidence that trauma is a cause of incompetence.

In 3 cases, independently of congenital lesions, calcareous nodules were present in the valves.

There is no liability to the development of tuberculosis such as occurs with pulmonary stenosis.

Clinical Symptoms of Pulmonary Incompetence.—The cases may be divided into three groups :—(1) Those which present themselves with pyaemic symptoms, in more than half of which in recent years a correct diagnosis has been made. (2) Those with cardiac symptoms. It is often not easy to decide whether it is the pulmonary or the aortic orifice which is affected. This is more especially so when there are multiple lesions, as, for instance, an aortic aneurysm or infective endocarditis of several valves. (3) In a few cases there has been no evidence during life to indicate that the heart was affected, and no bruits were audible. Such cases must remain for post-mortem diagnosis.

Signs and Symptoms.—(i.) One of the first changes is a displacement of the cardiac impulse downwards and considerably to the left, in consequence of the right-sided enlargement which results from the pulmonary incompetence. The right ventricle dilates and hypertrophies, and ultimately, when the right auricle enlarges, the dulness will extend beyond the right side of the sternum. When the muscular accommodation is good the force of the impulse is increased, and epigastric pulsation is marked ; sometimes a systolic thrill is palpable (Bamberger). When there is advanced cardiac failure they are absent. A distinct pulsation may also sometimes be felt in the second left space.

(ii.) On auscultation a variable and usually soft diastolic bruit may be heard on the left side near the base ; according to Bryant it is better heard half-way between the left nipple and the sternum than it is near the sternum ; he noticed it in the third space in 12 out of 16, in the second in 7, and in the fourth in 4 cases. It is localised and is not transmitted to the aorta or the arteries of the neck.

(iii.) The absence of a quick water-hammer pulse, and of marked pulsation in the main systemic vessels.

(iv.) As was first noticed by Gerhardt, the diastolic bruit is intensified by expiration.

(v.) Emboli may occur in the lungs, and as a result haemoptysis. This is present in one-third of the cases, chiefly in those due to infective endocarditis (Weckerle).

(vi.) Not infrequently both the aortic and pulmonary valves are incompetent. Can the murmurs due to the two lesions be distinguished ? Gerhardt thinks that the latter is deeper in tone and rougher than the aortic, owing to the feeble pressure in the pulmonary artery.

(vii.) The murmur is intensified by the erect position.

(viii.) A capillary pulse can be detected in the pulmonary circulation. When long, deep breaths are taken, the vesicular murmur is jerky, and

can be heard to wax and wane with the cardiac contractions at places on the chest, such as the angle of the right scapula, where normally such tides never occur. Gerhardt has also shewn this graphically by registering the variations of pressure in the tracheal column of air.

Although the number of diagnostic symptoms is not small, practically the difficulties of diagnosis may be enormous, because the lesion is excessively rare; and it is generally complicated by other serious mischief.

The development of a diastolic bruit towards the end of life, especially in cases of mitral stenosis, is not infrequently due to a relative pulmonary incompetence, but structural change is most exceptional. It should, moreover, be borne in mind that murmurs, which apparently have a distribution similar to that which we have described above as characteristic of pulmonary incompetence, may ultimately be found to have been produced at the aortic orifice.

Dyspnoea, cyanosis, and oedema are symptoms which generally appear as the heart fails. Of a specially suggestive import when associated with these is haemoptysis, which may arise from sudden changes in the pulmonary pressure, or from emboli which have broken off from friable vegetations.

Prognosis.—Pulmonary incompetence is always a serious lesion. It is usually the result of an acute infective process, or is the final stage in other serious heart trouble. Apart from such complications, it would appear that patients with pulmonary incompetence may live for a considerable time; it is the associated lesions which cause death.

Treatment.—No special line of treatment is indicated unless there be reason to suspect infective endocarditis. Of recent years many such cases have been treated by making a culture from a syringeful of blood drawn from a vein, and preparing a vaccin. The extremely good results which have been obtained in controlling cutaneous and subcutaneous infective lesions, by injecting such killed cultures, have led to the impression that vaccine treatment is equally valuable for arterial pyaemia. From an experience of a considerable number of cases I have come to the conclusion that in several cases the patient's end has been accelerated by their use, owing to the active changes which have been set up; in others, benefit has resulted. The treatment is not to be lightly recommended, as the possibility of injury is at least equal to that of benefit. The dose injected should be much smaller than that usually employed.

Acute Endocarditis of the Pulmonary Valves without Evidence of any Incompetence.—Minute vegetations may exceptionally be found on the pulmonary valves. Out of 22,000 inspections there were three in which acute vegetations were found on the pulmonary valves only. (α) A man with pernicious anaemia and old aortic and mitral incompetence. (β) A man, aged twenty, with old aortic, mitral, and tricuspid stenosis. (γ) A woman, aged fifty, with cancer and old pulmonary stenosis. The two former were rheumatic in origin.

PULMONARY STENOSIS.—In this lesion the valves are usually united and form a perforated dome. It is almost invariably congenital in origin, and has already been considered (*vide* p. 283). Occasionally, however, we meet with cases — there are over 10 on record — in which there is evidence that the stenosis originated after birth, and in others it has considerably increased after birth as the result of an attack of rheumatism.

Mitral, pulmonary, aortic, and tricuspid stenosis were found in a woman aged sixty-one; there was also a dermoid cyst attached to the heart in front. The right pulmonary artery was greatly dilated and admitted three fingers, the left was normal in size. The pulmonary valves were matted together into a dome, resembling that due to a congenital lesion. The other valve troubles were due to rheumatism. The cause of the dilatation of the right pulmonary artery is obscure.

Paul reports the case of a man, aet. thirty-six years, admitted with advanced phthisis and haemoptysis, who had enjoyed good health until the age of ten, when he was laid up for three months with endocarditis and acute rheumatism; since then he had been troubled with palpitation and slight dyspnoea. The onset of phthisis was two years before his death. The right ventricle was hypertrophied, and there was a basic systolic bruit on the left side not propagated up into the vessels of the neck. After death it was found that the ductus arteriosus was closed; the pulmonary artery was not wasted, but the orifice was stenosed owing to adhesions between the valves. Paul considers that cyanosis is generally absent in the cases which are not of congenital origin.

Besides these cases, the orifice or the lumen of the artery may be stenosed by the bulging of an aortic aneurysm, or by masses of fungating vegetations blocking the aperture, to which reference has already been made (p. 310).

DISEASES OF THE TRICUSPID VALVE

In order to define the causation of the lesions of the tricuspid valve as clearly as possible, and to determine the relative frequency of the symptoms of the various associated lesions, the clinical and post-mortem records of Guy's Hospital for over twenty years were examined, and the lesions of the tricuspid valve were tabulated in which (i.) acute vegetations were found upon the valve, (ii.) the valvular cusps were thickened, (iii.) the cusps were affected with infective endocarditis, (iv.) the cusps were incompetent, and (v.) the tricuspid orifice was stenosed.

1. ACUTE VEGETATIONS ON THE TRICUSPID VALVES.—Seventy-four cases. The proportion of male to female cases was 4 to 3. The youngest patient was only fifteen days old; the case was one of pyaemia with acute vegetations on the mitral and tricuspid valves. There were only two other patients under one year of age; the number of cases for the consecutive decennia were 8, 23, 20, 7, 3, 3, 1.

On examining the records, we were struck at once by the frequency with which vegetations were also met with upon the mitral and aortic valves; and also by the still greater frequency with which old lesions of these valves were present. In 57 cases there was evidence of old mitral disease, and in 49 of recent vegetations on the mitral, in the majority of cases on thickened valves. In nearly half the cases there was mitral stenosis. In these tricuspid cases the aortic valves also shewed evidence of old damage in 26 cases; and in 44 cases recent vegetations, but occurring more frequently on previously healthy valves than on those which had been diseased. In 20 cases there was recent pericarditis, and in 16 the pericardium was adherent. In only one case out of the whole series were the mitral valves stated to have been normal, and this presented acute pericarditis, and also vegetations on thickened aortic valves. On three occasions the vegetations were limited to the tricuspid valve; in two of these cases the mitral valves were thickened, in the third there was thickening of the mitral, aortic, and pulmonary valves. Recent lymph over the surface of the pleura was found in over 20 cases. With regard to the history of rheumatism, it was noted as present during the final illness in 28 cases; in 20 other cases there was a history of previous attacks, and in only 10 cases was it stated that there had never been any acute rheumatism. In one quarter, the vegetations were large and fungating. The evidence was therefore conclusive that the dominant cause of vegetations on the tricuspid is a severe rheumatic process; not invariably shewn by acute joint troubles, but practically without exception by evidences of either acute or old mischief, involving the mitral, and very frequently the aortic valves; by inflammation of the pericardium in more than half the cases, and by acute pleurisy in nearly half the cases. An examination of the records for the past thirty years presents no evidence of any importance in favour of any other causation.

2. THICKENING OF THE TRICUSPID VALVES.—The 148 cases, in which thickening of the tricuspid flaps was noted, fall into three great groups :—

A. Those which are secondary to *rheumatism, or left-sided valvular disease.*

B. Those which are the result of *degenerative changes involving several organs.*

C. Those associated with *pulmonary stenosis of congenital origin.*

That these distinctions are fundamental ones, is at once proved by the relative age-distribution; as we find that the majority of cases in the first group occur between the ages of twenty and thirty, and five-sixths of the cases between the ages of ten and forty.

In the second group, one patient, aged twenty-seven, with emphysema, was the only case in which the age was under thirty : the decennium in which the greatest number of cases occurred was between fifty and sixty, and six-sevenths of the cases occurred between the ages of forty and seventy.

A. In the first group *mitral stenosis* was present in a very large

number.   In 48 there was a definite history of rheumatism; and 20 others with left-sided heart lesions, although there was no history of rheumatism, were, nevertheless, most probably due to it.   As a sub-group of this class we would put the cases of mitral stenosis in which the kidneys were found to be granular, of which there were 11. This tendency of granular kidneys to induce mitral stenosis in a heart previously damaged by rheumatism was recorded by me many years ago ; and it is interesting to find that interstitial changes in the kidneys also tend to induce thickening of the tricuspid valve.   It will be shewn later that, in the more severe cases, this proceeds to stenosis.   There were 5 cases in which the kidneys were granular, without stenosis of the mitral orifice (*vide* also p. 341).

B. The second group, that is, the *degenerative one*, may be divided into—(*a*) Cases with widespread degenerative fibroid change affecting several organs, such as the valves (aortic, mitral, and sometimes the pulmonary), the arteries, the kidneys, the liver, and the elastic tissues of the lung.   Of such widespread degenerative change there were 15 cases; but, most probably, the succeeding groups should be considered merely as subdivisions, although the fibroid change is limited to one organ.   (*b*) Cases of emphysema, obstructive lung disease, dilated bronchial tubes, fibroid lung, or tuberculosis of the lungs : of emphysema there were 25 cases ; of fibroid phthisis, 7; of fibroid lung, 4; and of emphysema, with granular kidneys, 17.   (*c*) Cirrhotic liver and perihepatitis, 3 cases. (*d*) Granular kidneys, 16 cases, to which reference has already been made.

From a pathological point of view also emphysema should not be considered solely as a disease of the lung, but in many cases rather as part of a widespread degenerative change affecting more particularly the elastic tissues of the body, and damaging the various viscera secondarily by means of arterial degeneration.   Amongst groups of cases we note the association of emphysema with bronchiectasis, fibroid lung, granular kidneys, and cirrhosis of the liver ; and, in 8 cases, with thickening of the aortic and sometimes of the mitral valves, in one case with marked stenosis.   Malignant disease, which was present in 4 cases, had probably accelerated the degenerative changes ; twice there was also lardaceous disease.

C. The third group; associated with *pulmonary stenosis*.   Six cases. In these the change resulted from the increased pressure in the right ventricle secondary to the congenital lesion.

3. INFECTIVE ENDOCARDITIS OF THE TRICUSPID VALVES.—From the records at Guy's Hospital, between 1860 and 1906, out of 21,000 post-mortems we have collected 35 cases ; 10 are also recorded in the Pathological Society of London's *Transactions*, making a total of 45.

The age-distribution during the various decennia are 3, 12, 8, 9, 5, 3, 3, and 2 ; the youngest child being only three months old.   The majority of cases as usual were between the ages of 10 and 40.   There were 22 male and 23 female cases.

Etiology.—Cause not determined, 10; pyaemia and inflammatory conditions, 17; pneumonia, 9; puerperal, 3; patent ventricular septum, 2; pulmonary stenosis, 1; chorea for ten years, 1. In 2 cases in which pneumonia was present pneumococci were found in the vegetations, but otherwise there are no notes as to micro-organisms. It is doubtful how far it is legitimate to separate acute vegetations from infective endocarditis. In 2 cases with large fungating vegetations on the mitral and aortic valves, there were minute vegetations the size of pins' heads on the tricuspid. In both there was old thickening of the valves on the left side from rheumatism.

No definite history of rheumatism was found in 19, a definite history in 9.

Associated Valve Lesions.—Other valves all healthy, 12; vegetations on all valves, 4; only on pulmonary, 1; on aortic, 16; on mitral, 19 (valves also thickened, 6); vegetations on sclerotic tricuspid valves, 9; sclerotic aortic valves, 6; sclerotic mitral, 6 (and with vegetations, 8); acute pericarditis, 7; adherent pericardium, 5.

Murmurs.—No note, 14; no cardiac bruit, 4; no tricuspid bruit, 10; loud systolic bruit, 5; diastolic bruit, 1.

In the majority of cases the valves were incompetent, yet in only 5 cases was a systolic tricuspid murmur noted, and this corroborates the fact, mentioned under tricuspid incompetence, that there is not necessarily a systolic murmur when the right side is dilated; as although the blood may flow back the muscle may often be too feeble to produce a murmur.

4. Tricuspid Incompetence.—This is by far the most frequent right-sided valvular lesion.

Pathology.—(A.) *Functional.*—Sir D. Macalister shewed that the size of the auriculo-ventricular orifice can be reduced to one-half by contraction of the ventricle, and so long ago as 1880, Dr. G. A. Gibson proved that the pulmonary valves became incompetent when exposed to a pressure of more than 8 inches of a solution with a specific gravity of 1050, but that when the slightest external support was given they would stand a pressure of 6 feet. The tricuspid orifice gushed with a pressure of 12 inches, and at no pressure were they competent after death unless supported outside. Hence it can readily be understood that the tricuspid orifice leaks very easily on the slightest strain, and W. King drew attention to this and to the moderator band on the right side of the heart in 1837. This relative insufficiency may arise (*a*) when holding the breath as the right auricular dulness may increase an inch to the right, from exertion, and from any cause that raises the pulmonary pressure; (*b*) from failure of the nutrition of the cardiac muscle, as in the various forms of anaemia, fevers, local heart disease. Slight leakage may be indicated by a soft local systolic bruit over the 3rd and 4th left spaces. In some cases there is no discomfort, in others dyspnoea and palpitation. Stadler produced incompetence of the tricuspid in

rabbits yet neither oedema nor ascites developed, hence these symptoms, when present, may be due to associated lesions.

B. *Organic.*—When primary, the valve lesions may result from infective endocarditis, due to puerperal infection, gonorrhoea, or pneumonia ; and, occasionally, from the regurgitant stream driven through an imperfect ventricular septum. Eight per cent of cases of infective endocarditis involve the tricuspid orifice.

In a second group of cases the valves are thickened and shrunken, owing to chronic changes the result, sometimes, of rheumatism, but more frequently of chronic atheroma following increased ventricular pressure, and forming but one item in widespread fibroid degeneration. This increased ventricular pressure may result from (*a*) mitral incompetence, and, still more frequently, from mitral stenosis ; (*b*) from chronic bronchitis and emphysema, cirrhosis of the lung, or bronchiectasis ; and (*c*) in the final stages of granular kidneys or of chronic myocardial degeneration, whether the result of alcoholic poisoning or of arterial changes.

Third Group.—The valves may be healthy, but the orifice dilated. This occurs in connexion with some of the conditions above noted, but it is also frequently observed as the temporary result of cardiac dilatation, whether due to disease or over-exertion (Clifford Allbutt). The size of the auriculo-ventricular orifice varies with the efficiency of the muscular contraction of the right ventricle, and a very slight increase in size is sufficient to allow leakage through the orifice. This is looked upon as a safety-valve action, and is considered of great importance in relieving the strain upon the heart on occasions of sudden exertion. This temporary incompetence is probably of frequent occurrence.

The size of the tricuspid orifice may become permanently enlarged as the secondary result of other lesions. According to Hamilton's measurements, the greatest dilatation of the tricuspid orifice is associated with a dilated aortic orifice with incompetent valves, or with a dilated mitral orifice. Hamilton's measurements also shewed that bronchitis, emphysema, fibroid lung, and chronic Bright's disease often fail to produce much dilatation of the tricuspid orifice. The tricuspid orifice may be dilated up to as much as $6\frac{1}{4}$ inches. Some authors lay stress on the not infrequent occurrence of tricuspid incompetence in connexion with digestive disturbance, whether in the stomach or in other parts of the alimentary canal.

That tricuspid incompetence may occur during fetal life is proved by the following case, in which Professor Peter heard a rough murmur followed by a sharp sound, instead of the normal fetal sounds, when auscultating the abdomen of a pregnant girl, aet. 17, at full term. It was thought probable that the tricuspid was the valve affected ; and at the necropsy the tricuspid valves were found covered with abundant vegetations and the flaps were shrunken.

During a period of twenty-five years, out of over 11,000 necropsies at Guy's Hospital there were 405 cases of tricuspid incompetence, excluding cases of stenosis. In 12 cases the reports were imperfect.

They may be classified as follows :—

A. 200 cases. *Left-sided Failure with Valvular Disease.*—Mitral regurgitation with mitral endocarditis or adherent pericardium, 64 ; mitral stenosis, 66; mitral and aortic disease, 61 ; valvular lesion not named, 9. The tricuspid incompetence occurs late and is unusual with aortic disease if the orifice is not dilated: it is more common and occurs sooner with mitral disease.

B. 71 cases. *Left-sided Failure without Valvular Disease.*—Bright's disease, 46 ; malignant disease, 8 ; cirrhosis of liver, 11 ; various, 6.

C. 56 cases. *General Muscular Failure of the Whole Heart.*—Fibroid or fatty heart, 19 ; in the course of acute rheumatism, 17 ; in the course of acute fevers, 20 (pneumonia, 8 ; diphtheria, 6 ; typhoid, 2 ; typhus, 1 ; scarlatina, 1).

D. 7 cases. *Right-sided Failure with Pulmonary Valvular Disease.*—Pulmonary stenosis, 5 ; pulmonary incompetence, 2.

E. 55 cases. *Right-sided Failure without Disease of other Valves.*—Bronchitis and emphysema, 21 ; cirrhosis of lung with and without tubercle, 18 ; bronchiectasis, 4 ; fatty or fibroid right heart, 12.

F. 4 cases. No cause found.

An examination of the clinical reports for six years—235 cases, that is, 40 cases per year—gives the following results :—

In only 2 cases was dyspnoea not mentioned, and in these there is no note of its absence.

In 84 a systolic bruit was noted ; in most reports there is no definite note about a tricuspid bruit.

In very few reports was the condition of the pulmonary second sound noted ; in 10, of which 3 were cases of mitral stenosis, a diminution in intensity was remarked. Oedema was present in 200 ; ascites in 140 ; oedema alone in 76, and together with ascites in 124 ; ascites alone in 14, neither in 21 ; cyanosis in 107 ; venous pulsation in neck in 41 ; hepatic pulsation in 15.

Physical Signs.—(i.) A *systolic bruit*, best heard in the fourth and fifth spaces to the left side of the sternum, traceable sometimes to the right and for a short distance upwards, but very rarely as high as the third rib on the left ; not traceable out beyond the apex ; occasionally, however, best heard in the fifth space to the right of the sternum. Not infrequently the bruit is faint and soft, and often of a character quite distinct from the systolic bruit at the apex. Occasionally it has been noted as loud, when it may be audible over a wide area. The bruit is increased with expiration, and may cease during forced inspiration. It is not audible in the back, a character distinguishing it from a mitral bruit. In cases of relative incompetence the feeble contraction of the right heart may be insufficient to produce a bruit ; hence in many cases its presence is variable, and very frequently no bruit has been heard during life, although at the necropsy the evidence of incompetence was indubitable.

(ii.) Regurgitation through the right auriculo - ventricular orifice

produces *dilatation of the right ventricle*, just as mitral incompetence dilates the left ventricle ; but hypertrophy results much less rapidly, as there is so much less muscle ; and a hardening of the wall, from the venous engorgement, is the more notable change.   The right auricle becomes overdistended, hence the cardiac dulness extends in the fourth space beyond the right side of the sternum, even as far as the parasternal line ; but in many cases this increase is masked by an emphysematous condition of the lung.

(iii.) Except in cases of extreme cardiac failure with advanced emphysema, *pulsation will be both visible and palpable at the epigastrium* and also to the right of the sternum.

(iv.) *Pulsation of the Veins.*—Pulsation from time to time may be noted in the veins, especially in the internal jugular on the right side just above the clavicle.   It is most marked in the recumbent position.   This may be due to various causes, some of which are not of any pathological import-ance.   The veins become distended normally during expiration especially with dyspnoea.   We are still somewhat in the dark as to the conditions
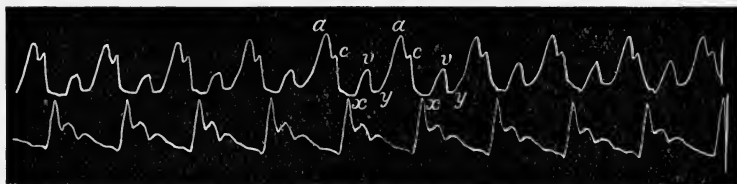
Fig. 30.—Simultaneous tracings from the radial artery below, and from the jugular vein above. The latter shews a well-marked auricular wave, the ventricular wave is the smaller, and its com-mencement is late in the systole. (Mackenzie.)

which determine the appearance of the pulsation.   It may be present in health, at varying times with illness and after exertion and emotion, but especially in cases of right-sided failure, yet it is by no means always present with tricuspid incompetence.   Pulsation is only visible in the veins of the neck when the right heart no longer efficiently drives the blood into the lungs.   A systolic impulse is also transmitted to the veins by the underlying artery.   This impulse may be eliminated by compressing the proximal part of the artery, and noting the cessation of the pulsation.   With venous pulsation the collapse is more sudden than the distension, the converse is true of an arterial pulse.   The jugular pulsation can be much increased by pressure upwards on the liver (Pasteur).

We are mainly indebted to Drs. James Mackenzie, G. A. Gibson, Riegel, Wenckebach, etc., for our present views on this matter.   Dr. James Mackenzie has kindly supplied the blocks for the three subjoined tracings.

There are two types :—(1) The more common *auricular* type (Fig. 30) (sometimes called the normal, negative, or physiological venous pulse). Two main venous waves are recorded, an auricular *a* corresponding to the

systole of the auricle, and a ventricular v, which always ends with the opening of the tricuspid valves, but which commences at a varying period of the ventricular systole. This wave is due to the stasis of the blood that has flowed into and filled the auricle and veins from the periphery, while the tricuspid valves are closed : c is a pulsation transmitted from the carotid artery. The fall x is due to the diastolic expansion of the auricles, and the fall y to the diastole of the ventricles; the former is the greater. This type may occur independently of any obvious tricuspid defect, although Dr. J. Mackenzie thinks that the ventricular wave, however small, may be partly due to some tricuspid leakage. Well-marked pulsation is met with in tricuspid incompetence, and is especially marked in tricuspid stenosis.

(2) The *ventricular* (positive or pathological) venous pulse (Fig. 31), in which the auricular wave has disappeared from its normal position in the cardiac cycle. The wave v commences earlier, and occupies most of the ventricular systole; this is evidence of tricuspid incompetence, and occurs when the contraction of the heart starts abnormally, probably at the node in the auriculo-ventricular bundle. The change to this type occurs
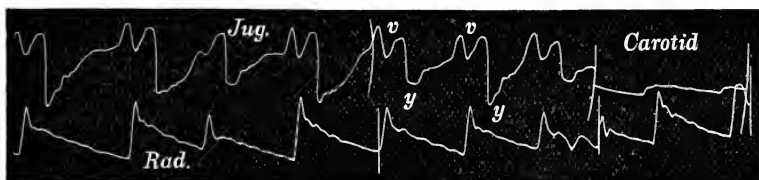
Fig. 31.—Simultaneous tracings of the jugular vein and radial artery, and of the radial and carotid arteries. The venous pulse is of the ventricular type, the auricle being inactive. The ventricular wave occurs at the same time as the carotid impulse. (J. Mackenzie.)

with the onset of irregularity and the disappearance of a presystolic bruit.

Hence with tricuspid incompetence there may either be no venous pulsation, or it may be of either type; of the auricular type when the incompetence is marked and the auricle vigorous; and of the ventricular type when there is a vigorous right ventricle with a large auriculo-ventricular orifice and when the cardiac contractions start at an abnormal place.

(v.) *Hepatic Pulsation.*—A hypertrophied right auricle with a stenosed tricuspid orifice may produce a vigorous hepatic and venous pulse of the auricular type. Such auricular hepatic pulsation is strongly suggestive, though not quite pathognomonic of stenosis, as in two recorded cases it has been due to pericardial mischief. The true expansile pulsation of the liver may be appreciated by taking the liver between the two hands; it is not a mere transmitted pulsation through the diaphragm. Friedreich, Matot, and, in this country, Drs. James Mackenzie and F. Taylor, have fully established the importance of this sign; the left lobe, it may be noted, pulsates more than the right, as the blood more readily regurgitates into that part. Either type of pulsation may occur in the liver, but it

is always the same as that in the veins. To get a well-marked ventricular wave in the liver tracing there must be a vigorous right ventricle with a feeble auricle and marked tricuspid incompetence.

(vi.) *Diminution in the Intensification of the Second Pulmonary Sound.*—This sound is intensified in cases of mitral stenosis or of any chronic lung mischief which impedes the pulmonary circulation—the very conditions which, as stated above, induce tricuspid incompetence. Hence a diminution in the accentuation of this sound is not infrequently of great value, as indicative of the fall in pulmonary pressure brought about when the tricuspid valve begins to become incompetent. It is especially valuable in those cases in which the patient has been under observation for some time, so that both the accentuation and, later, the diminution of the sound have been observed. The sound, however, will still of course generally be louder than normal.
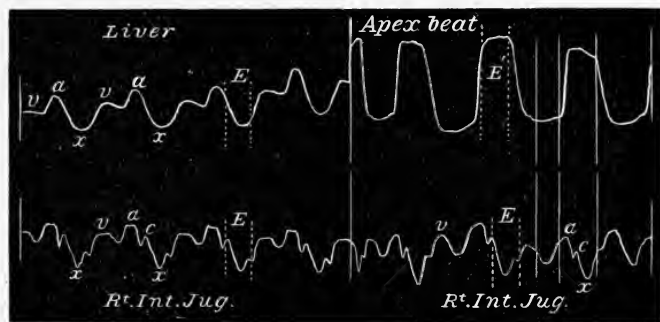
Fig. 32.—Simultaneous tracings of the liver, apex-beat, and jugular pulses, shewing the absence of the carotid wave *c* from the liver pulse ; from a case of tricuspid stenosis and incompetence. The liver pulse of auricular type suggests tricuspid stenosis as the auricle must be hypertrophied to produce it. As there was also undoubted tricuspid regurgitation, the reflux of the blood would hasten the filling of the auricle and help to produce the wave *v* in the tracings. (J. Mackenzie.)

Symptoms.—*Cardiac Dyspnoea.*—This, as pointed out already, is almost invariably present, and in the majority of cases in the form of orthopnoea ; exceptionally, however, cases are met with which are difficult to explain, in which the patient is more comfortable when lying down flat, and is distressed when the head is at all raised. In some of these cases, at any rate, this has occurred when the left ventricle was unaffected, while the right was dilated. The orthopnoea varies in degree, and comes on at night when the patient tries to sleep, or at any time on the least exertion ; it is also much increased by flatulence. In advanced cases the patient is cyanosed, often extremely so, with lividity of the lips, ears, face, and extremities. With this cyanosis it will be found that there is a great increase in the proportion of erythrocytes, not infrequently reaching 8-9 million per c.mm. The surface temperature falls, the pulse is small, irregular, and often impalpable, and the extremities are, owing to the low arterial pressure, bathed in a cold sweat ; albuminuria is not infrequent.

When there is much oedema of the lungs there is a troublesome cough with bloody, foamy, serous expectoration.

The oedema varies in amount, but is generally extreme towards the end, and is most marked in the dependent parts. Ascites is present in more than one-third of the fatal cases. The extreme venous engorgement causes the organs to become enlarged and indurated. The liver particularly is affected.

Diagnosis.—When the three cardinal signs—systolic epigastric bruit, ventricular venous pulsation, and the reduction in the intensity of a previously accentuated pulmonary second sound—are present, there can be no doubt as to the diagnosis ; but in the earlier stages there may be great uncertainty. Duroziez has especially insisted upon the non-transmission of the tricuspid systolic bruit to the back as an important distinction from a mitral bruit. The various conditions under which leakages occur should be borne in mind in forming a diagnosis.

Prognosis.—When the leakage is due to over-exertion, which has induced dilatation of the right ventricle, the prognosis may be favourable, as in these cases the cause is an intermittent one. Not infrequently a soft systolic bruit may be heard over the middle of the sternum for a few minutes and then disappear as the heart quiets down. On the other hand, in many cases it is the final scene in the failure of the heart. The prognosis will then mainly depend on the condition of the heart-muscle and on the severity of the associated lesions ; hence the evil prognosis when the heart has long been struggling against the extra work thrown upon it by other valve lesions. The muscle no longer responds to digitalis, and diffusible stimulants do but postpone death.

Treatment.—In cases in which the incompetence has arisen from dilatation of the ventricle, and not from organic disease of the tricuspid valves, there is no drug equal to digitalis. Under its use in such cases it is not infrequent for a tricuspid systolic bruit to disappear within twenty-four or forty-eight hours ; the pulse is slowed and diuresis increases ; but when dyspnoea and oedema are present they disappear more slowly. Venous pulsation of the auricular type may quickly disappear, but when there is a ventricular venous pulse, which indicates a paralytic condition of the right auricle, the return to the auricular type is a very gradual change. The lesion is organic when the bruit and venous pulsation persist in spite of improvement in the other symptoms. The interesting observation has been made by Potain that occasionally in such cases the administration of digitalis will be followed by haemoptysis for a few days. He offers the following explanation ; namely, that the returning competence of the tricuspid valve throws a greater strain upon the pulmonary vessels, and thus causes a rupture of the smaller branches ; but that later, as the right heart regains its power, the normal condition of the circulation becomes re-established, and the excessive arterial pressure in the lungs returns to the normal.

The patient is generally best in bed, but not infrequently, when the orthopnoea is intense, he may be less miserable in a comfortable arm-chair, well wrapped up, particularly when the legs require to be punctured. Digitalis may be given as Nativelle's digitaline in 1 milli-

gram dose, and repeated in four or five days; or 0·25 mg. daily, and gradually increased : or it may be given in the form of a fresh infusion (℥j) or ℥x. of the tincture every three or four hours, if preferred. Strophanthus and convallaria are of much less value. Venesection, wet cupping, or leeches often render the greatest service, mainly in cases of acute dilatation, when there is a rapid, irregular, feeble pulse, and are especially indicated when there is still a vigorous epigastric pulsation. Cardiac dyspnoea may be greatly relieved by morphine and diffusible stimulants, by hot poultices to the heart, and sometimes by belladonna.

Paracentesis of the abdomen and the drainage of the oedematous legs, either by acupuncture, Southey's tubes, or incisions, are not infrequently necessary before the end ; and often give the patient a fresh lease of life for a time. Strychnine, camphor, and diffusible stimulants are also of great value. Not infrequently fluid which has insidiously accumulated in the pleura requires to be drawn off. Many water-logged patients suffer much from persistent attempts to feed them. They certainly do better if not given too liquid a diet, and as they often fail to digest the food it should be limited in amount. Most remarkable results may sometimes be obtained by stopping all food for thirty-six to forty-eight hours and only allowing the patient water. The oedema and ascites will disappear and the kidneys again act efficiently. All the nourishment the tissues require can be amply supplied by the effusions. Calomel in doses of 1 to 2 grains, three times a day for three days, is often capable of re-establishing the excretion of urine ; but in cases of diseased kidneys much smaller doses should be given. Constipation is especially injurious and must be prevented ; many patients, especially when the liver is large and tender, benefit by free purgation from time to time.

5. TRICUSPID STENOSIS.—Although this lesion is decidedly rare it is much more common than any lesion of the pulmonary valves. Both Niemeyer and Skoda state that they had never met with an example, and were only familiar with it through the specimens in the museum at Vienna. Flint also speaks of it as a rare curiosity, yet we have been able from the records of a single hospital to collect 112 fatal cases during a period of thirty-five years, out of 18,000 necropsies.

Our knowledge of the lesion is largely due to Dr. Bedford Fenwick (who in 1881 and 1883 collected 70 cases, in all of which the mitral orifice was stenosed), and later to Leudet, who in 1888 collected 114 fatal cases ; to these Ashton and Stewart, in 1895, added 17 more, and Prof. W. Griffith, in 1903, 19 ; in both of these series there was mitral stenosis in every case. Their conclusions practically agree with those given later on. In 1908 W. W. Herrick brought the number of reported cases up to 187.

Of Leudet's cases, in 57 there was no preceding illness ; and in 41 a history of rheumatism. The lesion was four times as frequent in women as in men. Two-thirds of the cases occurred between the ages of twenty and forty. A presystolic bruit was heard in 13 cases.

Leudet points out that mitral stenosis was almost always present, but gives 11 as the number of cases in which it was absent; this number should be considerably reduced. Of these 11 cases there was infective endocarditis in 5, and 3 were very imperfectly reported. One was a remarkable case, published by the late Sir William Gairdner (in 1862), of a tumour which had invaded the orifice and given rise to a presystolic bruit; the stenosis was diagnosed ten years before death. (The case was really one of tricuspid obstruction, and not of stenosis.) In one case (No. 116) the aortic valves were involved, and, excluding the cases of infective endocarditis, in only one case, that of Torres Homen (No. 102), were the other valves healthy. This list has also been criticised by Prof. W. Griffith, who shews that it presents evidence of the association with mitral stenosis in almost all sufficiently reported cases. Dr. Philip has recorded a case in which there was a mass of fungating vegetations on the tricuspid valve, which also shewed signs of old stenosis, while the other valves were healthy; and Prof. Delépine one in which a mass of calcified clot blocked the tricuspid orifice, and another of infective endocarditis with calcification of the valve. Apparently calcification of vegetations has taken place in several cases of right-sided lesions, and probably is more rapidly produced there than on the left side.

Etiology and pathology of tricuspid stenosis (109 cases).

(1) *Age-Distribution.*—Only one patient was less than ten years old, and only three more than sixty.

> Between 11 and 20 years, 19 cases.
>   ,,   21 ,, 30 ,, 41 ,,
>   ,,   31 ,, 40 ,, 29 ,,
>   ,,   41 ,, 50 ,, 12 ,,
>   ,,   51 ,, 60 ,, 4 ,,
>   ,,   61 ,, 70 ,, 3 ,,

From this it is clear that more than a third of the cases proved fatal between the ages of twenty and thirty, and more cases ended in death between the ages of thirty and forty than between ten and twenty; this corroborates the conclusion that the lesion in the majority of cases is the result of severe rheumatism, but requires many years for its production.

(2) Tricuspid atresia and more rarely stenosis may occur in a malformed heart in association with pulmonary stenosis or an imperfect ventricular septum (Rauchfuss, Leudet). In many specimens of pulmonary stenosis, even of the most extreme degree, the tricuspid valve is normal or but slightly thickened—the explanation being that the associated abnormal deficiency of one of the septa relieves the right side of the heart from what would otherwise be an excess of pressure. But whether we judge from published cases, from the continuous records at Guy's Hospital for over thirty-four years, or from an examination of all the specimens which have been saved in the London museums, we should conclude that only very few are due to a congenital defect, and

then it is probably always associated with other congenital lesions, and the children are either stillborn or only survive their birth a few days.

Out of 109 cases dying in the hospital only one patient was under ten years of age, a fact which is quite opposed to the lesion being of congenital origin. When the foramen ovale is patent the tricuspid is generally normal, when the ventricular septum is defective it is usually thickened.

(3) *Sex.*—72 women ; 36 men.

(4) *The associated valvular lesions* were as follows :—(*a*) In every case except two there was *mitral stenosis.* An examination of the records shews that the stenosis of the mitral is far more severe than that of the tricuspid. Of the two cases in which mitral stenosis was absent, in one the valves were said to be thickened ; and in only one case of the whole series were they said to be healthy.

(*b*) Aortic valves—thickened, 36 ; stenosed orifice, 27 ; incompetent, 29 ; recent vegetations, 10.

(*c*) Pulmonary valves were thickened in 11 cases ; in 2 there were recent vegetations ; in 24 the pulmonary artery was atheromatous, in 3 it was thickened, and in 2 dilated.

The general conclusions to be drawn are—

(*a*) That, almost without exception, tricuspid stenosis is the sequel of preceding stenosis of the mitral orifice (omitting infants which had not lived one week).

(*β*) In almost half the cases there is evidence of some change in the aortic valves also.

(*γ*) In a large number the aortic valves are thickened, not infrequently they are incompetent, and in a very fair number of cases there is a very well-marked stenosis.

(*δ*) In all such cases the evidence is in favour of the changes being the effects of rheumatism, extreme either in its severity or in the frequency of the attacks. In fact, rheumatism dominates the lesion to the exclusion of every other cause.

(5) *The Relation of Tricuspid Stenosis to Mitral Stenosis.*—In 76 cases the mitral orifice admitted one finger only, or its circumference did not exceed 2 inches ; shewing that in two-thirds of the cases a severe form of mitral stenosis predominated. The amount of the tricuspid stenosis in 48 cases was such that the orifice admitted two fingers only, that is, its circumference was not more than 3 inches. In 8 cases it was extremely small, admitting not more than one finger.

(6)[1] *On the Relation to Rheumatism.*—In 24 cases there had been only one or two attacks of acute rheumatism. In 18 cases there had been more than two ; in many cases the attacks had been numerous ; in 6 there was a history of vague rheumatic pains. In 16 it is definitely stated that there had been no rheumatism, but in 12 there had been one or two attacks of chorea.

---

[1] The remaining figures are based on 87 of the cases.

(7) *Duration.*—In 3 cases there was a history of heart trouble for over twenty years. In 6 cases there was a history of symptoms for five to ten years; in 28 cases of symptoms for one to five years; in 3 cases for rather less than a year; and in 8 cases the symptoms had been observed for less than three months. Twice the patients were quite well within six weeks of death; twice within three weeks of death; once within ten days; and one patient was supposed to have been in good health until his death took place.

Duroziez considers that the length of life varies with the degree of
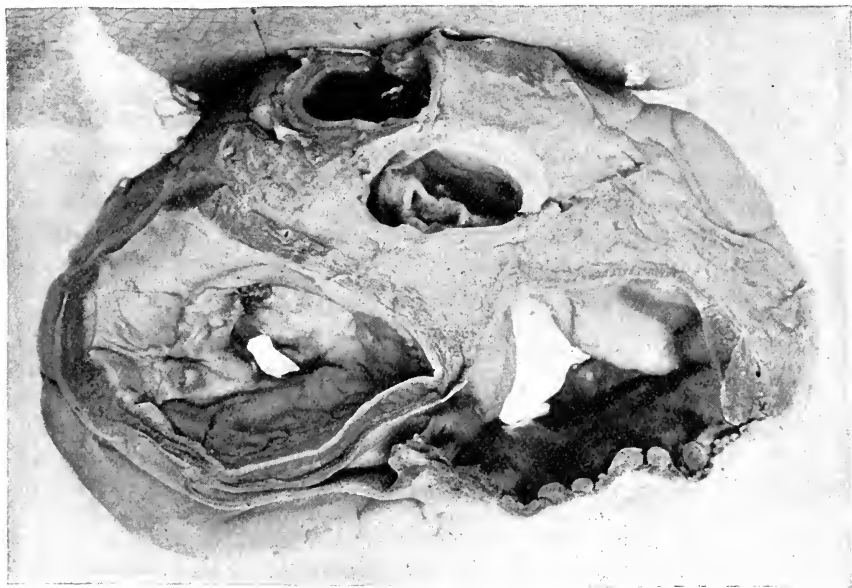
Fig. 33.—A transverse section of a heart viewed from above, shewing, underneath the extremely stenosed mitral orifice on the left, the moderately stenosed tricuspid on the right; and above, the aortic valves fused into a dome.

From a man aged nineteen, who died in 1878 under Dr. S. O. Habershon. He gave no history of rheumatism. The chief symptom was haemoptysis. There was an apical diastolic bruit with a thrill, and a loud basic systolic bruit. The heart weighed 16 ounces. The tricuspid orifice admits two fingers, and the cusps are much thickened. The mitral orifice is still more stenosed, with a calcareous deposit in its wall. The aortic valves are blended together into a dome-shaped septum, with a small central aperture, but were still capable of supporting a column of water.

stenosis; the average age at death, when the orifice would not admit more than one finger, being thirty-two years, and forty-two when the orifice admitted two fingers.

(8) *On the Size of the Heart.*—In 6 cases the heart weighed less than 10 ounces; in 29 cases from 10 to 15; in 10 cases between 20 and 25; in one case it exceeded 30 ounces, and in three the weight was between 25 and 30 ounces.

(9) *Associated Lesions.*—In 38 cases there was brown induration of

the lungs, and in 37 marked pleuritic adhesions; in 33, effusion into
one or both pleural cavities. The lung was oedematous in 13; in 12
there was recent inflammation over the surface of the pleura. In 27
there were pulmonary apoplexies, but in only 4 were thrombi found in
the pulmonary artery. Twice there was bronchiectasis, twice emphysema,
and only twice phthisis. Two cases were especially remarkable: in one
it is stated that there was a complete absence of any induration of the
lung, such as is usually met with in cardiac failure, yet there had been
symptoms of mitral failure for two years, and the mitral orifice would
only admit one finger. In another patient, who died from septic
bronchopneumonia with secondary peritonitis, there were no previous
symptoms of any cardiac disease.

In the case in which the mitral valve was stated to be only thickened,
but otherwise healthy and competent, there was emphysema with dilata-
tion of the bronchial tubes. The tricuspid valve was much thickened
and atheromatous, and its orifice contracted. The aortic valve was
thickened and somewhat calcified, but competent; and there was a
history of two attacks of haemoptysis.

The pericardium was adherent in 38 cases; there was recent peri-
carditis in 10. The comparatively frequent occurrence of perihepatitis
in these cases has not been previously noticed. It was noticed that in
43 of the cases the kidneys were granular, and in two there was acute
nephritis. Especial attention was directed to the exclusion of cases of
scarred kidneys, of which there were many; as some may have been
the secondary results of the mitral stenosis. This entirely unanticipated
result corroborates the views put forward in 1887 on the important
influence which granular kidneys have in the production of mitral
stenosis in adults; it would appear that they have also a potent influence
in the production of stenosis of the tricuspid orifice. In three cases the
suprarenals were caseous; twice there was acute peritonitis, once due to
renal disease and once to thrombosis. In 2 cases infective endocarditis
was present.

Symptoms.—The symptoms most frequently met with are dys-
pnoea, oedema, albuminuria, enlarged and tender liver, and cyanosis; the
order being that of their relative frequency. Dyspnoea was met with in
73 cases; in 19 of these it amounted to orthopnoea. In 65 cases there
was oedema; in 63 albuminuria; in 47 the liver was enlarged; and in 45
there was cyanosis. Ascites was only present 30 times. In 32 cases it
is expressly stated that there was no cyanosis; in 24 the liver was not
enlarged; and in 23 there was no albuminuria. In 15 cases there was
no oedema. The pulse was small, often impalpable, irregular, and rapid.

Haemoptysis was noticed in 20 cases. This is due to the associated
mitral stenosis and not to the tricuspid lesion; in 8 of these pulmonary
apoplexies were found at the inspection. A jaundiced condition of the
skin was noticed 14 times; petechiae 4 times; extreme pulsation in the
veins of the neck 4 times; and once extreme distension of the veins
generally.

An examination of the records brings out the remarkable fact that, of the symptoms oedema, cyanosis, and albuminuria, it was not at all infrequent for one to be absent, while the other two were present; but it has not been possible for us to determine the corresponding differences in the pathological lesions. Why one patient with mitral stenosis, or marked stenosis of both mitral and tricuspid valves, suffering with dyspnoea and oedema, should have no cyanosis, whilst another cyanosed and dyspnoeic should have no oedema, it is difficult to say.

There are some other points of interest; for example, in 12 cases the liver was cirrhosed, and in 11 cases there was perihepatitis with marked thickening of the capsule of the liver. The fact that 15 of the patients were free from oedema is of interest, as we also know that with pure mitral stenosis oedema is most exceptional. It is worth bearing in mind that in more than a quarter of the cases the liver was not found to be enlarged, and in 23 there was no albuminuria. True expansile pulsation of the liver, due to associated tricuspid incompetence, was only recorded eight times. It is difficult to understand why seven of the patients were free from dyspnoea, and why more than one of them was not ill till within three weeks of death. Glycosuria was noticed only once. While certain deductions may with confidence be drawn from statistics based upon observations made by medical students, still undoubtedly the frequency of common symptoms must inevitably be understated.

Concerning the *bruits* which were heard : the difficulty of diagnosing cases may be realised when it is pointed out that in 20 cases—that is, in nearly a quarter of the whole—no bruits were specially noted in reference to the tricuspid area in the region of the ensiform cartilage. In 10 cases a systolic bruit, widely distributed over the cardiac area, was noted. In 5 cases no cardiac bruits were noted while the patients were under observation; and in one case there was some doubt as to the presence of a cardiac bruit. In 28 there were bruits audible near the ensiform cartilage on the left, and sometimes on the right side of the sternum, which were distinct in character from those heard elsewhere. The bruits were described as high-pitched, as distinct in character, and sometimes as rougher than those heard in the mitral area; several times as very distinct, occasionally as loud, and sometimes as harsh. In scarcely any case is it stated that the bruit was of a lower pitch than the mitral, as has been suggested by certain German observers. In several of the cases the bruit has been limited to a very narrow area; and not infrequently a thrill has been palpable. In only 10 cases out of the whole series was a presystolic or mid-diastolic bruit heard in the tricuspid region. In some of these it was doubtful, and in 2 cases it was heard on one occasion only. It was, however, very definitely stated in some reports that the presystolic bruit was distinct in character, and its area of maximum audibility definite. In one case the registrar described the systolic bruit as distinct from the mitral one, more harsh, and followed by a faint bruit which ran up to the second sound. When we bear in mind that a vigorously acting auricle is

required for the production of the presystolic bruit in mitral stenosis, we need not wonder that it is infrequent in cases of right-sided stenosis ; the right side works at a very much lower pressure, because the auricle is thinner and contains less muscular fibre which can hypertrophy. Most authors state that a presystolic bruit should be heard ; but clinical experience shews that such a bruit occurs in less than 10 per cent of the cases of tricuspid stenosis. A localised, definite systolic bruit is much more frequent ; and in half the cases no characteristic bruit is detected. As a natural sequence of this it follows that the lesion usually remains undiagnosed, and, not unfrequently, unsuspected. The contraction of an incompetent orifice seems to be beneficial, and Dr. Mackenzie has watched cases with systolic hepatic pulsation for some years.

General Conclusions.—1. Tricuspid stenosis is rare, but not excessively so ; and in adults is not of congenital origin.

2. It is almost invariably preceded by mitral stenosis, and in one-fourth of the cases there is also aortic stenosis. A very well-marked example is shewn in Fig. 33, in which there is marked stenosis of the mitral and aortic orifices and moderate stenosis of the tricuspid.

3. The degree of stenosis of the mitral is generally more severe than that of the tricuspid.

4. The principal cause is a severe form of rheumatic endocarditis, but a history of rheumatism is only obtainable in about half the cases.

5. The stenosis generally advances insidiously, and in exceptional cases has been present in patients feeling fairly well.

6. Dyspnoea is almost invariably present, oedema and albuminuria in two-thirds, enlarged liver and extreme cyanosis in one-half of the cases. The presence of oedema and albuminuria with mitral stenosis suggests some further lesion.

7. A presystolic bruit at the lower end of the sternum is only heard in a small proportion (one-eighth) of the cases ; a localised epigastric systolic bruit is much more common, whilst in one-quarter no bruit has been noted.

Diagnosis.—If we bear in mind that this lesion is almost invariably found in connexion with mitral stenosis, this may not infrequently assist us to make an accurate diagnosis. The stenosis arises most insidiously ; and to determine the diagnosis we must sometimes be satisfied with the collateral symptoms of great cardiac failure—dyspnoea, oedema, enlarged liver, and notable cyanosis. The presence of a characteristic bruit must be repeatedly hunted for, and if a well-marked auricular venous pulsation in the liver be recorded, it would be conclusive. Often the veins in the neck will be persistently full, without much pulsation. Marked increase of dulness to the right of sternum, without much epigastric pulsation, is common (Griffith).

Treatment.—The treatment is practically that already indicated for mitral stenosis and tricuspid incompetence, but the beneficial results are much less apparent.

PRIMARY TUMOURS AND PAPILLARY EXCRESCENCES ON THE VALVES. — Occasionally smooth, solitary tumours, and very rarely papillary excrescences, have been noted on the inner surface of one of the valves, while the rest of them have been normal. The smooth tumours are derived from organised thrombi, and the papillary excrescences are probably the results of local endocarditis ; but it has been suggested that some of them are primary myxomas. They are generally covered with a layer of endothelium ; they may be either sessile or pedunculated, and may be slightly grey-red in colour. Microscopically they consist of fine parallel fibres with numerous round and spindle-shaped cells, and some of them have been described as shewing a myxomatous structure. Koechlin and Leonhardt have collected the recorded cases. The majority have occurred in the left auricle, but three have been noted on the pulmonary valves, two by Hedinger and one by Ribbert; and five on the tricuspid valve, three by Debove, Reitmann, and Guth, and two by Ribbert.

G. NEWTON PITT.

REFERENCES

1. ALLBUTT. "On Overwork and Strain of the Heart," *St. George's Hosp. Rep.*, London, 1870, v. 23.—2. ASHTON and STEWART. "Report of a Case of Tricuspid Stenosis, associated with Mitral Stenosis and Aortic Stenosis," *Am. Journ. Med. Sc.*, Philadelphia, 1895, cix. 177.—3. BARIÉ. "Sur l'insuffisance des valvules de l'artère pulmonaire," *Arch. gén. de méd.*, Paris, 1891, clxvii. 650. — 4. BLATTMANN. *Zwei Fälle von Insufficienz der Pulmonalarterien - Klappen*, 1887 ; Dissertation, Zürich.—5. BRAMWELL. "On right-sided Endocarditis," *Am. Journ. Med. Sc.*, Philadelphia, 1886, cxi. 419.—6. BRYANT, J. H. "Functional Pulmonary Incompetence as a Complication of Mitral Stenosis," *Guy's Hosp. Rep.*, 1901, lv. 83.—7. CHARCOT-BOUCHARD. *Traité de médecine*, 1893, vol. v.—8. COUPLAND. "Diseases of the Pulmonary Valves, etc.," *Trans. Path. Soc.*, London, 1875, xxvi. 22.—9. DEBOVE. "Myxome pédiculé developpé sur la valvule tricuspide," *Bull. Soc. anat.*, Paris, 1873, xviii. 247.—10. DILG. "Ein Beitrag zur Kenntniss seltenen Herzanomalien in Ausschluss an einen Fall von angeborener linkseitiger Conusstenose," *Virchows Arch.*, Berlin, 1883, xci. 193.—11. DUCKWORTH, Sir D. "Incompetent Pulmonary Artery with Mitral Stenosis," *Trans. Clin. Soc.*, London, 1888, xxi. 114.—12. DUROZIEZ. *Bull. soc. de méd. de Paris*, 1868 ; *Union méd.*, Paris, 1883, xxxvi. 1084.—13. FENWICK. *Trans. Path. Soc.*, London, 1881, xxxii. 42, and 1882, xxxiii. 64.—14. GAIRDNER. *Clinical Medicine*, Edinburgh, 1862, 602.—15. GEIGEL. "Ueber den Venenpuls," *Würzburg. med. Ztschr.*, 1863, iv.—16. GERHARDT. *Klinische Untersuchungen über Venenpulsation*, Leipzig, 1894 ; "Ueber Schlussunfähigkeit der Lungenarterien-Klappen," *Charité-Ann.*, Berlin, 1892, xvii. 255.—17. GIBSON, G. A. *Diseases of Heart and Aorta*, 1898.—18. Idem. "Jugular Reflux and Tricuspid Regurgitation," *Edin. Med. Journ.*, 1880, xxv. 979.—19. GOURAUD. *De l'influence pathogénique des maladies pulmonaires sur le cœur droit*, Paris, 1865.—20. GRAWITZ. *Virchows Arch.*, 1887, cx. 426.—21. GRIFFITH. "Affections of the Tricuspid," *Edin. Med. Journ.*, 1903, N.S. xiii. 105 ; *Lancet*, 1907, ii. 1147.—22. HERRICK, J. "Tricuspid Stenosis," *Bost. Med. and Surg. Journ.*, 1897, cxxxvi. 245.—23. HERRICK, W. W. "Tricuspid Stenosis," *Arch. Intern. Med.*, Chicago, 1908, ii. 291.—24. HOLMES. *Trans. Clin. Soc.*, ix. 114, and 1881, xxi. 146.—25. KERSCHENSTEINER. "Endocarditis pneumonica der Pulmonalarterial - Klappen," *München. med. Wchnschr.*, 1897, xliv. 808. —26. KING, T. WILKINSON. "The Safety-Valve Action of the Right Ventricle of the Human Heart," *Guy's Hosp. Rep.*, 1837, ii. 132.—26a. KOECHLIN. "Ueber primäre Tumoren des Herzklappen," *Frankfurter Ztschr. f. Path.*, Wiesbaden, 1908, ii. 295. — 26b. LEONHARDT. "Ueber Myxoma des Herzens," *Virchows Arch.*, 1905, clxxxi. 347.—27. LEUDET. *Essai sur le rétrécissement tricuspidien.* Paris, 1888. — 28. MACKENZIE, J. *Diseases of the Heart*, Oxford, 1908. — 29.

Idem.   The Study of the Pulse, 1902.—30. MORISON.   "Dextral Valvular Disease
of the Heart," *Edin. Med. Journ.,* 1879-80, xxv. 102, 439, etc.—31. PAPAS.   *Thèse
de Paris,* 1894.—32. PASTEUR.   "A New Method of Estimating the Condition of the
Right Side of the Heart," *Lancet,* 1886, i. 914.—33. PAUL.   "Rétrécissement de
l'artère pulmonaire," *Gaz. hebd. de méd.,* Paris, 1871, viii. 431.—34. PAWINSKI.
"Ueber relative Insufficienz der Lungenarterien-Klappen bei mitral Stenose," *Deut.
Arch. f. klin. Med.,* Leipzig, 1894, lii. 519.—35. PETER.   *Maladies du cœur,* 1883, p.
612.—36. PITT.   "Association of Mitral Stenosis with Gout and Granular Kidneys,"
Brit. Med. Journ., 1887, ii. 118.—37. POTAIN.   "Insuffisance et rétrécissement
tricuspidiens," *Semaine médicale,* 1891, xi. 346 ; "Des mouvements et des bruits
qui se passent dans les veines jugulaires," *Soc. méd. des hôp.,* March, 1867.—38. RIEGEL.
"Ueber den normalen und pathologischen Venenpuls," *Deut. Arch. f. klin. Med.,* 1882,
xxxi. 1.—39. ROGERS, L.   "Extensive Atheroma and Dilatation of the Pulmonary
Arteries," *Quart. Journ. Med.,* Oxford, 1909, ii. 1.—40. SANSOM.   *The Diagnosis of
Diseases of the Heart and Thoracic Aorta,* 1892.—41. SCHIPPMANN.   "Ueber
angeborene Stenose oder Atresia des Ostium dext.," Virchow und Hirsch's *Jahresb.,*
1889.—42. STEELL, GRAHAM.   "High Pressure in the Pulmonary Artery," *Med.
Chronicle,* Manchester, 1889, ix. 182.—43. WEBER and FÜRTH.   "Malignant
Pulmonary Endarteritis after Gonorrhoea," *Edin. Med. Journ.,* 1905, N.S. xviii. 33.
—44. WHITLEY.   "Disease of the Pulmonary Artery and its Valves," *Guy's Hosp.
Rep.,* 1857, 3rd Ser., iii. 252.

G. N. P.

DISEASES OF THE MITRAL VALVE

By the late A. E. SANSOM, M.D., F.R.C.P.
Revised by G. A. GIBSON, M.D., D.Sc., LL.D.

ALTHOUGH for purposes of convenience obstruction of and regurgitation
at the mitral orifice are considered separately, it must be borne in mind
that, excluding functional incompetence, in the great majority of instances
both disorders are present.   There are, it is true, a certain number of
patients who, at any rate for a time, furnish absolute evidence of mitral
obstruction without any accompanying incompetence ; but even in these
instances regurgitation is almost certain to appear sooner or later.
Incompetence, on the other hand, may be produced by causes and lesions
which, from first to last, exert no influence upon the course of the
blood from the auricle to the ventricle.   In the following pages obstruc-
tion and regurgitation will, in accordance with conventional methods, be
discussed separately, but the considerations which have been mentioned
must be kept in view.

MITRAL OBSTRUCTION

Definition.—A morbid condition of the structures at the left auriculo-
ventricular aperture, causing an obstruction to the normal flow of the
blood from the left auricle to the left ventricle.

History.—From the dawn of morbid anatomy obstruction of the
mitral orifice has been recognised ; thus, the lesions giving rise to the

obstruction were fully described by Morgagni and his successors. The clinical recognition of the disorder was of much later date, and the growth of our knowledge has been chiefly due to the labours of observers in the second and third quarters of the nineteenth century. Corvisart at an earlier period (in 1806) demonstrated the importance of the thrill which is so frequently present, but it was nearly forty years later (in 1843) that Fauvel shewed the significance of the distinctive murmur resulting from the obstruction. He was almost forestalled by Bertin, who seems to have heard the presystolic murmur, and as Hayden says, "actually founded thereon the positive diagnosis of left auriculo-ventricular contraction." But since Bertin believed with Laennec that the second sound of the heart was due to the systole of the auricles, he cannot be regarded as being very definite in his teaching. Adams also, a few years later, elucidated some of the special characteristics of the murmur, in ignorance, so far as can be seen, of Bertin's work. Hope is sometimes regarded as having discovered the diastolic murmur of mitral obstruction. It is quite true that he describes a diastolic murmur, but his phraseology leaves the reader in grave doubt as to his meaning. He says :—" When the valve is contracted, the second sound loses, on the left side, its short, flat and clear character, and becomes a more or less prolonged bellows-murmur." This is very unlike the mitral diastolic murmur as we know it now. In spite of these interesting observations, therefore, the definite recognition of mitral obstruction begins with Fauvel. Since the appearance of his epoch-making work the natural history of the affection has been chiefly elaborated through the investigations of Stokes, Gairdner (28), Hayden, Balfour (4), and Fagge.

Etiology.—Mitral obstruction occurs more frequently in women than in men. The proportion relatively affected in the two sexes, according to different authors, necessarily varies to a considerable extent, but almost all statistics bear out the general statement just made. The admissions to the wards of the Royal Infirmary of Edinburgh during the sixteen years 1893-1908 shew that 324 males and 523 females entered the medical wards on account of pure mitral obstruction. The affection is associated with early life, and the greatest number of admissions takes place between the ages of twenty and thirty. Undoubtedly a very large proportion of such cases have their origin in a period considerably younger even than this, but the symptoms which lead the patients to seek admission do not as a rule become urgent until the third decade of life. The disease is sometimes congenital, dating from fetal life (*vide* p. 291). Endocarditis is, however, far from common before birth, and the affection usually begins after the commencement of independent existence.

The most frequent cause of mitral obstruction is endocarditis. The lesion is, in fact, more closely associated with acute changes than is the case in regard to any other valvular disease. The endocarditis which has given rise to the chronic lesion may arise from any of the numerous causes now recognised, all of which are associated with the presence of micro-organisms, but it is certain that rheumatism in one or other of its

numerous manifestations is the most important factor. The determining cause of the lesion is sometimes quite obscure. In such cases of latent endocarditis the subsequent development of one or other of the protean forms of rheumatism clears up the etiology. Whilst recognising this, it must be admitted that in a considerable proportion of cases the cause of mitral obstruction is not ascertainable. The association of the disease with chlorosis and with tuberculosis is dealt with elsewhere (pp. 345, 372). Whether the nexus is in any real sense of an etiological nature cannot be affirmed. The most important considerations are discussed on the page referred to. Chronic degenerative changes due to sclerotic alterations of the endocardium, absolutely independent, so far as can be ascertained, of acute endocarditis, give rise to a certain number of cases of mitral obstruction. Such instances occur considerably later in life than those of endocardial origin.

Traumatic influences occasionally give rise to the lesions of mitral obstruction. Indirect injury very rarely produces any changes in the mechanism of the mitral valve unless there has been previous disease, but direct violence, such as a blow upon the anterior chest-wall, is not a very rare cause of the affection.

Morbid Anatomy.—In the early stages of the disease comparatively slight degrees of obstruction at the mitral orifice are marked by a ring of vegetations—in some cases friable and easily detached, in others sclerotic and firmly fixed—situated on the auricular aspect of the cusps near their edges. The fibrous structures subjacent to the vegetations are firmer than the normal, the thickening frequently involving the mitral curtains, the chordae tendineae, and the musculi papillares. In a more advanced stage the marginal portions of the curtains are joined by fibrous adhesions. At a still later stage the two curtains are so completely fused together that the valve presents the form of a hollow cone or membranous funnel, the wider portion of which is at the auriculo-ventricular orifice, and the narrower points downwards towards the apex of the heart. The funnel form of mitral stenosis, and the smooth polished membrane, regular in its conformation as a hollow cone, have suggested that the malformation of the valve is a congenital anomaly. It is undoubtedly true that in rare cases such an obstruction of the mitral orifice has been found in association with congenital malformation. In a case of this kind recorded by Parrot, the aorta and pulmonary artery were united in a single trunk. In such cases in which the mitral orifice is found on post-mortem examination to be obstructed in infants who die shortly after their birth, the vegetations of endocarditis are often found. In one of Sansom's cases (76), a baby of two months, a ring of granulations was found encircling the mitral orifice, and the valve was thickened. There can be no doubt that mitral stenosis, as observed in these cases, is not a congenital malformation, but the result of intra-uterine endocarditis—the smooth and regular conformation of the funnel constituted by the cohering curtains of the valve being due to the even pressure of the fluid blood both on the auricular and ventricular surfaces during the rhythmic movements of

the heart. The terminal aperture of the funnel, by which the blood issues into the ventricle, may be extremely small, allowing the passage of nothing thicker than a goose-quill.

The fibrous thickening of the valve, of the chordae tendineae—which may be much shortened as well as thickened—and of the musculi papillares is in some instances very dense ; in many examples these structures present the characters of cartilage. Though the funnel form of transformation of the valve is by far the more common in childhood, the "button-hole" form is sometimes observed ; it has been noted by Hayden in the case of a boy aged seven. The auriculo-ventricular orifice, as seen from the auricular side, then presents the form of a slit or chink, or a crescentic opening in the firm, thick, fibrous septum of the welded valve-structures. The division of cases of mitral stenosis into the "funnel" and "button-hole" forms, first made by Sir R. Douglas Powell (62), is a very practical one from the point of view of morbid anatomy. In some cases, however, the auriculo-ventricular aperture on its auricular aspect presents a very irregular form. It may be surrounded by thickenings and nodosities, and the opening may have a puckered appearance.

The "button-hole" form of mitral stenosis is observed with much greater frequency in adults. In childhood the proportion is about one "button-hole" to eight "funnels" ; in adult age and later life twenty-five "button-holes" to one "funnel." The association with the rheumatic form of endocarditis is abundantly manifested in the necropsies of cases shewing constriction of the mitral orifice in adults. Cases of chronic endocarditis or repeated endocarditis affecting the mitral valve—whether the signs during life have indicated combined stenosis and regurgitation, or regurgitation only—are rarely, if ever, seen without the anatomical examination demonstrating that the left auriculo-ventricular orifice is more or less constricted, and the surrounding fibrous ring firmer than the normal.

In many instances in adult age and later life the fibrous material is infiltrated with calcareous salts, the resulting plates having the hardness of bone. In rare cases the curtains of the valve have been found normal, whilst calcareous plates have been observed in the adjoining muscular wall of the ventricle. These may be associated with atheromatous changes, or may represent syphilitic gummas which have become calcified. In a case of chronic interstitial nephritis the vegetations surrounding a stenosed mitral orifice have been found by Lancereaux to contain urates. Dr. Goodhart, in an analysis of the post-mortem records of 192 cases shewing the changes of chronic interstitial nephritis, found that about one-fourth of the whole number presented either thickening or contraction of the mitral valve. Dr. Newton Pitt observed, on examination of the records of the post-mortem department of Guy's Hospital, that the cases of mitral stenosis in the subjects of granular kidney were to those not manifesting renal lesions in the proportion of three to one. In many cases in this category atheroma of the aorta was also found, more rarely atheroma obstructing the coronary

arteries. Huchard has designated the cases as "rétrécissement mitral artérioscléreux." In some instances chronic fibrotic changes have been found in various situations—in the pleurae, the lungs, the capsules of the kidneys, the liver, the spleen, and the intracranial membranes. Sometimes in association with such conditions the mitral valve presents the funnel form of stenosis. This form is exceptional in the subjects of chronic renal disease, but cases have been recorded. It is obvious that the funnel form of transformation of the mitral valve, the "pure" mitral stenosis of Duroziez and other French observers, is found not only in childhood (when it simulates a congenital malformation), but also in advanced life. In some cases it is certainly associated with rheumatism ; in others such association is not proved ; but it may be found in the subjects of chronic renal disease and of arteriosclerosis.

The left auricle in cases of this affection is frequently hypertrophied and dilated. In some cases the cavity is greatly enlarged, but the walls are thin. In a child aged eight years Sansom found hypertrophy so far advanced that the muscle was a quarter of an inch thick (the normal being about $\frac{3}{20}$ of an inch) ; in another case, that of an aged woman, it was as thin as an ordinary visiting-card, almost destitute of muscle, and lined with laminated coagulum. The appendix of the auricle is usually the portion which manifests hypertrophy in the greatest degree. When, on opening the pericardium, the heart is viewed in position, the hypertrophy of the auricle is in some cases very striking: instead of being flaccid it stands out firm and muscular. On section it does not collapse, and pronounced reticulations mark its internal surface. In other cases, when dilatation preponderates, the capacity of the auricle is increased, in some cases enormously. The pulmonary veins are also greatly dilated and the intima thickened. In Sansom's records of 40 cases of mitral stenosis at all ages observed after death, the left auricle was found dilated in 18, dilated and hypertrophied in 10, and hypertrophied without notable dilatation in 3. Dr. D. W. Samways (68), who examined the records of necropsies at Guy's Hospital for four years, found that in 70 cases of mitral stenosis the left auricle was hypertrophied in 36. In 36 cases of well-marked obstruction— the mitral orifice admitting only one finger or the extremity of a finger— the left auricle was hypertrophied in 26, dilatation coexisting in 14. In 3 cases only was there dilatation without hypertrophy. In the cases of less pronounced stenosis the state of the auricle was precisely noted in 11 only, and of these 5 shewed dilatation without hypertrophy. The conclusion is probably correct that hypertrophy is the rule—with the hypertrophy some dilatation nearly always coexisting, the relation of the two varying with the extent of the obstruction and of the regurgitation. When compensation fails, the muscle becomes enfeebled, and dilatation progressively increases. The dilatation only becomes possible when the tonicity of the cardiac muscle fails.

The endocardium lining the auricle is usually thickened ; in some cases all over—the probable cause then being the excess of blood-pressure to which it is subjected, and in many cases in patches by

chronic endocarditis or atheromatous change. The posterior wall of the auricle is most frequently thus affected. On the internal surface of this part of the auricle coagula are frequently observed. These are sometimes stratified and composed of alternating layers of coloured and colourless fibrin closely adherent to the endocardial surface. In some cases the whole auricle, thus distended with layer upon layer of coagula, resembles an aneurysm.

The vegetations observed on the lining membrane of the auricle may be sessile or pediculated—warty, globular, or polypoid. The warty vegetations are simply coagula of fibrin on the diseased surfaces of the endocardium. Globular thrombi are found especially in the auricular appendix, and between the ·muscular bundles; in rare cases they almost fill the auricle. Their external portion is smooth and tough; on section they are found to contain a creamy fluid. Polypoid thrombi are more rare; they are attached by a pedicle to the wall of the auricle or to the auriculo-ventricular ring. Some, like the globular thrombi, are masses of firm fibrin; others are hard and calcified. Thrombi at the left auriculo-ventricular aperture are found with greater frequency in mitral stenosis than in mitral regurgitation. They may be detached and become emboli, which are arrested at some point in the arterial channels; or one or more may persistently block the aperture; or, again, one may obstruct the orifice, in the manner of a ball-valve, during certain periods of the cardiac cycle (*vide* p. 724).

The left auricle is occasionally so much dilated as to cause obstruction of the left bronchus, and thus to lead to the series of morbid changes in the lung which result from its partial or total occlusion. These pass through the stages of hyperaemia and oedema until the final conditions of collapse and induration are reached. A greatly dilated left auricle may exert pressure on the left recurrent laryngeal nerve (*vide* p. 363). Pressure on the left subclavian, with relative weakness of the left radial pulse, has also been described as due to a dilated left auricle (Popoff).

The *pulmonary veins* are in some cases much dilated; their coats may be thickened and sclerotic. Sir James Barr has described well-marked changes in the pulmonary veins in cases of mitral stenosis.

The *left ventricle* in the majority of cases presents characters which do not obviously differ from the normal; its cavity is not enlarged; in some instances its capacity is less than the normal. In the cases of young children the smallness of the left ventricle is striking; in some of these patients the whole heart is correspondingly diminished in size, the lungs are small, and the thoracic capacity reduced. On account of the imperfect blood-supply to the ventricle the whole organism has been impoverished (Wilks), and the entire economy has suffered from arterial starvation. In other cases the contrast with the large and muscular left auricle is very obvious. In about three-fourths of the cases observed after death the wall of the left ventricle is not hypertrophied. When hypertrophy is manifest, as in the remaining fourth of the cases, there is usually an obvious concurring cause—in the young pericardial adhesions, in the old

chronic renal disease or arteriosclerosis.   Globular thrombi are sometimes found in the interstices between the musculi papillares of the left ventricle remote from the valve.

With the exceptions above noted, when death has occurred in the period of childhood, the *right cavities* are dilated in marked degree, and the walls of the right ventricle and right auricle are hypertrophied.   The hypertrophy is often evidenced by the massive muscular columns in the ventricle and the thick interlaced muscular bands in the auricle.   The orifice guarded by the tricuspid valve is, as a rule, abnormally wide ; the valve in some cases is competent to close this orifice, in others its incompetence is obvious ; indeed, cases have been recorded of such dilatation that auricle and ventricle appeared to form one enormous cavity.   The *pulmonary artery* is often dilated and its intima thickened.

Thrombi are observed in the right auricle and right ventricle in many cases ; the surfaces of the endocardium, on which they are formed, are not necessarily diseased.   Such thrombi, when they become detached, plug the larger or smaller branches of the pulmonary artery.   Their inception is no doubt due to the retardation of the blood-flow.   The chain of consequences is as follows :—Obstruction at the mitral orifice, abnormal strain of the walls of the left auricle, auricular hypertrophy and dilatation, obstruction to the blood-flow from the pulmonary artery to the pulmonary veins, increased labour of the right ventricle, tension of its walls, hypertrophy and dilatation of right cavities (*vide* p. 719).

In some cases of mitral stenosis vegetations are observed on the *tricuspid valve*, and these are evidently the results of endocarditis.   An induration of the structures at the right auriculo-ventricular aperture may take place, and lead to a series of morbid changes producing obstruction of the tricuspid aperture closely resembling that of the mitral. Tricuspid obstruction is rarely found except along with mitral obstruction, and the morbid changes producing it are often more recent in the right heart than in the left (*vide* p. 330).   When mitral and tricuspid changes coexist, the tendency to the formation of thrombi and emboli in the right cavities is more pronounced than when mitral obstruction exists alone.

In some cases the *venae cavae* have been found greatly dilated ; the inferior in greater degree than the superior.

Venous thrombosis, which is rare, or at least seldom reported in heart disease, is, when it occurs, almost exclusively met with in mitral disease, more especially in obstructive disease.   In 23 out of 27 cases collected by Welch it was in the veins of the upper extremities or neck.   (See also p. 735.)

The lungs generally present the appearances—congestion, consolidation, brown and pigmentary changes and fibrosis—characteristic of venous stasis.   In cases of obstruction haemorrhagic extravasations in the lungs, and blocking of the pulmonary artery, are observed to a greater extent and with greater frequency than in mitral regurgitation.   Not seldom there are signs of pulmonary infarction, old and recent.   From

an analysis of the post-mortem appearances in 36 cases of mitral stenosis, Sansom (76) found that infarctions, or haemorrhages, were observed in 22 instances. In rare cases a coagulum, evidently detached from the auricle, has plugged the pulmonary artery itself. Cases of mitral obstruction have been recorded by Friedreich in which an extremely dilated left auricle has compressed the left bronchus to such extent as to reduce its calibre to a mere chink. In no inconsiderable number of cases the lesions of tuberculosis have been found in the lungs. Only two cases of tuberculosis in association with mitral stenosis came under the notice of Sansom (76), but according to Potain (58) the coexistence is frequent. In 35 necropsies, in which mitral obstruction was demonstrated, tuberculous changes were found in 12 instances. Taking the cases recorded by Teissier, Dr. Kidd, and other observers, there is a total of 31 in which the association of mitral stenosis with tuberculosis was proved after death : of these cases 11 presented also the signs of tricuspid obstruction or of endocarditis affecting the tricuspid valve, and 5 others manifested disease of the aortic valves. Uncomplicated mitral obstruction, therefore, was present in 16 cases only. Potain (58) considered that the occurrence of mitral stenosis in the course of pulmonary tuberculosis is so frequent that there seems to be a causal relationship between the two diseases. Teissier has gone much farther than this ; he considers that some form of tuberculosis is the cause, direct or hereditary, of the " pure " form of mitral stenosis. Nevertheless, his own observations agree with those of Letulle that the search for bacilli and for any lesion demonstrably tuberculous in the diseased structures surrounding the mitral orifice has always been fruitless. To ascribe the origin of the fibrous thickening to an attenuated tuberculosis seems an extraordinary example of special pleading. A more tenable hypothesis is that in some cases the anaemia resulting from the delivery of an insufficient volume of blood from the imperfectly supplied ventricle, especially in the case of coexisting aortic disease, disposes to the tuberculous invasion ; and in others the failure of the right ventricle, or the obstruction to the supply to the pulmonary artery in the case of concurrence of tricuspid obstruction, disposes to tuberculosis of the lungs ; for it is to be remembered that in obstruction of the pulmonary artery, in which there is a like physical impediment to the blood-current to the lungs, pulmonary tuberculosis is very frequently the mode of death.

The stomach, liver, spleen, and other abdominal viscera in mitral stenosis shew, for the most part, the appearances to be described in mitral insufficiency. Embolism and its consequences are much more frequent in mitral obstruction. Taking post-mortem evidence alone, embolism is most frequently observed in the arteries of the brain and the kidneys, and these in equal proportions. Next in order of frequency are pluggings of the splenic arteries. In a small minority of cases the arteries of the pancreas, stomach, and intestines have been blocked by emboli.

In the cases in which emboli have obstructed the intracranial arteries the plugs are most commonly seen in the vessels of the left hemisphere. The resulting softening is found chiefly in the frontal and parietal convolutions and in the corpus striatum. According to the evidence obtainable, fatal cerebral embolism, the result of chronic mitral conditions, is most frequently left-sided. In cases in which acute endocarditis has supervened, the limitation to the arteries of the left hemisphere is not so decided. When there is necrosis of the tissues adjacent to the valve there are often multiple emboli. The clinical evidence in cases of mitral obstruction sometimes indicates a lesion of the right hemisphere, but the emboli which are fatal—probably slowly formed and comparatively large —are usually those which plug the arteries of the left hemisphere. There can be no doubt that the well-known physical explanation of their more common occurrence in the arteries of the left hemisphere is correct. The left carotid has its axial current in the same direction as that from the ascending aorta; the stream, therefore, carries the dislodged coagula most readily through the aorta into the left common carotid, the internal carotid, and the middle cerebral, the current continuing in these vessels without deviation. If the embolus be large, it is sufficient to block not only the trunk of the middle cerebral artery, but also that of the anterior cerebral at its bifurcation with the former. If small, the embolus may be in one only of the branches of the middle cerebral. The right hemisphere is less liable, because the right carotid, arising from the innominate, is placed at such an angle with the aorta as to lie off the axial current.

The Working of the Heart in Mitral Obstruction.—In the slighter forms of obstruction the mechanism is similar to that obtaining in the sclerotic form of mitral insufficiency. The orifice may be so narrowed as to admit only two fingers or even the thumb only; but the thickened curtains of the valve are retracted, and the physical signs, symptoms, and consequences are those of mitral regurgitation.

The conditions are characteristically different when the mitral orifice is so narrowed or obstructed that the outflow from auricle to ventricle is seriously impeded; and when, as may be inferred with great probability, there is no regurgitation at the time of the systole of the left ventricle. The most pronounced effect in such cases is upon the left auricle. The muscular wall may be greatly hypertrophied, while the diameter of the chamber remains not notably greater than the normal. Or, again, the auricle may be greatly enlarged, so that in some cases its capacity is more than double the normal; its muscle in some cases is hypertrophied, in others atrophied, even so far as to be represented only by a few muscular fibrillae scattered through a shell of fibrous tissue. Observers have differed as to the relative preponderance of hypertrophy and dilatation in the auricle. Potain (59) considers that, suffering as it immediately does a "contrecoup" on account of the obstructive lesion, the left auricle dilates and hypertrophies simultaneously, and that these changes are never wanting in mitral stenosis. It is obvious

that the muscular auricle is strong enough to inject its blood-content forcibly into the ventricle even though the mitral orifice be considerably diminished. The muscular wall of the auricle may be as thick as that of the right ventricle. Cases have been recorded by Gérard, in which the left auricle has maintained life for a long time when the left ventricle, converted into a completely calcified chamber, had been incapable of any active contraction.

It has been generally considered that the auricle ceases its active contraction before the systole of the ventricle begins. This was the doctrine deduced from the graphic records obtained by the experimental methods of Chauveau and Marey in the horse. Subsequent investigation, however, has demonstrated that the auricular systole may continue after the commencement of the contraction of the ventricular muscle, both auricle and ventricle continuing to contract simultaneously until the moment when the sigmoid valves are opened and blood begins to be expelled from the ventricle into the aorta. Dr. Keith (44) has recently given excellent reasons for believing that the ventricle begins its systole before that of the auricle ends. Potain considers that the auricle is in action from the beginning of its systole until the precise moment of closure of the auriculo-ventricular valves—that it is this muscular contraction of the auricle which ordinarily causes the propulsion of the heart's apex against the wall of the chest, and that thus it plays a notable part in the production of the impulse which is felt by the hand applied over the situation of the apex-beat. In obstruction of the mitral aperture this lifting of the apex by the force of the contracting auricle may be greatly exaggerated, as will be mentioned on page 356.

Dr. Samways (66) has urged that the abnormally powerful contraction of the left auricle prevents regurgitation in compensated mitral stenosis. He shews from mechanical and experimental data that the force of the auricle, seeing that its active contraction is continued until the aortic valves are opened and a free outflow is permitted into the aorta, is adequate to prevent any reflux during the ventricular systole. It seems very probable that this view is correct. It affords a good explanation of the post-mortem appearances when a contracted mitral orifice, evidently of slow development, is accompanied by a very small left ventricle. If mitral regurgitation had occurred in such a case the ventricular cavity would in all probability have become dilated. Yet in the early stages of the transformation of the mitral orifice it would seem that such regurgitation would have been inevitable unless prevented by some cause apart from the sclerosis of the valve. A compensatory hypertrophy of the muscular wall of the auricle—whence an abnormally prolonged and powerful auricular systole—occurring early in the morbid process would explain not only the absence of the characteristic signs of mitral inadequacy during life, but the absence of hypertrophy and dilatation of the left ventricle observed after death.

It is obvious that the increased force of the auricle, shewn by the muscular hypertrophy, is an important, if not the chief, factor in

maintaining compensation during the survival—many months or many years it may be—of the subjects of mitral obstruction. It is equally certain that it is not the only factor, for hypertrophy of the right ventricle may be looked upon as a constant sequel of mitral obstruction. Dilatation in most cases accompanies the hypertrophy, but for long periods the tricuspid valve is competent to close the right auriculo-ventricular orifice. Abnormal pressure is thus maintained in the pulmonary blood-circuit. The hypertrophied right ventricle co-operates with the hypertrophied left auricle in augmenting the force by which the blood is urged through the narrowed mitral orifice. In the later stages of the affection, however, the right ventricle may become dilated on account of the exaggerated blood-pressure, as well as the loss of tonicity, to such degree that the tricuspid valve is no longer competent, and there is reflux into the great veins. Compensation is then no longer maintained. The failure of compensation, however, in a given case may be not by failure of the right ventricle, but on account of enfeeblement of the left auricle. We have seen that the auricular cavity may be enormous, but with practically no effective muscle in the wall. The evidence, especially the deposition of layer upon layer of fibrin, shews that failure has been slow and life has been prolonged without any active participation of the auricle in the work of the circulation.

In a case of compensated mitral stenosis we may thus summarise the work of the heart—*Systole of the ventricles.* Left unimpeded, discharging less than or just enough for the needs of the organism ; right abnormally forcible, thus distending the pulmonary veins and the left auricle. *Left auricle* over-distended after right ventricular systole ; this distension is in greater or less degree relieved immediately on diastolic relaxation and suction action of left ventricle, its own elastic recoil probably aiding the inflow into the ventricle in the earliest stages of diastole. Probably muscular contraction of the pulmonary veins is a concurring cause ; possibly such contraction in the manner of a sphincter preventing reflux from the auricle into the pulmonary veins ; the proper auricular systole following and, being abnormally forcible and protracted, contributing to produce the apex-impulse.

Diagnosis.—The diagnosis is in many cases easy, in some attended by considerable difficulty ; at any rate all the ordinary means of physical investigation should be put in force.

Inspection may reveal no signs. The apex-beat may be invisible or observed in the normal situation—if displaced to the left, causes external to the heart being excluded, the explanation may be enlargement of the right cavities or a general increase of bulk of the heart due, in the early periods of life, sometimes to adherent pericardium ; in the later periods to the hypertrophy and dilatation of the left ventricle accompanying arteriosclerosis. In some instances the precordial region over the right ventricle is rendered prominent and visible ; pulsation is seen below the ensiform cartilage. In any case when there is this prominence over the right ventricle, whilst the left ventricle is not observed to pulsate to the left of the normal position, mitral obstruction is prima facie more probable

than mitral insufficiency. A systolic impulse may be seen in the second left intercostal space in cases in which the right cavities are dilated and the conus arteriosus extends too far out.

Examination of the veins of the neck is important, as it reveals to some extent whether the right side of the heart is implicated or not. On inspection there may be the physiological venous pulsation, synchronous with the auricular systole ; both auricular and ventricular movements may in other instances be visible, shewing that the right side of the heart has undergone such changes as to render the tricuspid valve incompetent ; a still further development of the morbid process may be seen in a venous pulsation which occurs solely with the ventricular systole, shewing that the auricle has become incompetent. These various appearances may be more thoroughly investigated by means of the graphic method, which allows clear differentiation of the different waves (*vide* Figs. 34, 35, pp. 355, 356). Dr. J. Mackenzie has, within recent years, given a very full exposition of the whole subject.

Palpation may reveal some very important evidence or may be negative. In a case of marked mitral stenosis of long standing a heaving impulse may be found over the position of the right ventricle, under the false ribs to the left of the ensiform cartilage, whilst there may be no palpation-signs of a forcible ventricular systole abnormally to the left. Palpation may thus confirm inspection in indicating that the right side of the heart is enlarged and the right ventricle hypertrophied, whilst the left ventricle does not shew these abnormalities. Any such deduction, however, must be made cautiously, for the left heart may be more enlarged than the signs indicate, as it may be covered by lung-tissue. There is one sign obtained by palpation to be observed in a considerable number of cases of mitral stenosis which, provided it has certain essential characters, may be regarded as almost a crucial sign of the affection. This is the *thrill* —" frémissement cataire "—originally observed by Corvisart. The feeling of vibration communicated to the hand lightly laid upon the apex-beat or slightly to the right of it may be fine or coarse, protracted throughout the whole diastole and period of repose and ceasing with the apex-beat —it may be immediately after the commencement of this event—or occupying a very brief period just before the systole of the ventricle. It can best be timed by the finger of the other hand placed over the carotid artery, when the thrill is found to cease at the moment of the carotid pulse. If in the case investigated there are well-marked signs of incompetence of the aortic valves, it is to be borne in mind that the diastolic-presystolic thrill may be present without mitral stenosis. With regard to the incidence of Flint's murmur or a mitral presystolic in aortic insufficiency uncomplicated by mitral stenosis, Thayer has shewn that it is commoner than is usually believed ; in 58 cases of aortic insufficiency without mitral stenosis, as shewn by necropsy, Flint's murmur was heard in 33 or 56·8 per cent. In the absence of signs of aortic valve disease a well-marked diastolic or presystolic thrill when observed in the apex region is always indicative of mitral stenosis. It is important that the thrill

be investigated in varying positions of the patient. Vibrations which are scarcely felt when the patient is in the recumbent position may become much more marked in the sitting posture with the body bent forwards. The observation, however, does not excuse the omission of all other ordinary means of investigation. It is to be remembered that thrill may be absent at some periods and present at others during the observation of a case. Sometimes it is absent when the patient is at rest, and developed after exertion or when the arms are elevated. When tricuspid obstruction is present along with mitral obstruction there may be thrills of different characters to the right and left respectively of the sternum. Such a case was recorded by Dr. Halliday Croom from Dr. Gibson's wards in the Royal Infirmary of Edinburgh.

Percussion is chiefly of importance to determine the outline of the heart : it gives more precision to the evidence obtained by inspection and palpation, and when disproportionate enlargement of the right chambers of the heart is thus indicated, this method of investigation is valuable for diagnosis not only of the nature of the affection, but of its extent and significance. In many cases of pure uncomplicated mitral obstruction there is no change in the size of the heart, but in most instances, while the left border is unaltered, the right extends beyond the normal limits. Sometimes the dulness extends above the upper border of the left third costal cartilage ; when this is found it may be assumed that with the dilatation of the right ventricle the conus arteriosus has extended upwards. In such cases there is usually a systolic impulse in the left second intercostal space.

The signs obtained by *auscultation* are of chief importance in the diagnosis of mitral obstruction—they are murmurs, double-shock sound during the period of ventricular diastole (reduplication of the second sound), accentuation of the pulmonic second sound, loud and sudden snap at the acme of ventricular systole, and inaudibility of the second sound at the heart's apex. The murmur characteristic of mitral obstruction is that known as the presystolic murmur. It is generally of rough quality, vibratory or bubbling. It may begin almost immediately after the second sound of the heart, be prolonged in "diminuendo" fashion through the whole period of ventricular diastole, become reinforced towards the end of this period in a "crescendo" manner, and end with a sudden tap or snap. This terminal snapping sound is in some cases coincident with the impulse of the apex as felt by the finger ; in others it is noted to occur very shortly after the first shock of the impulse ; but it is always synchronous with the pulse felt in the carotid artery. The sound of the murmur may begin long before the proper systole of the auricles—it may therefore be correctly designated diastolic-presystolic. The evidence leaves no room for doubt that the reinforcement towards the close is coincident with and due to the muscular contraction of the auricle. Gairdner (29) used the term "auricular systolic" (A. S. murmur) to denote the murmur. Whether the term presystolic or auricular systolic be used, it must be remembered that the active muscular contraction of

the auricle is not the only force on which the murmur depends. In some cases the bruit is not prolonged throughout the periods of diastole and presystole, but is audible as a short murmur closely following the second sound (early diastolic murmur), or isolated with a pause before or after (mid-diastolic or meso-diastolic murmur). Usually these disjointed murmurs are found in a case which at some periods òf observation manifests the more typical presystolic murmur.

The causation of these murmurs has been the source of much discussion. The lucid descriptions of Gairdner (28) made the mode of production perfectly clear, but Ormerod, a few years later, attacked the views enunciated by Fauvel and elaborated by Gairdner, chiefly on the ground that the auricle does not possess sufficient energy to produce so loud a murmur. Barclay and F. C. Turner, at a later period, espoused the opinions of Ormerod, and more recently Dr. Dickinson and Dr. M'Vail followed in their footsteps. Just about the date of the publication of the first edition of this work, Dr. Brockbank, in a very ingenious paper, expressed the same opinion, differing, however, from the others already mentioned in certain details. All these observers have held that the murmur is produced by the systole of the ventricle, which, according to them, begins at an earlier period than usual and is continued until the closure of the valves. That this opinion is shared by certain continental observers is proved by the use of the term protosystolic for this murmur in a recent treatise on medicine by Tripier and Devic. The explanation of Fauvel and Gairdner cannot for a moment be questioned; two considerations are in themselves sufficient to negative the hypothesis of Ormerod and his followers. The fact that the murmur begins in many instances with the second and continues without intermission until the first sound is in itself fatal to Ormerod's explanation. The interesting case of obstruction of the tricuspid orifice described by Gairdner (30) is moreover a piece of incontrovertible evidence in favour of his views. In this case a tumour projected from the wall of the right auricle and could not in any way interfere with the closure of the tricuspid valve, but during the auricular systole it descended so as to interfere with the passage of the blood from the auricle into the ventricle and caused a loud presystolic murmur. There can be no doubt that the auricle possesses sufficient energy to produce the presystolic murmur. Many years ago Dr. Malet and Dr. Gibson shewed that it was capable of causing distinct sounds even in physiological conditions; when hypertrophied, as in most instances of mitral obstruction, its possibilities must be greater. When the venous movements in the neck and the murmurs arising at the mitral orifice are studied together, it is found that the presence of the auricular type of venous pulsation is frequently associated with a distinct presystolic murmur, whereas the ventricular form is not as a rule attended by this murmur. In the latter case the auricle has become paralysed. The fluorescent screen furnishes additional evidence in favour of this contention. Dr. Gibson has discussed the whole subject fully elsewhere (33).

The production of the early diastolic murmur is easy to understand,

and all authors are in practical accord on the subject. It is caused by the aspiratory force of the ventricle, while in active diastole, drawing a current of blood from the auricle into the ventricle. The mid-diastolic murmur is probably the result in some instances of irregular auricular systole, in other words, of auricular systole which is not followed by contraction at once of the ventricle, so that heart-block is present. Examination of patients with the fluorescent screen bears out this conception, at least in some cases. But it must be admitted that this cannot be held to be the cause of the mid-diastolic murmur in all instances, and it is probable, as has been urged by Dr. Rolleston, that the aspiratory action of the ventricle is the cause in many such cases.

The sudden snap which generally terminates the presystolic murmur is peculiar and characteristic. In some cases it is observed without any bruit leading up to it. It is evidently an unusually short and sudden first sound of the heart; if in any case it be observed in the near neighbourhood of the apex—in some cases it may be noted at the back under the angle of the left scapula—mitral obstruction should be suspected and the concurrent signs searched for. The cause of this phenomenon is not definitely settled. It closely resembles the sound of sudden tension which may be imitated by abruptly stretching a piece of moist membrane. In the left ventricle of some hearts with a narrowed mitral aperture observed after death, in which the phenomenon had been manifested during life, it would seem that there are no structures likely to give rise to this sudden sound at the moment of contraction of the ventricle—the mitral curtains being thick and leathery, the chordae shortened, and, with the papillary muscles, forming thick fibroid bands; the muscle of the ventricle not obviously differing from the normal, and the ventricular cavity small rather than large. On the other hand, the tricuspid valve is seen to be thin and membranous, and it seems probable that to its sudden tension by a forcible right ventricle the loud snap may be ascribed.

Another very important auscultatory sign is the double sound heard during the period of the diastole of the ventricles. This phenomenon, which vividly recalls the "postman's knock," has been generally named the *reduplicated second sound*. To avoid speculation as to its mode of production we may be permitted to call it a double-shock sound in diastole. It may be manifested in the neighbourhood of the apex or at the base of the heart. When audible in the neighbourhood of the apex of the heart and not over the base the double-shock sound indicates an early stage of mitral stenosis. This view enunciated by Sansom was confirmed by Dr. Cheadle. As a sign of mitral stenosis in later as well as earlier stages, it has been noted by many observers. The explanation of the mechanism of this sound suggested by Sansom has been for the most part accepted. It is not a true doubling of the second sound, and cannot be ascribed to the asynchronous closure of the semilunar valves of the aorta and the pulmonary artery, but is of mitral origin. It is due to the first inrush of blood into the ventricle, such inrush being more sudden and forcible than under normal conditions

from the increased blood-pressure in the left auricle due to the constriction of the mitral orifice. Potain (58), Rouchès, and other French observers have described this sound as the " claquement d'ouverture de la mitrale." Potain thus explains the mechanism of the sound. The opening of the mitral valve is normally noiseless ; but in the subject of mitral obstruction the valve curtains at the moment when they separate, moved by the blood-wave that enters the ventricle, are abruptly checked by the adhesions of their free borders ; the sudden tension which results produces the sound, which is the more dull as the normally thin curtains have become more dense and have lost their elasticity.

When the double-shock sound is audible over the base of the heart and not in the close neighbourhood of the apex the problem of its cause admits of a ready answer. It is undoubtedly over the base of the heart that the double sound, when manifested in mitral stenosis, is heard in the majority of cases. The diagnostic value of the sound is very great ; the double sound either at base or apex is found in more than one-third of all cases of mitral stenosis. The generally accepted view of its mode of production is that the semilunar valves of the aorta and the pulmonary artery respectively do not close in normal synchronism, but those of one vessel coapt in advance of those in the other according to the relative degrees of blood-pressure. The sound of tension of the aortic valves cannot be produced until the systole of the left ventricle ceases ; if this sound be followed by that of the tension of the sigmoid valves of the pulmonary artery, it follows that the end of the systole of the right ventricle is not synchronous with that of the left, but is delayed. Potain (57) has advanced the following hypothesis : Premising that the precession of the two sounds of tension is aortic in the earlier, and pulmonic in the later phases of the disease, he considers it probable that when the obstruction at the mitral orifice is slight, but yet sufficient to bring about some difficulty in the entry of blood into the left ventricle, the aspirating power of the latter in diastole is augmented ("elle est moins aisément satisfaite "), and the semilunar valves, drawn upon with more force than ordinarily, close more rapidly. Later, when the obstacle at the left auriculo-ventricular orifice has notably impeded the circulation in the lung, and the right ventricle has become hypertrophied, the over-pressure in the pulmonary artery compels the semilunar valves of this vessel to close more forcibly and more rapidly at the beginning of ventricular diastole. Some tracings distinctly prove the asynchronous termination of the systole of the two ventricles (34).

Accentuation of the pulmonic second sound is a sign to be noted in mitral obstruction. The cause is over-pressure in the pulmonary artery. The irregular rhythm of the heart in many of the cases, however, prevents a due appreciation of this accentuation ; the sound then is very loud in some cardiac cycles, in others feeble or almost inaudible.

Another auscultatory sign to be noted in a section of the cases is *inaudibility of the second sound of the heart at the apex*. This extinction of the second sound at the apex is usually manifested in the later stages

of mitral stenosis (Broadbent (11), Acland) ; its causes are—(a) a diminu-
tion of blood-supply to the aorta, and consequent feeble recoil against
closed aortic valves (it is the aortic element of the second sound that
is chiefly audible over the heart's apex) ; (b) the enlargement of the
right auricle and ventricle, which, coming more and more to the front,
displace the left ventricle, the chief conductor of the sound.

Another modification of the second sound occurs as the result of
relative or functional incompetence of the pulmonary cusps in consequence
of high pressure in the pulmonary artery. Stokes realised this as a
possibility, and his views were shared by Gouraud, but it has only been
within recent years that the matter has been adequately recognised
(6, 13, 20, 35, 78). The second sound belonging to the pulmonary
artery, which has previously been accentuated or doubled, is found to be
followed or its place taken by a soft blowing murmur, usually short in
duration, with its maximum intensity in the left third intercostal space,
about an inch from the sternal border. It is sometimes conducted
to the fourth intercostal space, but is rarely heard further from
the pulmonary area. There can be no doubt of the relative
frequency of this functional regurgitation, but it is probable that
the murmur caused by the escape at the pulmonary orifice is often
confounded with, or mistaken for, the rougher diastolic murmur
produced at the mitral orifice by obstruction. The pulmonary diastolic
murmur varies much from time to time in its intensity, and frequently
disappears with improvement in the condition of the circulation, only to
reappear once more when the pulmonary pressure becomes elevated.
Speaking generally, it disappears with the advent of tricuspid incom-
petence, which at once leads to lowering of the pressure in the right
ventricle and pulmonary artery. The mode of production of this
functional pulmonary incompetence was first suggested by Hunter, whose
researches were amplified by Adams, and in recent years by others
(Gibson (35), Keith (45), and Mackenzie (52)). The real origin of the
escape has been found to be a loss of tone in the conus arteriosus.

In the latest stage of mitral stenosis the presystolic murmur may
be inaudible, the second sound absent, and the short and sudden first
sound, to which attention has been already called, the only notable
auscultatory sign. More frequently, however, in later as well as in earlier
stages, a systolic murmur is to be heard in the neighbourhood of the
apex. This murmur may have the ordinary characters of that of mitral
insufficiency, audible over the apex and at the back under the angle of
the left scapula, or may be a short systolic " puff " having a very limited
area of audibility, but over the site of the apex. It may coexist with
the presystolic murmur, which in such cases is usually heard for the
most part slightly to the right of it ; or it may be heard when no
presystolic or diastolic-presystolic bruit is audible. Nearly always in
these cases the sudden tap indicating the first sound is heard over some
part of the apex region. In another series of cases the systolic murmur
has an area of audibility to the right of the apex, encroaching more

and more on the tricuspid region, and in some instances localised at the base of the ensiform cartilage, that is, the area of a tricuspid regurgitant murmur. It has been urged by Dr. Samways (66) (and the contention has a great show of validity) that in some of the cases in which a systolic murmur has been ascribed to regurgitation through the mitral orifice the real cause of the phenomenon has been tricuspid reflux. In some instances, however, there are two areas of audibility of the systolic murmurs, when it is most probable that there is regurgitation through both mitral and tricuspid orifices. If the hypothesis be correct, that the
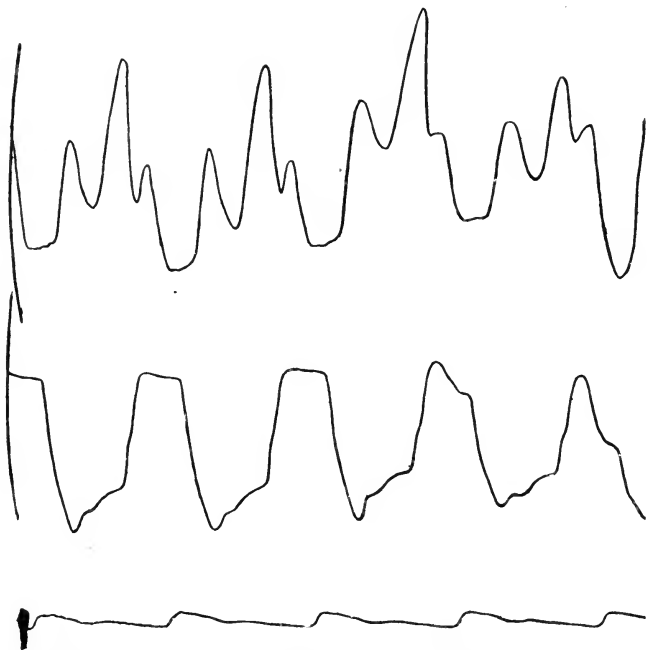
Fig. 34.—Tracings from the cervical veins, the apex-beat, and the brachial pulse in a case of pure mitral obstruction. The venous tracing shews the characteristic physiological auricular type of pulsation, and the apex-tracing gives a distinct auricular pulsation before the ventricular systole.

abnormally powerful muscular contraction of the left auricle prevents regurgitation in the compensated stages of mitral stenosis, it is probable that some such regurgitation is inevitable when compensation fails and the auricular muscle has become feeble.

It is to be noted that a very marked irregularity of the heart's rhythm is by no means infrequent in mitral stenosis, and that this irregularity may modify all the physical signs already described. The murmurs, the doubling of normal sounds, the snap sound, and the thrill may be observed in some cardiac cycles, and may be absent in others. The irregularity is more fully discussed in the remarks upon the pulse in the following paragraphs.

Cardiographic Evidence.—The use of the cardiograph has afforded evidence of some importance in the elucidation of certain problems connected with mitral obstruction. The results of such investigations require to be analysed with great caution, as must be obvious when so many considerations present themselves for discussion. The ventricular impulse may undergo considerable variations from the effect of external influences ; the apex, for example, may be hidden by an emphysematous condition of the lung ; it may, on the other hand, be unduly prominent
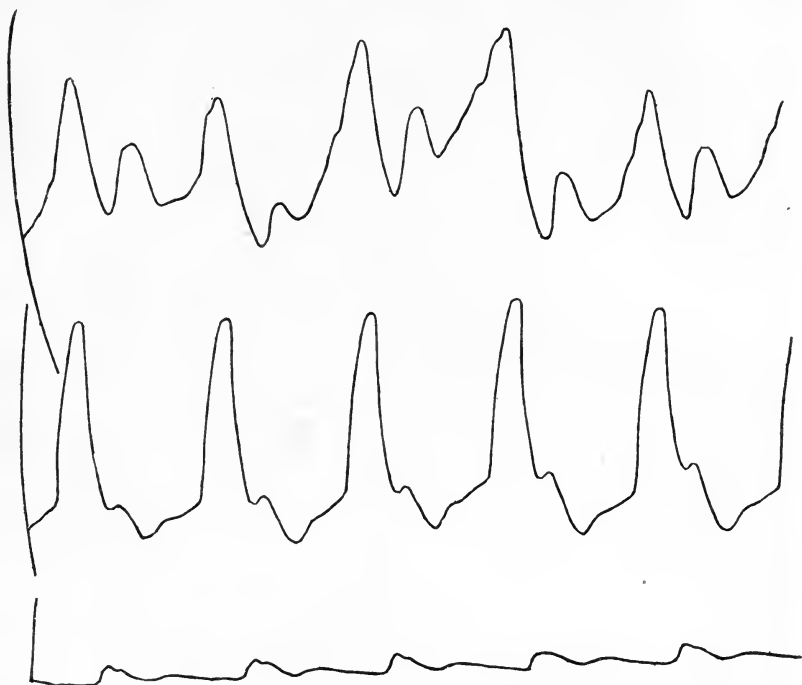
FIG. 35.—Tracings from the cervical veins, apex-beat, and brachial pulse in a case of mitral disease. The venous tracing shews scarcely a trace of the auricular impulse, which is entirely absent in the apical curve.

on account of pulmonary retraction. From causes arising within the heart itself, moreover, considerable changes may occur in the ventricular pulsation ; it is very common to find, for instance, that the apparent apex - beat is produced by the right ventricle, which by means of its dilatation occupies a position in front of the apex of the left ventricle. In such cases the cardiogram is often inverted, shewing depression instead of elevation during the systole.

The apex-beat in mitral obstruction often does not shew any departure from normal appearances, and exhibits a slight auricular elevation, followed by a well-marked ventricular rise, which is succeeded by the impulse produced by the arterial recoil accompanying the second sound.

In many cases, however, the auricular systole is found to be early and prolonged, whilst in others it disappears entirely. In the former there is usually a well-marked presystolic murmur, and in the latter the murmur disappears. The differences between cardiograms produced by the two ventricles respectively are not difficult to distinguish, as in the case of the right ventricle the systolic period is characterised by retraction in-

Fig. 36.—Tracings taken from the fifth intercostal space and the sixth intercostal space, as well as from the brachial artery, in a patient with mitral obstruction. It will be observed that the tracing from the fifth intercostal space is inverted, whilst that from the sixth intercostal space, the real apex-beat, shews a ventricular rise immediately before the pulsation of the brachial artery. The impulse of the auricle is imperfectly present.

stead of elevation. The tracing (Fig. 36) shews a tracing from the apex-beat in the sixth intercostal space, from the right ventricle in the fifth, and from the brachial artery, in order to determine the time accurately. In addition to these various points a cardiogram from the apex shews many different types of arrhythmia which need not now be discussed.

The pulsation of the conus arteriosus in the second and third intercostal spaces is occasionally so definite as to furnish the opportunity of obtaining cardiograph tracings. One of these is reproduced here to shew the type of pulsation (Fig. 37), and another is given along with the radial

pulsation (Fig. 38). In both the diastolic murmur of escape at the
pulmonary orifice, in consequence of high pressure, was present.

FIG. 37.—Tracing from the conus arteriosus in a case of mitral obstruction which exhibited a diastolic
pulmonary murmur of high pressure.

FIG. 38.—Tracings from the conus arteriosus above and the radial pulse below in a case of mitral
obstruction with a diastolic pulmonary murmur of high pressure.

Sphygmographic Evidence.—In a large number of cases of mitral
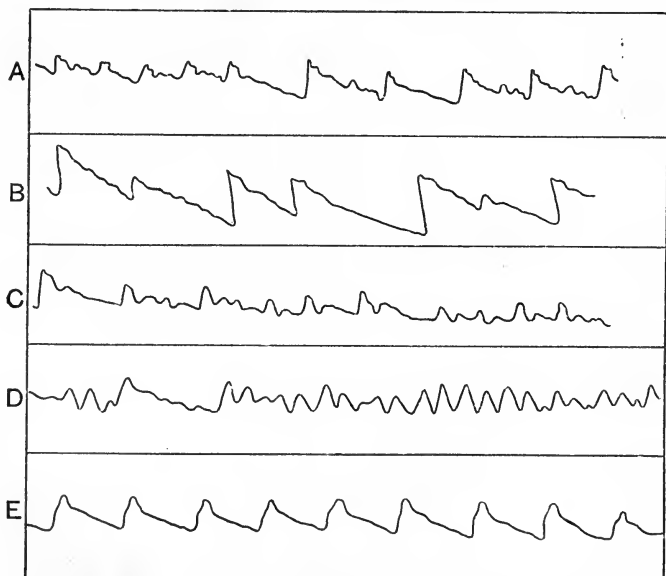
FIG. 39.—Sphygmograms in mitral obstruction. A, In stage of compensation ; man aged forty-four,
observed during five years. B, Case manifesting typical presystolic murmur ; no signs of failing
compensation ; patient in good health. C, Mitral stenosis with failure of compensation (tricuspid
regurgitation, pulsating liver). D, Late stage of extreme mitral obstruction (female aged seven-
teen). E, Regular anacrotic pulse ; mitral obstruction in a female, aged forty-one, with rheumatic
antecedents.

stenosis the sphygmograph indicates very notable irregularity as well as inequality ; these features may be observed when the lesion is compensated and the patient appears to be in perfect health. In some instances in which the rhythm of the heart is apparently regular, a slight exertion serves to provoke the irregularity. The administration of digitalis may produce or increase it ; but it is often found in cases in which the drug has not been administered. The most frequently observed form of irregularity is produced by the occurrence of extra-systoles. In late stages of the disease the irregularity may be extreme. In many instances the irregularity is undoubtedly caused by a lesion in the auriculo - ventricular bundle, whereby the conductivity is impaired, and certain of the impulses from the auricle fail to pass down to the ventricle. But in other cases, the cause seems to be in extra-systoles taking their origin in the ventricle—the nodal rhythm of Dr. Mackenzie (52a). The irregular pulse of mitral obstruction has been discussed by many observers, such as Balfour (3).

In another large series of cases the sphygmograms shew a perfect regularity in the heart's rhythm. Many observers have considered such regularity to be the rule in mitral stenosis. The up-stroke of the tracing is inconsiderable, and it has been held that the vessel is full between the beats. Broadbent (10) considered that this modified high-pressure pulse is almost constant in mitral obstruction, and indicates resistance in the capillaries. Such resistance may be due to contraction of the arterioles consequent upon the overloading of the blood with impurities arising from defective elimination or, pos-

Fig. 40.—Tracing of the arterial pressure obtained with Gibson's sphygmomanometer from the arm of a healthy man.

sibly, from the backward pressure in the veins effected through the capillary network, or from the contraction of the entire arterial

system upon a diminished supply of blood from the imperfectly filled left ventricle.

Sphygmomanometry.—The arterial pressure shews wide limits of variation. It is dependent on so many factors, such as the energy of the heart, the elasticity of the arteries, and the resistance in the arterioles, that it is impossible to give any general rule. It is often
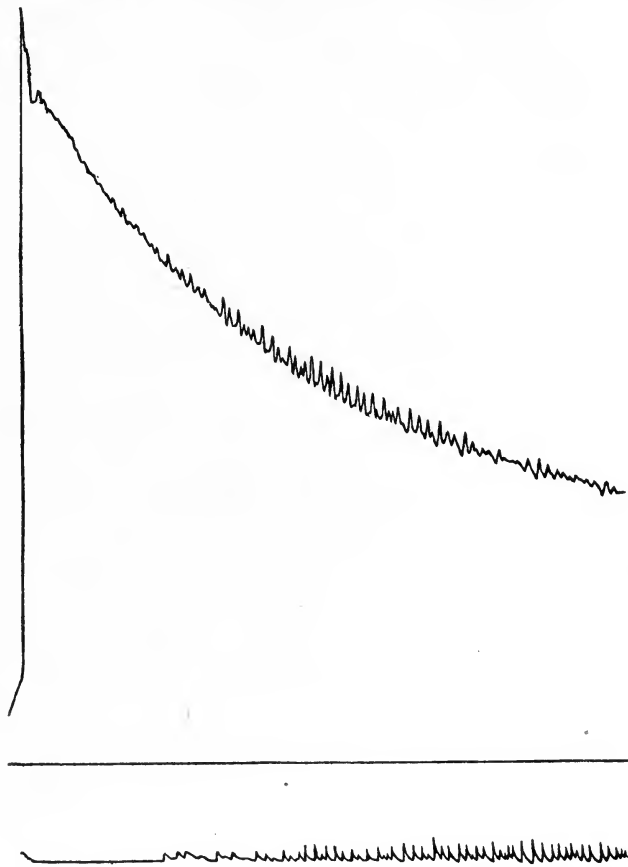
FIG. 41.—Tracing obtained from the arm of a patient suffering from pure mitral obstruction, before the establishment of compensation, after cardiac failure.

below the normal, but even more frequently above it. Deficient aeration of the blood and faulty elimination of waste products invariably bring about an elevation, whilst failing cardiac energy, and relaxation of the arterioles, may bring about a reduction. A careful comparison of the arterial pressure, as measured by modern instruments, with the amount of cardiac energy, as estimated by ordinary clinical methods, is necessary in every case. It is very common indeed to find that the

arterial pressure is too high for the power possessed by the heart. Three tracings obtained with Gibson's sphygmomanometer (36) are shewn in Figs. 40, 41, and 42. Figure 40 gives the curves from a healthy man, exhibiting a systolic pressure of 140 and a diastolic pressure of 114 mm.
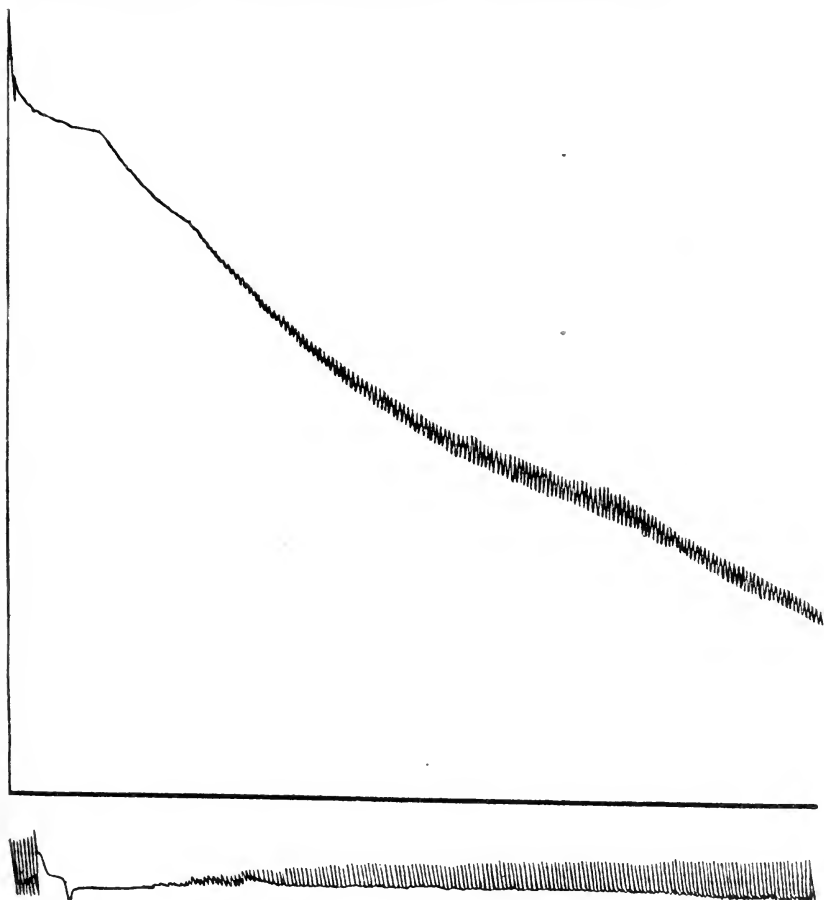
Fig. 42.—Tracing obtained from the arm of a patient with mitral obstruction, in which there had been no disturbance of compensation.

Hg. Fig. 41 shews great irregularity of pulsation, and inequality of pulse-wave, both in the excursions of the column of mercury and in the oscillations given by the transmission sphygmograph. The systolic and diastolic pressures in this case, one of uncompensated mitral obstruction, were 125 and 96 mm. Hg. Fig. 42, from a case of compensated mitral obstruction, demonstrates, both in the kymographic and sphygmographic curves, wonderful regularity and equality of oscillation. The systolic

and diastolic pressures are respectively 160 and 80 mm. Hg. In this case there was evidence of some chronic interstitial change in the kidneys, the association of which with mitral obstruction is discussed on p. 341.

Some Difficulties in the Diagnosis.—Although the presystolic murmur and the thrill observed in the positions mentioned, close to the heart's apex, are indications of mitral obstruction, in certain cases they are not absolutely pathognomonic.

Austin Flint was the first observer to shew that a murmur having the characters of that of mitral obstruction could be produced in cases of insufficiency of the aortic valves in the absence of mitral stenosis. These observations have been confirmed by many observers. The presystolic thrill of mitral stenosis also can be exactly simulated under conditions of aortic regurgitation. Dr. Phear has carefully analysed the records of 46 cases in which there was a presystolic apex-murmur without mitral stenosis ; in 12 of these, thrill, presystolic or diastolic, was present. In 17 of the cases the aortic valves were incompetent ; in 20 the pericardium was adherent ; in the remainder there was no valve-lesion, but in some of these there was dilatation of the left ventricle. Thayer has shewn that Flint's murmur is comparatively common in aortic incompetence, and has indicated the diagnosis from the presystolic murmur of true mitral obstruction ; he finds that Flint's murmur is never so loud or rasping as the more marked murmurs of true mitral obstruction ; the presystolic thrill is a less frequent concomitant, but the most important distinction is the character of the systolic shock, which is tapping in true mitral obstruction, forcible and rather heaving in aortic insufficiency with a Flint's murmur. The snapping valvular first sound is also rare in association with that murmur. The hypotheses which have been adduced to explain these phenomena are the following : (i.) That in the cases of aortic regurgitation the regurgitant stream tends to lift the great anterior mitral curtain, and so to obstruct the mitral orifice at the end of diastole as to impede the current from the auricle ; (ii.) That the mitral valve is thrown into vibration by the two currents, the regurgitant from the aorta and the direct from the auricle, such vibrations lasting until the commencement of ventricular systole ; (iii.) That in the absence of aortic valve disease, but in the presence of adherent pericardium, vibrations may be set up by the current propelled from a dilated and hypertrophied auricle into a ventricle whose muscular walls are deficient in tone ; (iv.) That shortening of the chordae tendineae, or dilatation of the left ventricle, may bring about a virtual narrowing of the aperture through which the blood passes from auricle to ventricle, the auricular muscle continuing to be sufficiently powerful to generate a fluid vein. It must be admitted that these opinions are for the most part conjectural, but the fact remains that in some cases the physical signs have led most competent and careful observers to an erroneous diagnosis of mitral stenosis. The practical lessons are the following :—In cases in which the concurrent signs indicate dilatation of the left ventricle, and in which the previous history tells of an antecedent pericarditis, we must be

cautious in interpreting a presystolic murmur as pathognomonic of a stenosed mitral orifice. In all cases careful investigation must be made as to concurring signs of incompetency of the aortic valves. If the murmur of aortic regurgitation be absent from the base of the heart and the line of the sternum, it may yet be found alone at the apex, and may then closely simulate the murmur of mitral stenosis. In such a case, however, according to usual experience, the terminal tension sound, the tap or snap, is not marked—the sound is dull. All available means, including the use of the cardiograph and sphygmograph, should be used to effect the differentiation (*vide* also p. 471).

It must be remembered that aortic insufficiency and mitral obstruction may coexist, and the diagnosis of the combined lesion may present great difficulty. On examination of the post-mortem records of the London Hospital evidence of the combined lesions was found in 39 instances. Uncomplicated aortic insufficiency was to aortic insufficiency plus mitral obstruction as 88 to 39. The association of the two valvular affections, therefore, is not very rare, and the diagnosis of such association can only be made with an approach to certainty when there are decided physical indications of each separate morbid condition.

When the left auricle is greatly enlarged, it may (as was mentioned in dealing with the pathological features of the disease on page 343) exert pressure upon the left bronchus; when it does, it brings about the development of a definite series of clinical results. In an early stage, the most important symptom is stridulous respiration, confined to the left side of the chest, and sometimes accompanied by fine crepitations at the base of the left lung. After a time there is dulness on percussion, without much change in the vocal fremitus on palpation, the probable explanation of which is that, although the condensation of the lung which is going on ought to exaggerate the fremitus, the diminished lumen of the bronchus interferes with the access of the sound-vibrations. The respiratory murmur in this latter condition becomes harsh and bronchial, but as the stridor continues to accompany it, the character is sometimes masked. The latest stage shews absolute dulness on percussion, with total absence of the vocal fremitus and resonance, while the respiratory murmur becomes entirely obliterated. A considerable degree of retraction may be observed on inspection in such cases. It must be added that such a pronounced condition as that last mentioned is very rarely present.

Sometimes the dilated left auricle gives rise to pressure on the recurrent laryngeal nerve. The earliest symptom produced in this way is harshness of the voice, and a characteristic type of cough consisting in the loss of its explosive quality. These pass into complete aphonia, but it is very common to find that the symptoms wax and wane from time to time with the varying extent to which the auricle is dilated, and the consequent degree of pressure which it exerts upon the nerve.

In those cases in which pressure on the bronchus or on the recurrent laryngeal nerve is manifested, the diagnosis may sometimes be difficult,

seeing that the effects are mostly observed in the later stages of the disease when many of the characteristic manifestations may have disappeared, and the symptoms may therefore closely resemble those produced by aneurysm or tumour (*vide* p. 669). Some of the difficulties thus arising have been dealt with in a paper by Dr. Gibson (36), and three pronounced instances have recently been placed on record by Prof. Osler (55a).

CLINICAL GROUPS OF CASES OF MITRAL STENOSIS, THEIR SYMPTOMS AND TREATMENT.—**Group I. Cases Associated with Rheumatism.**—The intimate relation between mitral obstruction and rheumatism is shewn by every series of statistics. In some cases the rise and progress of the endocarditis (the cause of the obstructive lesion) can be traced by clinical observation. The patient may shew all the signs of acute rheumatism, an occurrence comparatively rare in children, the acute symptoms being often very slightly pronounced, though in some instances they are fully manifested, and then usually the first sign of implication of the valves is the systolic murmur of mitral regurgitation. The child in the course of months or years may suffer from repeated attacks of acute rheumatism, and after a longer or shorter interval the systolic murmur is preceded by a presystolic murmur, the other signs of mitral obstruction concurring. It may be difficult in some cases to estimate the significance of the systolic murmur. The murmur may be very loud, and heard in the left axilla and at the back : if so, there can be little doubt that it is due to regurgitation from organic disease. Or it may be heard over a very restricted area, not conducted to the axilla, but just over the apex itself. In such cases the auriculo-ventricular orifice may not be widened by any retraction of curtains or columns, and the anatomical lesion may be obstruction, but the auricular muscle may have become weak ; therefore regurgitation, which previously had been prevented, is now permitted. Or the murmur may be observed to the right of the position of the apex close to the tricuspid area ; in such cases the probability of tricuspid regurgitation must be borne in mind.

In some cases rheumatic phenomena are declared, not in the early stages of the affection, but subsequently, during the observation of the case. For instance, a girl of fourteen, without any rheumatic antecedent —though there was a hereditary tendency thereto on the mother's side— manifested a prolonged systolic and a short presystolic mitral murmur. There were no rheumatic phenomena for thirteen months when polyarticular rheumatism appeared. At that time a marked thrill was felt at the apex ; a grating presystolic and a prolonged blowing systolic murmur were heard, and the heart was enlarged, especially as regards the right chambers. The necropsy shewed a funnel-shaped transformation of the mitral valve and a ring of small vegetations (recent rheumatic endocarditis) encircling the auriculo-ventricular orifices. This affords one of many pieces of evidence that the rheumatism which is associated with mitral disease may be unattended for long periods by obvious symptoms.

A sign of the advent of the structural change in the valve inducing obstruction at the mitral orifice is a double-shock sound heard during the period of ventricular diastole, and resembling a doubling of the second sound over the apex of the heart. This simulated doubling of the second sound at the apex has been found in a large number of cases which eventually manifested all the usual signs of the lesion. Dr. Cheadle found " 33 cases with presystolic murmur, and 24 with reduplicated second sound at the apex indicating commencing stenosis, out of 273 cases of organic heart disease in children." He adds : " There can be no question as to the connexion of this morbid sound with early mitral stenosis, and of its clinical significance." Potain (58) has confirmed these observations, ascribing the sound to causes affecting the mitral valve. The first element is the normal second sound heard at the apex, the second element occurring soon after it, the " claquement de l'ouverture de la mitrale."

In a large number of cases the clinical signs of association with rheumatism are insignificant. In a considerable proportion the origin and progress of the morbid changes in the valves and the adjacent structures are insidious and gradual. The disease which initiates these is not independent of rheumatism, but is often unaccompanied by pronounced rheumatic phenomena. It is far from uncommon to find mitral obstruction following attacks of chorea or of erythema nodosum ; and, although not so common, it is sometimes the sequel to scarlet fever, attended by peliosis rheumatica. The endocarditis which results in mitral insufficiency is more violent and more obviously associated with ordinary acute rheumatism ; that which induces obstruction is more protracted and symptomless, giving rise to a gradual welding of the curtains and a slow formation of fibrous tissue which, under the even pressure of the blood within the auricle and the ventricle, tends to the production of a smooth septum. This septum becomes gradually thicker, for it has to bear the chief strain of the auricular pressure—not the ventricle, as in the case of mitral insufficiency.

When the acute signs of rheumatic endocarditis have passed away, or when, in the absence of any obviously acute manifestation, the obstructive lesion has been gradually induced, compensation enduring for protracted periods may ensue. Such compensation is a simpler matter than in the case of mitral insufficiency, for an increase of power in the muscle of the right ventricle and of the left auricle only is necessary to maintain it ; enhanced force and increased capacity of the left ventricle not being also required as in the structural lesion inducing mitral regurgitation. The left ventricle may deviate but little from the normal, and a strong right ventricle, aided by a hypertrophied, or at least not enfeebled, auricle, will urge a sufficiency of blood through the narrow orifice.

The *symptoms* of failure of compensation differ in many points from those in cases of mitral insufficiency. In the latter the signs are more uniform—the dyspnoea of effort, or the paroxysmal dyspnoea progressively increasing in intensity, the gradual oncome of dropsy, and other signs to

be detailed in the next section are evidenced ; in mitral obstruction, on the other hand, the symptoms are more erratic, the accidents of the disease predominate, and it is these rather than the gradual heart-failure that have in the greatest degree to be reckoned with.

One of the earliest symptoms to attract attention in cases of mitral stenosis is epistaxis ; Duroziez has noted this, and his observations have been fully substantiated.   Probably we are not told of this symptom in many of our cases in hospital because it is considered trivial.   In some, though in a less proportion than might be imagined, there have been complaints that the patient is soon "out of breath."   Precordial pain and distress are noted, however, in a considerable number of patients, and in some of these palpitation.   Haemoptysis has often been recorded ; it also occurs in the course of the lung affections in this disease.   The most frequent of all the induced morbid states is that evidenced by dyspnoea, cough, and other symptoms referred to the lungs.   In some cases there is a general bronchitis ; but in the great majority there are signs of a localised pneumonia, in the course of which the sputa are frequently blood-stained.   The bronchitis can be referred to the general venous engorgement of the lungs, but the localised consolidations are proved, by morbid anatomy as well as by clinical evidence, to be due to infarctions of branches of the pulmonary artery.   These occur with the highest degree of frequency in mitral obstruction ; and probably in half the cases observed they have been manifested at some time of the life-history. The haemoptysis with lung signs often suggests the probability of pulmonary tuberculosis ; but in the vast majority of cases this is negatived. Its occurrence in a small minority has been mentioned ; investigation should accordingly be made for tubercle bacilli in the sputum, and the other related signs should be duly weighed.   Other symptoms which occur in the course of mitral obstruction, increasing the dangers of the disease and adding new difficulties to its treatment, are those due to embolism of the systemic arteries.   These will be considered in the next group of cases.   In only a few cases are they clinically observed in the spleen, though morbid anatomy teaches that this is a very frequent site of embolism.   Probably the symptoms thus occasioned—consisting in painful enlargement, with friction and tenderness, as well as pyrexia, which may simulate pleurisy—pass in many cases unnoticed and unknown.   It is otherwise when an intracranial artery is thus blocked —then the danger of the condition is proclaimed.   It is to be remembered that these emboli—whether in the pulmonary or in the systemic circulation—do not always occur in mitral stenosis from detachment of the vegetations of acute endocarditis, but very frequently from plugs passively formed within the chambers of the heart.   Frequently, therefore, they are the first manifestations of disease, and not symptoms developed during an acute or subacute illness.   They occur both in the cases which are obviously associated with rheumatism, and those which present no such evident relation.   Of course they tend further to disturb compensation, though in many cases there is recovery for long periods.

Generally speaking, in the cases of mitral obstruction oedema is not nearly so marked a symptom as in the cases of mitral insufficiency. A fugitive and slight oedema occurs in many of them, but general dropsy rarely until the final stages, when the right chambers of the heart have become dilated and the tricuspid valve incompetent; many patients die before this stage is reached. Sir W. Broadbent (10) noted that great enlargement of the liver with true pulsation of this organ is more frequently found as a consequence of mitral obstruction than of other valvular affections; and it is not uncommon to find fluid in the peritoneal cavity before oedema of the feet and legs. The oedema will disappear with rest in bed while ascites remains for a time; whereas cardiac dropsy in mitral and tricuspid insufficiency begins, as a rule, in the connective tissue of the most dependent parts.

In the rheumatic group of cases the influence of sex in the disposition to the obstructive mitral lesion is well marked and difficult to explain. Of 263 cases of all forms of mitral obstruction collected by Sir Dyce Duckworth (19), 177 were female and 86 male. In Hayden's cases the proportion of females to males was two to one. In Broadbent's list (10) of 53 cases examined post-mortem, 38 were females and only 15 males. Sir Dyce Duckworth concluded that in 70 per cent of the cases of mitral stenosis tabulated by him there was a certain or strong presumption of rheumatic antecedents; and he considered this estimate of the relation to rheumatism to be rather under than over the mark. In regard to Sansom's cases, in 17 necropsies of children manifesting mitral obstruction in conjunction with pericarditis or endocarditis of the rheumatic form, 10 were females. Of 35 children under twelve years clinically observed, 22 were females; of 31 adults with mitral stenosis in distinct association with rheumatism, 18 were female. It would appear, therefore, that the preponderance of cases in the female sex in his experience is not so great as in that of other observers. It must be remembered that this statement refers to those only in which the rheumatic association seemed to be strongly accentuated; the groups of cases not decidedly associated with rheumatism will be considered hereafter.

Prognosis.—Sansom (76) found the average age at death of 61 patients with mitral stenosis to be 32·7. Hayden's cases—42 in number—gave an average age of 37·8. Broadbent stated that the average age at death, deduced from 53 cases abstracted from the post-mortem records at St. Mary's Hospital, was thirty-three for males and thirty-seven for females; and he adds: "Mitral stenosis stands next to aortic regurgitation among valvular affections in the order of gravity." Sansom (76) had records of 17 cases fatal before the age of twelve years, the average being $9\frac{1}{2}$ years; 10 of these at the age of ten. The association with rheumatism is shewn by the fact that, in addition to the valve-lesion, in 14 of these either pericarditis or recent endocarditis of rheumatic characters were found on necropsy. The rheumatic associations of the majority of cases of mitral obstruction constitute a very great, if not the chief, element of danger. The other causes of fatality will be

pointed out in the consideration of the other groups. It must be accepted as a general proposition that the subjects of mitral obstruction (discovered at an early age) rarely survive the age of forty; the disease, therefore, when dating from childhood and adolescence, and in such cases having its origin in a rheumatic affection, is of grave significance.

Treatment.—The recognition of the rheumatic association of the disease is of much importance in treatment. In childhood and adolescence a slight febrile attack in the subject of mitral obstruction, or in one who presents signs of the advent of the lesion, should be held as a probable indication of a subacute rheumatism; and treatment by complete rest with the administration of salicin or the salicylates should be enjoined. If cough and difficulties of breathing are also present, symptoms of bronchitis or pneumonia, the systematic administration of ammonia in addition is valuable. The frequency of blocking of branches of the pulmonary artery has already been pointed out. The frequent administration of liquor ammoniae, well diluted, has been urged as a means of lessening the tendency to coagulation of blood. There are differences of opinion regarding this matter, but there are many reasons in favour of the treatment. Besides diminishing the coagulability of the blood-plasma, ammonia is a valuable stimulant of the nervous mechanism of the heart and of the respiratory centre; by increasing the bronchial secretion and rendering it more fluid, it acts very favourably as an expectorant, while it has also some value as a vaso-dilator. The best mode of administration in young subjects is the liquor ammoniae fortior in doses of one to five minims, with liquid extract of liquorice well diluted with water; the dose being repeated—according to the urgency of the case—every half-hour, every hour, or every two hours until signs of improvement appear. It may then be continued every four hours for several days. Whether there be bronchitis from venous congestion, or local consolidations of the lungs from infarcts, the ammonia treatment is valuable. It may be well to issue a caution against the use of digitalis during any febrile manifestation in these cases. It is often worse than useless. The haemoptysis which may occur should not be treated by styptics or opium. As a general rule it is better that any haemorrhage which breaks out in the course of mitral stenosis should not be checked by drug treatment. A like medicinal treatment to that just mentioned may be put in force in cases in which precordial pain or distress is manifested in the subjects of mitral stenosis.

It is to be remembered that pericarditis arises not infrequently in this connexion, when the special treatment for this disease must be carried out. The occurrence of pericarditis or of lung complications of any kind may rapidly break the compensation in mitral obstruction; and inadequacy of the right heart, with dropsy and other signs of heart-failure, may occur. In such case the treatment should be that described under mitral insufficiency. The symptoms, however, are frequently recovered from, and compensation is restored.

While there are any indications of acute changes—of endocarditis,

of pericarditis, of rheumatism, or of any symptoms attended by pyrexia
—perfect rest in bed should be enjoined. It is otherwise, however, in
convalescence, when it is to be presumed that sclerosing changes in the
valve structures are going on. Then systematic exercises, gradual and
tentative at first, should be recommended, for they fulfil important
indications ; they not only aid the venous circulation, but by expanding
the thorax they tend to aspirate the heart, increase the overflow from
auricle to ventricle, and perhaps prevent the imminent danger of the
progressively increasing contraction of the auriculo-ventricular aperture.
It may be urged that a danger of such exercises may be a detachment of
a vegetation left by the rheumatic endocarditis ; this is possible, but it is
proved that the greater danger is the passive formation of thrombi within
the heart in consequence of retarded circulation within it. The patient
should be cautioned against violent movements, but there can be no
doubt of the value of systematic exercises. During convalescence from
any acute febrile manifestation in the subject of mitral obstruction, the
first method employed should be gentle massage—especially vibratory
massage of the precordia ; then movements of the legs, the patient being
quite in bed ; next in order the arms. Later, expansion movements of
the thorax, made by the patient himself cautiously and deliberately,
should be practised, with judicious intervals of rest. Concurrently, or
just subsequently to these movements, there should be spongings, first
with warm and afterwards with cool water, followed by dry towel friction.
Later systematic muscular exercises may be used (*vide* p. 409).

Although moderate exercise in the fresh air in the subject of fairly
compensated disease is salutary, sudden over-strain is dangerous. In
some cases breathlessness does not come as a warning, and patients
persist in overtaxing their strength. The subject of mitral obstruction
should be protected from chills by suitable apparel; pure woollen clothing
is the best. A light woollen night-dress is also to be recommended.
Heavy overcoats and sealskins, which weigh down the shoulders and
thus prevent good expansion of the thorax, should be deprecated.

The late Sir Andrew Clark, in a clinical lecture which was published
after his death, gave some valuable hygienic rules for patients with mitral
obstruction. In the daily dietary fluids should be restricted, for after
their absorption they distend the vascular system, and increase the bulk
without increasing the nutritive value of the blood within the vessels.
The ingestion of much liquid enfeebles the heart and increases the labour
of the right ventricle and left auricle in the transmission of blood
through the narrowed aperture into the left ventricle. The patient
should have three good meals a day as dry as he can take them ; over-
eating and indigestible foods must be strictly guarded against. It is a
good plan to advise that the two meals of the day of which meats form a
portion should be taken without alcohol, and with a little pure water or
toast water only ; and subsequently to each of these a wineglassful of
milk with two teaspoonfuls of good old brandy or whisky may be
allowed. In some patients there is a slight appearance of jaundice, the

liver is embarrassed, and there is often constipation. There may be basic congestion of the lungs. Sir Andrew Clark said, "To relieve the lungs give something to relieve the bowels." Sulphate of sodium and phosphate of sodium, equal parts in powder, may be administered in doses of two or three teaspoonfuls dissolved in water in the morning, or a teaspoonful of sulphate of magnesium may be taken in hot water. Such aperients relieve the portal system, and so the right side of the heart and the lungs. Mercurial purgatives are frequently of signal service.

The routine administration of digitalis in cases of mitral obstruction is to be condemned. Very often it does harm. When once a patient manifesting the physical signs of mitral stenosis has recovered from any intercurrent disease which has disturbed the compensation, careful hygienic treatment and the administration of ordinary tonics are all that is necessary ; all the special heart tonics should be avoided. When, however, the right heart begins to fail, or dropsy to appear, some special heart treatment becomes necessary. Even then in many cases the administration of digitalis cannot be advised with the same confidence as in cases of mitral regurgitation. In many it causes the heart's action to become irregular, or increases an already existing irregularity ; in some it induces nausea and vomiting, in others precordial oppression. Strophanthus is useful in many cases, but, like digitalis, should not be continued for long periods. Sometimes convallaria acts more beneficially in these cases than digitalis ; it favourably influences the irregularity, and acts as a powerful diuretic. The extractum convallariae fluidum in doses of 5 to 10 minims, or the tinctura convallariae in doses of 10 to 20 minims, may be administered every four hours, or three times a day, in adults. When there are serious symptoms of heart-failure—the radial pulse small and irregular, while the right ventricle is felt to beat forcibly, and the veins of the neck are seen to be distended, and perhaps pulsating, the patient being pale or dusky and breathing badly—relief of the venous engorgement by venesection is a valuable means of treatment. The ordinary method of opening the vein in the arm and permitting the flow of about six ounces of blood is the best, but this is often objected to ; if so, six or eight leeches may be applied over the epigastrium. In children the relief given by the abstraction of blood by two or three leeches is very well marked. After abstraction of blood digitalis and other heart tonics often act more favourably than they would have done before the relief of the venous engorgement.

Group II. Cases in which the Disease is first declared by Symptoms of Lesion of the Nervous System.—Not uncommonly a patient comes under medical care for a lesion of the nervous system which has suddenly shewn itself and the diagnosis of mitral obstruction is made for the first time. If rheumatic manifestations existed at any period of the previous history of the patient these were trivial and unnoticed. The physical signs indicate a pure mitral obstruction ; there is no evidence of mitral regurgitation. In fatal cases, for the most part, the funnel form of mitral constriction is found. In many there is good reason, from the

hereditary bent, or from the occurrence of some symptoms which suggest such a proclivity, to suspect that these insidious morbid changes had their origin in rheumatism ; but it may not be so in all cases.   It is possible that the haematomas of the delicate mitral flaps in infancy may be the starting-points of the fibrous proliferation ; or vascular dilatations or haemorrhages from the fine vessels of the growing valve may be the earliest changes.   At any rate the only cause concerning which we have precise evidence is rheumatism.

The most characteristic amongst the severe lesions of the nervous system is hemiplegia.   In one case, a girl of ten, the first detected sign was sudden paralysis of the right arm and leg ; the child recovered completely from the paralysis, but died seven months afterwards after having manifested much precordial distress.   Mitral obstruction was demonstrated at the necropsy, and there was universal adhesion of the pericardium.   In another patient, a woman aged twenty-two, who had never manifested any symptom of rheumatism, and who had no hereditary tendency thereto, sudden right hemiplegia occurred with aphasia.   There were pronounced physical signs of mitral obstruction without regurgitation.   The patient made a perfect recovery from the paralysis of motion, but complete aphasia persisted (75).   In another case, also a woman, left hemiplegia occurred ; after full recovery from this lesion right hemiplegia came on suddenly ; from this latter attack the recovery was but partial.   In Duroziez's 43 cases of "pure" mitral obstruction in females, 11 manifested right hemiplegia with aphasia, and 4 hemiplegia without aphasia ; there were no such cases in the male sex.

Another nervous disorder which may suddenly arise in subjects of the affection is hemichorea.   In a series of 38 cases of mitral stenosis, there were 4 of hemichorea.   Duroziez records a case of a woman, aged twenty-four, with mitral stenosis declared by right hemichorea in which the convulsive movements of the limbs ceased, but chorea of articulation remained, so that the beginning only of each word was uttered.   One of Sansom's patients (76), a boy aged $3\frac{1}{2}$, was suddenly seized with epilepsy, the unconsciousness lasting twenty minutes.   Nine months afterwards chorea became manifest ; recovery took place, but after a second period of nine months another attack of chorea occurred ; there were well-marked physical signs of mitral stenosis.   In a boy, aged five, who manifested a presystolic murmur and thrill, a fit had occurred eighteen months previously, attended by unconsciousness so profound that the child was thought to be dead ; nine months afterwards chorea appeared.   In another case, a girl aged five, epilepsy occurred, and the attacks were repeated and severe.   In a lad, aged eighteen, in whom there was an opportunity of watching the physical signs of the gradual establishment of mitral obstruction, from the manifestation of a soft apical systolic murmur to that of complete and characteristic presystolic murmur, thrill, and doubled second sound, there occurred during his exercise in the garden a sudden unconsciousness, which was complete for a minute or two, but was not attended by muscular spasm.

It is reasonable to conclude that many of these sudden perturbations of the nervous system are caused by embolism of branches of the intra-cranial arteries; in some instances this was positively proved by necropsies. It is clear that the consequences of such embolism may in some cases pass away completely; in others the plugging of the vessel is followed by necrosis of the nervous structures thus supplied. It is, nevertheless, possible that in some cases, particularly of chorea, the organism of rheumatism or its toxins may be the cause of the symptoms.

In the *treatment* of such cases complete rest should be promptly enjoined. There is fair evidence that the ammonia treatment, as described in relation with embolisms of the pulmonary artery and its branches, may fulfil a useful purpose. The use of salicin or one of its congeners must be adopted if there be any rheumatic manifestations.

Group III. Cases presenting Disorders of Nutrition.—Children are not infrequently brought for treatment on account of their pro-gressive wasting. The parents, or those who have charge of them, think they are "in a consumption." On removal of the clothing the emaciation is seen to be considerable; the ribs stand out and the inter-costal spaces are sunken, except in some cases over the situation of the right ventricle, where there is a marked prominence; on further examination the physical signs of mitral stenosis are in full evidence. In those who have arrived at adolescence or adult life there are other signs of ill development. The patients are indisposed for exertion (though they seldom complain of breathlessness); they are unstable and infirm of purpose, are accounted very nervous, and in some instances are demented; they are frequently dyspeptic. The elucidation of the condition is in fatal cases made by the post-mortem examination; constriction of the mitral orifice is found, and the enlarged right chambers of the heart contrast with a small left ventricle and small aorta. The normal arterial blood-supply has been gradually diminished by the contraction of the mitral orifice, and has continued to be in minus quantity during the periods of development and growth. As Sir Samuel Wilks pointed out, "The lungs are small as well as the chest, and the respiratory process is correspondingly lowered, and with this probably the whole body is impoverished. At all events, the organism is working with a diminished amount of blood."

In young women—and in the great majority of such cases, even in childhood, the patients are of the female sex—there is frequently, though not invariably, an association with anaemia and chlorosis. The frequency with which a chlorotic patient has presented physical signs of mitral obstruction has been noted by many observers. Stokes was the first to record this in describing the case of a young girl, aged eighteen, who was chlorotic, and shewed the physical signs of organic mitral disease, the precise form of the lesion being then undiscovered. Death occurred after the manifestation of anasarca and congestion of the lungs, and the necropsy revealed the funnel form of mitral obstruction, with an auriculo-

ventricular aperture that scarcely admitted a goose-quill. This case may be regarded as an exemplary one. It is common to find instances of marked anaemia, some not presenting signs of wasting, in which there is well-marked physical evidence of mitral obstruction without regurgitation. Duroziez, who has given the notes of many cases, goes so far as to say that pure mitral stenosis is a *feminine* and a *chlorotic* malady. Teissier points out that a similar anaemia occurs, though more rarely, in the male subjects of mitral stenosis. The subject of anaemia as a cause of mitral disease has been reviewed by Dr. Goodhart.

In any of the cases in this group haemoptysis may occur, and local consolidations may be found in the lungs—the group of symptoms closely resembling those of pulmonary tuberculosis. In the great majority the diagnosis of pulmonary consumption is not justified ; the symptoms are the accidents of the mitral disease itself. Reasons for dissenting from the view that mitral stenosis can be considered as standing, even remotely, in any causal relation to tuberculosis have already been stated ; it is probable that the deficient arterial supply which is a consequence of the disease disposes to the occurrence of tuberculous changes in the lungs in a small minority of the cases, and the remote probability of this should be present in the mind of the observer. The presence or absence of tubercle bacilli in the sputa will settle the question, if there is any expectoration.

In the *treatment* of this group of cases physical training should hold a first place. It is evidently of the highest importance that the blood-flow from the right to the left ventricle should by judicious means be increased. It is possible that if this be accomplished by systematic muscular movements and careful hygiene at an early period of the mani-festation of the morbid condition, the insidious contraction of the orifice may be averted. The means to this end are friction, massage, carefully planned muscular movements, baths and bathing, the selection of suitable climates, and the regulation of diet. Medicinally iron, arsenic, strych-nine, and cod-liver oil are the chief agents to be employed. The treat-ment of complications and of failure of compensation will be as in other groups of the disease.

Group IV. Cases associated with Chronic Renal Disease and Arteriosclerosis. — As already stated, the association between mitral obstruction and chronic renal disease was first pointed out by Dr. Goodhart and confirmed by Dr. Newton Pitt. The observations were made chiefly from the standpoint of morbid anatomy, though Dr. Newton Pitt contributed some clinical data. It was made clear that the cases demonstrating the coexistence of the two morbid states are by no means infrequent. Nevertheless Gérard and others hold that mitral obstruction having its origin in arteriosclerosis is rare. It cannot be doubted that the explanation of this apparent conflict is that the cases demonstrating the conjunction of the diseases are most frequently found after death : they come under clinical observation with comparative rarity. The two morbid affections progress insidiously, and the patient is either

suddenly stricken down with apoplexy, or some sudden complication
which precludes any physical examination, or, if such examination has
been possible, the physical signs were supposed to indicate some form of
disease other than mitral obstruction.

Numerous cases have been observed, in which, without any evidence
of rheumatism or other disposing malady, there have been signs which
should bring them into the group under consideration. In many other
cases aortic valvular disease has been conjoined with the mitral. Others
again can be regarded as mixed cases, having rheumatic antecedents ;
but the subsequent evolution has been after the manner of arterio-
sclerosis. The patients are usually elderly, but occasionally the combina-
tion is found in those who are still young. Sometimes the association is
observed in patients who suffer from hereditary syphilis. In the majority
of cases the usual signs of chronic Bright's disease are present. The radial
and other arteries are thick and tortuous. The arterial pressure is higher ;
the hypertrophy of the left ventricle of the heart, however, is not so obvious
as usual. In some of the cases, in addition to the signs of thickened
arteries, there are obvious evidences of gout in the joints and else-
where. In some there are well-marked signs of arterial sclerosis. There
may be emphysema of the lungs or pulmonary fibrosis. Fibroid changes
may occur about the viscera, the perivisceritis of Huchard. The origin
of the disease is not to be traced, the progress is slow and imperceptible.
The physical signs of mitral obstruction in many of the cases do not differ
from those ordinarily observed—the presystolic, the entire diastolic, or
the early or mid-diastolic murmur, the sudden, loud first sound and the
double-shock sound in diastole. In some cases there is no presystolic
murmur, but a systolic. This may be heard at the apex and the back,
thus answering to the criteria of mitral regurgitation ; in such cases it is
probable that the auricle has become dilated and weak. The diagnosis
can then only be made from the evidence of a heaving and enlarged
right ventricle, contrasting with the absence of signs of enlargement of
the left ventricle, perhaps also from the absence of any second sound at
the apex. Exceptionally there is no loud, sharp, short, sudden first
sound, but a dull sound as in the case of a hypertrophied left ventricle.
In the cases manifested between the ages of thirty and forty, there have
been the evidences of the gradual onset of chronic renal disease with
thickened arteries or undoubted gout with deposits of urates. There
is no evidence of any pre-existing disease of the valve due to rheumatic
or other causes ; but there must be a remaining doubt whether any
change in the valve preceded the fibrous proliferations intrinsic to the
Bright's disease. It is improbable, seeing that the great majority of
cases due to rheumatism are fatal before the age of forty, that chronic
Bright's disease is a superadded factor, for if so the scene would be more
speedily closed, and death would ensue. It is at first sight more likely
that the changes are independent of rheumatism and due to a slow form
of sclerosis.

In one case, that of a lady aged fifty-two, under the care of the late

Dr. Sansom, there was ample opportunity of observing the gradual evolution of the disease as evidenced by the physical signs and confirmed by necropsy. There was at first no evidence whatever of cardiac disease, but gradually all the usual signs of mitral obstruction were manifested. The urine shewed normal characters for nearly the whole period of observation, and the case was observed during thirteen years. The symptoms were those of dyspepsia, with gradual implication of the nervous system, first evidenced by an epileptic attack and afterwards by dementia. The necropsy shewed funnel transformation of the mitral valve, with much fibrous thickenings of the surrounding structures. There were chronic fibroid thickenings in the pleurae, the lung, the spleen, the liver, the capsules of the kidneys, and the membranes of the brain. The granular changes in the kidneys were but slightly pronounced. The chief morbid change was the widely-spread fibrosis, the progress of which had been very gradual; and it seemed legitimate to infer that the affection of the mitral orifice and the fibrous transformation of the surrounding structures were due to a similar morbid process. This case is no doubt exceptional in that the fibroid changes in so many situations long preceded any signs of interstitial nephritis. In the majority of cases the evidence of chronic renal disease is well marked when the case comes under observation. The age fifty to seventy renders it improbable that obstruction of the mitral orifice from any cause had preceded the general evolution of the chronic renal disease with its attendant arteriosclerosis. In all cases the progress of the disease must have been very gradual and insidious. In many there have been signs of cerebral disease; indeed, it is for symptoms indicating such disease that the cases usually come under notice. Epileptiform seizures, apoplexy, dementia, or uraemia are the chief forms. In several instances the signs of albuminuric retinitis have been recorded. That the morbid changes have been slow and gradual is shewn also by the post-mortem evidence. In the case of a woman of sixty-five there has been found a funnel transformation of the mitral curtains, just as observed in the cases of earlier life; but in the majority the button-hole form of mitral stenosis is manifested with great thickening and firm fibrous transformation of the papillary muscles.

The *treatment* in this group of cases is subordinate to that of the chronic renal disease and the attendant thickenings of the arteries. It is important to realise that the prognosis is very grave. When a patient manifesting the signs of mitral obstruction at whatsoever age presents signs of firm and thick arteries, and the urine is found to be continually of low specific gravity and occasionally albuminous, it is well that for a few weeks a rigid milk dietary be enjoined. It may be a little difficult to convince a patient past middle age, whose stomach has been the receptacle of foods of many and various kinds, far more than adequate to the needs of his organism, whose nerves of taste have been frequently and abnormally stimulated, and whose absorption of nutritive material and excretion of effete products have been inversely proportional, that he must return to the sweet simplicity of the earliest months of his life. Yet it is best so.

It is the absence of the irritation to the arterioles caused by the complex albuminoids which turns the balance towards amendment. It may be necessary, however, to make some concessions. In the early morning, or on waking, the patient may take half a pint of milk with half an ounce of rum, or of cognac and an ounce of lime water. In some cases one or two ounces of fluid magnesia may be substituted with advantage for the lime water. Three or four hours afterwards a second half-pint of milk may be taken flavoured with a little hot coffee; the third half-pint, after a like interval, may be taken as a blancmange made with isinglass or gelatin. At similar intervals, during the remainder of the waking hours, the changes may be rung with the various flavourings; but no solids should be permitted other than light biscuits.

The total amount of milk taken in the twenty-four hours should be from three to six pints. The total quantity of cognac or spirits of any kind should be limited to two ounces. To break the monotony of the purely milk diet, it is a good plan to allow occasionally a firm jelly fully flavoured with madeira, rum, kirschwasser, or chartreuse. One or two tablespoonfuls of isinglass are to be melted in very hot water, and the milk added thereto; the small quantity of gelatin thus mingled with the milk is sufficient to prevent any firm curdling of the casein in the stomach, the coagulum being rendered much softer and its digestion facilitated (72).

In regard to medicinal treatment, the rule of Balfour (3) should be followed, that no cardiac tonic should be administered without a simultaneous unlocking of the arterioles. As Broadbent said, "Nitroglycerin and other vaso-dilators may sometimes be given with good effect for many weeks or even months in conjunction with general tonics, such as iron, quinine, and nux vomica" (11). But the most important remedies for such cases are the iodides. In moderate doses the iodide of potassium or of sodium should be administered for long periods. If for any reason they are not well tolerated, hydriodic acid in the form of the syrup or another of its modern preparations may be substituted.

REFERENCES

1. ACLAND, T. D. *Lancet*, 1889, i. 103, 149.—2. ADAMS. *Dublin Hosp. Rep.*, 1827, iv. 353.—3. BALFOUR. *Clinical Lectures on Diseases of the Heart*, London, 1876, 101.—4. *Idem.* *Lancet*, 1872, i. 714.—5. BARCLAY. *Ibid.*, 1872, i. 283, 353, 394.—6. BARR, Sir J. *Liverpool Med.-Chir. Journ.*, 1894, 85.—7. BERTIN. *Traité des maladies du cœur*, Paris, 1824, 176, 186.—8. BLIND. "Le rétrécissement mitral des artério-scléreux," *Thèse de Paris*, 1894.—9. BOYD, F. D. "Apparent Reduplication of the Second Sound in Mitral Stenosis," *Lancet*, 1896, ii. 1685.—10. BROADBENT, Sir W. *Amer. Journ. Med. Sc.*, Phila., 1886, N.S. xci. 57.—11. BROADBENT, Sir W. and J. *Heart Disease*, London, 1897, 192.—12. BROCKBANK. *Med. Chron.*, Manchester, 1894, vii. 161.—13. BRYANT, J. H. *Guy's Hosp. Rep.* lv. 83.—14. CHAUVEAU et MAREY. *Gaz. méd. de Paris*, 1861, sér. iii., xvi. 675.—15. CHEADLE. *The Various Manifestations of the Rheumatic State as exemplified in Children and Early Life*, London, 1889, 114. — 16. CLARK, Sir A. *Lancet*, 1893, ii. 1367. — 17. CORVISART. *Essai sur les maladies et les lésions organiques du cœur*, Paris, 1806, 236.—17a. CROOM. *Edin. Med. Journ.*, 1905, N.S., xviii. 231. — 18. DICKINSON, W. H. *Lancet*, 1887, ii. 650, 695. — 19. DUCKWORTH. *St. Barth. Hosp. Rep.*, London, 1877, xiii. 263.—20. *Idem.* *Brit. Med. Journ.*, 1888, i. 246.—21. DUROZIEZ. *Traité*

clinique des maladies du cœur, Paris, 1891, 259.—22. FAGGE. *Guy's Hosp. Rep.*, London, 1871, xvi. 247.—23. *Idem.* Reynolds's *System of Medicine*, London, 1877, iv. 601.—24. FAUVEL. *Arch. gén. de méd.*, Paris, 1843, sér. 4, i. 1.—25. FISHER. *Lancet*, 1895, i. 609.—26. FLINT. *Amer. Journ. Med. Sc.*, Phila., 1862, N.S., xliv. 29, and 1886, xci. 35.—27. FRIEDREICH. *Krankheiten des Herzens*, Erlangen, 1867, S. 230.—28. GAIRDNER. *Clinical Medicine*, Edinburgh, 1862, 574.—29. *Idem. Edin. Hosp. Rep.*, 1893, i. 221.—30. GÉRARD. "L'oreillette gauche dans le rétrécissement mitral," *Thèse de Paris*, 1894.—31. GIBSON, G. A. *Edin. Med. Journ.*, 1880, xxv. 982.—32. *Idem. Ibid.*, 1900, viii., N.S. 212.—33. *Idem. Diseases of the Heart and Aorta*, Edinburgh and London, 1898, 158.—34. *Idem. Birmingham Med. Rev.*, 1891, xxx. 329. — 35. *Idem. Edin. Hosp. Rep.*, 1894, ii. 332. — 36. *Idem. Practitioner*, 1905, lxxiv. 595. — 36a. *Idem. Quart. Journ. Med.*, Oxford, 1908, i. 103. — 37. GIBSON and MALET. *Journ. Anat. and Physiol.*, London, 1879, xiv. 1. — 38. GOODHART. "Anaemia as a Cause of Heart Disease," *Lancet*, 1880, i. 479. — 39. GOURAUD. *De l'influence pathogénique des maladies pulmonaires sur le cœur droit*, Paris, 1865, 45.—40. HAYDEN. *Diseases of the Heart and Aorta*, Dublin, 1875, 188, 893, 906.—41. HOPE. *A Treatise on the Diseases of the Heart and Great Vessels*, London, 1832, 341. — 42. HUCHARD. *Traité clinique des maladies du cœur*, Paris, 1893, 2nd ed., 180. — 43. HUNTER.—*A Treatise on the Blood, Inflammation, and Gunshot Wounds*, London, 1794, 162.—44. KEITH, A. *Journ. Anat. and Physiol.*, London, 1907, xlii. 1.—45. *Idem. Lancet*, 1904, i. 706.—46. KIDD. "The Association of Pulmonary Tuberculosis with Disease of the Heart," *St. Barth. Hosp. Rep.*, 1887, xxiii. 239.—47. KING, T. W. *Guy's Hosp. Rep.*, 1840, v. 27.—48. LAENNEC. *De l'auscultation médiate*, Paris, 1819, 211. — 49. LANCEREAUX. *Atlas d'anatomic pathologique*, Paris, 1871, texte 214.—50. LETULLE. *Anatomie pathologique*, Paris, 1897, 114.—51. MACKENZIE, J. *Journ. Path. and Bacteriol.*, Edin. and London, 1894, ii. 84.—52. *Idem. Brit. Med. Journ.*, 1905, ii.—52a. *Idem. Diseases of the Heart*, 1908, p. 223.—53. M'VAIL. *Brit. Med. Journ.*, London, 1887, ii. 786. — 54. MORGAGNI. "*De Sedibus et Causis Morborum*," Venetiis, 1762, tom. i. 208.—55. ORMEROD. *Med. Times and Gaz.*, London, 1864, ii. 154.—55a. OSLER. *Montreal Med. Journ.*, 1909, xxxviii. 79.—56. PHEAR. "On Presystolic Apex Murmur without Mitral Stenosis," *Lancet*, 1895, ii. 716 ; and "On Reduplication of the Second Sound," *Ibid.*, 1897, i.—57. PITT, G. NEWTON. *Brit. Med. Journ.*, 1887, i. 108.—58. POTAIN. *Clinique médicale de la Charité*, Paris, 1894.—59. *Idem. Bull. et mém. Soc. méd. des hôp. de Paris*, 1876, sér. 2, xii. 137.—60. *Idem. Gaz. des hôp.*, Paris, 1880, liii. 529.—61. POPOFF. *Virchows Festschrift* (Internat.), Berlin, 1891, iii. 333.—62. POWELL, Sir R. DOUGLAS. *Med. Times and Gaz.*, 1871, i.—63. *Idem. Lancet*, 1880, i. 277, and ii. 123.—64. ROLLESTON. *St. Barth. Hosp. Rep.*, 1888, xxiv. 197.—65. ROUCHÈS. "Du claquement d'ouverture de la mitrale," *Thèse de Paris*, 1888.—66. SAMWAYS. *Le rôle de l'oreillette gauche*, Paris, 1896.—67. *Idem. Brit. Med. Journ.*, 1897, i. 199.—68. *Idem. Ibid.*, 1896, ii. 1567. —69. SANSOM. *Trans. Med. Soc.*, London, 1890, 143.—70. *Idem. Liverpool Med.-Chir. Journ.*, 1892, 6.—71. *Idem. Diagnosis of Diseases of the Heart*, London, 1892, 383.—72. *Idem. Twentieth Century Medicine*, New York, 1895, 477.—73. *Idem. Lettsomian Lectures on Valvular Diseases of the Heart*, London, 1886, 137.—74. *Idem.* Keating's *Cyclopaedia of the Diseases of Children*, Phila., 1889, ii. 831.—75. *Idem. Trans. Clin. Soc.*, London, 1894, xxvii. 268.—76. *Idem.* This *System*, 1st. ed., 1898, v. 1007.—77. STEELL, GRAHAM. *Physical Signs of Cardiac Disease*, Edin., 1881, 43.— 78. *Idem. Med. Chron.*, Manchester, 1889, ix. 182.—79. STOKES. *Diseases of the Heart and Aorta*, Dublin, 1854, 191.—80. TEISSIER. *Clinique médicale de la Charité*, Paris, 1894, 913.—81. THAYER, W. S. "On the Frequency and Diagnosis of the Flint Murmur in Aortic Insufficiency," *Trans. Assoc. Amer. Physic.*, 1901, xvi. 303.— 82. TRIPIER et DEVIC. *Traité de pathologie générale*, par C. Bouchard, Paris, 1897, t. iv. 257.—83. TURNER.—*St Thomas's Hosp. Rep.*, London, 1876, vii. 199.—84. WELCH, W. H. "Venous Thrombosis as a Complication of Cardiac Disease," *Festschrift in Honour of A. Jacobi*, 1900, p. 463, New York.—85. WILKS, Sir S. *Lancet*, 1886, i. 7.

Mitral Incompetence

Definition.—A diseased condition of some of the structures constituting the mitral valve, or a defect at the left auriculo-ventricular orifice, preventing its normal closure during the systole of the left ventricle, and occasioning a backward flow of a portion of the output into the left auricle.

History.—The structural alterations involved in mitral regurgitation were known to Morgagni and his followers, but the disease was not recognised clinically until a considerably later date. It has been pointed out in the previous section that Laennec failed to apprehend the facts of mitral obstruction; upon the subject of regurgitation he is also most obscure, even in the later editions of his work, such as that so well known through the medium of Forbes's translation (22). The clinical study of mitral incompetence, in fact, does not commence until the appearance of Hope's work, in which the diagnosis for the first time began to assume a reasonable degree of probability. Since his epoch the gradual accumulation of clinical and pathological data has proceeded with but little intermission. The most important contributions will be referred to in the sequel.

Etiology.—Mitral incompetence may be the result of alterations in the cusps or in their tendinous cords, brought about by various forms of endocarditis, but chronic degenerative processes without endocarditis may cause sclerosis. Traumatic influences may bring about rupture of one of the cusps or some of the tendinous cords. Regurgitation may also be the result of myocardial changes leading to faulty closure of the valve. Any factors, whether functional or structural, which interfere with the nutrition of the heart may thus allow escape. The muscular tissue may be weakened by pyrexia, anaemia, inanition, myocarditis, or degeneration. Severe muscular stress is, moreover, sufficient to bring about such a degree of strain as to permit reflux (*vide* p. 242). The incompetence in such cases is, as a general rule, the result of interference with the ring or sphincter surrounding the orifice, whereby its normal systolic reduction is lessened, as Sir D. MacAlister has clearly shewn. It is possible that dilatation of the left ventricle, without any definite change in the orifice, may cause faulty adaptation of the cusps by means of a lack of proportion between the size of the cavity and the length of the tendinous cords, but this is not so thoroughly established. In all cases of the kind the real explanation of the dilatation, whether of the sphincter or of the ventricle, is to be found in a loss of the function of tonicity. This property of the cardiac muscle was originally demonstrated by Dr. Gaskell, and the bearing of his physiological researches upon practical medicine has been emphasised by Dr. J. Mackenzie (53).

The left auriculo-ventricular orifice differs from the right in not being provided with such an easy natural means of permitting escape. There

is, in other words, no physiological safety-valve action. This subject, originally observed from the anatomical point of view by Hunter, was thoroughly investigated by Adams, who shewed that what is absolutely essential for the maintenance of the systemic would be positively injurious to the pulmonic circuit. These views were extended at a later period by King, and the real explanation of the matter has been furnished in more recent times by Drs. G. A. Gibson (27), Keith (41), and J. Mackenzie (53).

As regards the incidence of mitral incompetence, it may be added that during the sixteen years 1893-1908 the admissions to the wards of the Royal Infirmary, Edinburgh, included 897 cases among men and 689 among women.

Morbid Anatomy.—A. The lesions may be primarily confined to the cusps or their immediate attachments. There are several varieties of these changes. (i.) *In the Chronic Stages of Endocarditis.*—The curtains of the mitral valve are thickened and comparatively rigid ; the neighbouring endocardium is also denser and more opaque than the normal, especially in the portion extending from the great anterior flap of the mitral valve to the base of the aortic semilunar valves. Many of the chordae tendineae, together with their columnae carneae, are thickened and shortened ; there are often adhesions between the curtains, the cords, and the columns, as well as between these and the endocardium of the wall of the ventricle. In some instances the chordae tendineae, especially the finer cords which are inserted near the free border of the curtain, are lengthened instead of shortened ; probably this is due to yielding under the pressure of the blood upon the under surface of the mitral flap, so that the edge of the latter is inverted towards the auricle during the systole of the ventricle. Whether the chordae be shortened or lengthened, the result is an imperfect apposition of the curtains at the time of ventricular contraction. The endocardium lining the left auricle is also thicker than normal, especially at the ring bounding the auriculo-ventricular aperture. From this ring extends a whitish or milky patch of the fibrously transformed endocardium into the auricle above and the ventricle below. Such thickening may involve the structures subjacent to the endocardium, and tend to narrow the orifice, though the signs may be entirely those of mitral insufficiency and not those of obstruction. Duroziez (20) says, that if the orifice be large enough to admit the passage of the thumb the signs will be those of insufficiency, and not of obstruction. Much, however, depends on the condition of the internal surface ; if this be smooth, as in many cases it is, there will be signs of mitral insufficiency only ; if rough, there may be those of obstruction in addition.

The thickening of the endocardium is due to fibrous proliferation of the original inflammatory exudation, a process of development of connective tissue extending into surrounding structures. Repeated attacks of endocarditis affecting the already diseased tissue cause further thickenings and retractions ; the thick fibroid material compresses the blood-

vessels, and tends to induce degeneration. Fatty metamorphosis is not often observed, but calcareous change frequently, even in young children. The calcified portion of the valve structure may act as a mechanical irritant producing inflammatory or necrosing changes in the tissues adjacent. A fragment of the calcareous or necrosed material may become detached and form an embolus. A change of the firm fibrous material into cartilage has been found, but it is rare.

(ii.) *In Verrucose Endocarditis.*—A form of chronic endocarditis is sometimes observed in which there are small, firm, warty outgrowths from the surface ; these are fibrous proliferations of the endocardium, usually attached by a broad base but sometimes pedunculated. They are covered by endothelium to which fibrin does not adhere ; the sclerous changes of rheumatic endocarditis are not associated with them. They have been most frequently observed in cases of chorea ; Lancereaux has found them also in alcoholism and in malaria. Sometimes in newly-born infants small spherical outgrowths are observed on the free border of the mitral ; they are probably, according to Luschka and Parrot, haematomas, due to rupture of blood-vessels situated under the most superficial layer of the endocardium ; usually they disappear in the first few months of life, but in some cases they may initiate the warty excrescences above described. It has been thought probable that in some cases of chorea, determined by sudden fright, similar ruptures of intravalvular vessels with subsequent fibrous warty transformations occur.

(iii.) *In the Chronic Forms of Infective Endocarditis.*—The valve curtains, the cords and columns, or the endocardium of the ventricle may shew the lesions of ulcerative or infective endocarditis, the tissues in the affected areas being destroyed by necrosis. Usually the ulcerated surfaces are covered by large vegetations. These changes in a large majority of the cases of infective endocarditis—about three-fourths of the total—are found on valves previously diseased. In all such cases some of the forms of pathogenetic micro-organisms are to be discovered. It is to be borne in mind, therefore, that on the chronic morbid products at the mitral orifice a destructive disease, which may have no relation with rheumatism, may be engrafted. In a minority of the cases the necrosing changes are slow ; there is evidence that the process may be arrested in some areas, cicatricial tissue covering the portions shewing loss of substance (*vide* Vol. I. p. 905).

(iv.) *In Rupture of the Mitral Valve.*—The valve curtains, cords, or columns may be ruptured. It is improbable that such an accident can occur from strain where the structures have been previously healthy. Post-mortem evidences of the rupture of a tendinous cord are not infrequent ; an occurrence which has sometimes changed fairly compensated mitral inadequacy into a hopeless disablement. In the majority of cases such rupture is due to ulcerative changes. In the case of a curtain of the valve there may be first aneurysmal pouching, and secondly perforation. A vegetation on the curtain, if it induce softening of the

endocardial surface, brings about a yielding under the blood-pressure within the ventricle, and a pouch is formed which projects into the left auricle; further pressure may cause rupture, when of course the valve is no longer competent. Rupture of the cusps or cords may be caused by direct violence. Such cases usually present the signs of obstruction as well as incompetence. A striking instance occurred within recent years in the Royal Infirmary, Edinburgh, from the kick of a horse.

B. The lesions may be entirely confined to the muscular ring surrounding the mitral orifice, or to the walls of the heart. Here again the changes present several varieties. (i.) *In Dilatation of the Left Ventricle.*—There may be considerable dilatation of the ventricle, and yet the mitral curtains be quite competent to close the aperture. In many cases, however, when there is no disease of the structures constituting the valve, the cavity is so greatly dilated that it is demonstrably impossible that the aperture between auricle and ventricle could be adequately closed during the ventricular systole. Amongst the post-mortem associations of the latter condition are the following : (*a*) There may be disease at the aortic orifice causing obstruction or regurgitation, or, as very frequently is the case, the combined lesion is present. The ventricle has become hypertrophied on account of the abnormal pressure to which it has been subjected, and dilated from loss of the normal tone of the muscle; the dilatation progresses continuously until the mitral curtains are no longer capable of closing the enlarged auriculo-ventricular orifice. (*b*) The signs of chronic renal disease may be found. In some cases there is great hypertrophy of the muscular wall of the ventricle; in others dilatation, even at early periods of the disease, preponderates over hypertrophy. Microscopical investigation has shewn that the causes for the changes in the cavity and the walls of the left ventricle are complex. The obstruction to the general arterial circulation due to the thickening of the arterioles in various situations causes abnormal intraventricular pressure during systole and thus there is a mechanical cause of dilatation; but the muscle of the ventricle also suffers from the process of disease. The morbid changes in the ventricular wall have been described by Bard and Philippe, as well as Lancereaux, as an excessive proliferation of the connective tissue; by Debove and Letulle, Huchard, Loomis, and Martin, as a special quasi-inflammatory affection of the smaller branches of the coronary arteries—endarteritis and periarteritis; and by H. G. Sutton as a fibrosis extending to the general connective tissue, but starting from the arterioles and capillaries—arterio-capillary fibrosis. The muscular fibres are altered, the transverse striae are obscured, some fibres are atrophied and encroached upon by the fibroid tissue, others are hypertrophied. Similar changes are sometimes noted in the walls of the ventricle in persons at and after middle age, when there are no signs of chronic Bright's disease. (*c*) As a sequence to inflammation of the pericardium, the pericardial surfaces being found adherent. An excess of fibrous tissue not only extends amongst the muscular bundles and fibres, but also compresses the blood-vessels; this is especially seen after

general rheumatic disease of the heart in children; the left ventricle may be extremely hypertrophied and dilated so that the mitral valve is incompetent, and yet there may be no sign of endocarditis affecting the structures of the valve. Dilatation of the left ventricle to the extent of mitral incompetence is also observed occasionally after rheumatic fever in childhood, with no evidence of pericarditis or endocarditis. (*d*) In syphilitic affections of the ventricle the muscular fibrillae have probably been weakened by myocarditis. In rare cases small gummas have been found in the wall of the ventricle; in others bands of fibroid material, probably the sequels of syphilitic endarteritis, and obliterations of the vessels, have been seen. (*e*) In Graves' disease, and other kinds of long-continued morbid acceleration of the heart's contractions, such as tachycardia. In some such cases the left ventricle has been found so hypertrophied and dilated that the mitral curtains were incompetent; it must be remembered, however, that in many of the fatal cases of these diseases the ventricular cavity had not been dilated, and the muscle of the heart was quite normal. Whether the dilatation of the left ventricle should be regarded as a sequel of the disturbance of the nervous and muscular mechanism of the heart, or as the result of toxic agencies, must at present be left uncertain.

(ii.) *In Degenerations or Transformations of the Structures of the Left Ventricle.*—In a large number of instances various forms of degeneration of the heart are associated with dilatation of the left ventricle; and the mitral regurgitation, which is a feature of their history, is thus explained. In a minority there is no such dilatation. The various pathological changes and degenerations of the myocardium, fatty, fibrous, and so forth, are described elsewhere (*vide* p. 105).

(iii.) In some cases in which there has been strong evidence of mitral regurgitation during life the heart has been found on post-mortem examination to present perfectly normal appearances. The pathology of such cases will be considered later (p. 385).

In mitral insufficiency from all causes the *left ventricle* is dilated and its muscular walls hypertrophied. The dilatation and hypertrophy proceed hand in hand, and both are the direct and salutary results of the regurgitation through the mitral orifice. As Davies pointed out, the process whereby, in sequence to mitral insufficiency, the cavity of the left ventricle becomes enlarged and the muscular tissue hypertrophied should not be considered morbid. The enlargement may be in just such a degree that the amount lost to the aorta by the leakage into the auricle is compensated; and the increased driving power of the ventricle is precisely regulated to deliver the normal supply to the great artery.

In mitral insufficiency the *left auricle* is dilated and hypertrophied, and the endocardium lining it is thicker and more opaque than normal. In some chronic cases the muscle of the auricle wastes, and is replaced by fibrous tissue. The pulmonary veins may also be much dilated. Occasionally in chronic cases globular fibrinous coagula are found adhering to the lining membrane, and projecting from between the fleshy

columns and trabeculae into the cavity of the ventricle or the auricle. These thrombi are firm and dense in their external portions, and often soft and fluid in their interior ; cysts thus formed may rupture or become detached, and their fragments may cause embolism of systemic arteries. In some cases the coagula undergo fibrous and calcareous transformations.

The *right auricle and ventricle* in cases of mitral insufficiency are also found dilated and hypertrophied. Hypertrophy is found to preponderate in the earlier stages, dilatation in the later. The wall of the ventricle is in some cases found thick and leathery, in others thin and flaccid. The tricuspid valve may be incompetent on account of dilatation of the ventricle or stretching of the orifice. The globular thrombi, described as sometimes visible in the left cavities, are much more commonly observed in the right. The detached coagula cause embolisms of branches of the pulmonary artery. The dilated condition of the right chambers of the heart is obviously associated with general venous engorgement. In the heart itself the coronary veins are turgid and dilated.

The *walls of the heart* are frequently found to have undergone chronic interstitial changes of a fibroid nature, in consequence of interference with blood supply. This condition, which was originally explained by Fagge, brings the results of chronic cyanosis upon the heart into line with its effects on other organs, and it completes the vicious circle.

The *pericardium* may shew signs of disease, recent or remote, and there is often transudation into the pericardial sac.

Morbid Anatomy of Other Organs in Mitral Insufficiency.—The *lungs* in cases in which there has been long-continued mitral regurgitation are found engorged with dark blood, and their fibrous tissues abnormally dense. The lung is tough ; the capillaries of the alveoli have become dilated and varicose, their walls thickened. Patches shewing the signs of bronchopneumonia may be scattered throughout the toughened lung. Blood escaping into the surrounding connective tissue produces brown pigmentation (brown induration of the lungs) ; it may transude into the alveoli, causing the tinged sputa and haemoptysis observed in some cases. The lining membrane of the bronchi often shews extreme engorgement, and blood exudes from the surface. The blood-tinged sputa, therefore, may be derived from the lung capillaries or from the bronchial mucous membrane. The lower lobes, or the more dependent portions of the lung in chronic cases, become engorged, dense, and often oedematous. In many cases there are multiple pulmonary lesions, with evidence that these arose at different dates. Effusions into the pleurae may have caused collapse of various portions of the lungs. The signs may indicate that local pulmonary infarctions have occurred in different areas at various dates. There may be the blood-clot and prominence of the pleural surface indicating a recent embolism of a branch of the pulmonary artery (pulmonary apoplexy) ; the sites of old infarctions may be indicated by pigmented indurations of portions of

the lung-tissue, with, perhaps, some depression of the pleural surface corresponding to the indurated portion.   In cases of comparatively recent embolism the corresponding area of the pleura may be covered with the yellowish exudation of pleurisy.   All pulmonary apoplexies, however, are not due to infarction.   The abnormal strain of the pulmonary artery may lead to degeneration of the vessel and dispose it to rupture (*vide* p. 799).   Old adhesions of the pleurae or of pleura and pericardium are often observed.   In many cases there is fluid effusion in the pleural cavities.   This is mostly from transudation, but exudation is by no means uncommon, for inflammatory changes are apt to supervene on the condition of chronic passive hyperaemia.

The *stomach* manifests greatly dilated veins ; its mucous surface shews much hyperaemia ; the venules are often varicose ; mucus, tough or fluid, is seen in abundance.   The *liver* is enlarged ; the intralobular capillaries are very greatly dilated and their walls thickenéd ; on section it shews the characteristic appearances of " nutmeg liver," the dark brownish-red stellate spots marking the centre of each lobule on the yellowish ground formed by the bile-stained liver-cells.   The bunch of greatly dilated capillaries in the centre of the lobule encroaches upon the hepatic cells and may cause atrophy or fatty change in them, some brown pigment granules being seen amongst them.   There may be a considerable increase in the amount of fibrous tissue.   The most marked signs of venous engorgement with increase of bulk of the liver are seen in cases in which tricuspid incompetence has followed mitral insufficiency.   It is to be remembered that the size of the liver in such cases may become greatly reduced soon after death, the organ being partially emptied of blood by gravitation.

The *spleen* shews hyperaemia ; the increased amount of fibrous tissue makes it much firmer than under normal conditions.   In some cases it shews infarctions, old or recent ; when recent, wedges of hard tissue with their bases at the circumference (that is, the capsule) are felt on manipulation.   Old infarctions are indicated by shallow depressions of the surface of the viscus.

The *intestines* shew venous engorgement.   In some cases embolisms of the small arteries supplying the intestinal wall have been found, with consequent necrosis of the bowel.   The veins of the mesentery are engorged.   The lymphatic glands within the abdomen are enlarged and congested.

The *kidneys* are abnormally firm from cyanotic induration ; the pyramids are especially engorged ; blood may exude from the glomeruli into the tubules.   In some cases the kidneys shew on section pale, wedge-shaped, recent infarctions, their base towards the cortex and their apex towards the hilum ; or deep depressions óf the surface, with cicatricial tissue visible on section, may indicate the situations of old embolisms. There may be much fibrosis in these kidneys.

The *peritoneal cavity* may be more or less filled with ascitic fluid.

The *membranes of the brain and spinal cord* may shew much venous

engorgement. Signs of embolism of the cerebral arteries are found in some cases.

The *subcutaneous tissue* generally, especially in the lower extremities, may be found infiltrated with dropsical fluid. In some cases patches of the superficial layer of the epidermis are raised in large bullae. In other chronic cases the fibrous elements of the skin are thickened—there is a brawny oedema.

Mechanism of Mitral Regurgitation.—In normal conditions of the structures, after the filling of the ventricular cavity from the auricle, the muscular wall of the ventricle immediately contracts ; the musculi papillares, according to Roy and Adami, do not begin their contraction until after an appreciable interval, then these muscles act with sudden energy, drawing down the mitral curtains and completely closing the auriculo-ventricular aperture, the apposed curtains presenting a convex surface in the auricle ; the energetic tug of the papillary muscles gradually ceases and they relax, whilst the muscle of the ventricular wall remains contracted. The contraction of the muscle of the ventricle has a direct effect upon the auriculo-ventricular aperture. Before the beginning of the systole of the ventricle this orifice is circular ; during the period of systole the contraction of the surrounding muscular fibres causes it to become narrower and of oval form (Ludwig and Hesse, and D. MacAlister). At the acme of systole the auriculo-ventricular orifice has an area not much more than half that which it presents in diastole. The shape of the papillary muscles is such that in the complete contraction of the ventricle they are accurately applied to each other in a manner clearly explained by Marc Sée.

The ventricular systole, therefore, consists in a series of co-ordinated rhythmic movements, dependent on the essential properties of the cardiac muscle analysed by Dr. Gaskell—rhythmicity, conductivity, excitability, contractility, and tonicity. There may be many causes of disturbance of the normal association and sequence of these actions, due to alterations in one or more of these essential properties, the result of which is insufficient closure of the mitral orifice and reflux into the left auricle occasioned by the ventricular systole. (*a*) There may be such structural disease in the curtains, cords, and attachments of the valve that due apposition is impossible. (*b*) The fibrous ring to which the flaps of the valve are attached at their circumference may be so much thickened that the muscles at the base of the heart are unable to compress it sufficiently to cause accurate closure by the curtains during ventricular systole. (*c*) The insufficient narrowing of the auriculo-ventricular aperture during systole may be due to no structural alteration of the ring, but to enfeeblement of the muscle of the ventricle. (*d*) The ventricle may be so greatly dilated, and with it the fibrous ring to which the mitral curtains are attached, that these latter fail to meet at their borders during the period of contraction of the ventricle. (*e*) The papillary muscles may be so enfeebled by disease that they fail to perform their function of approximating the valve curtains. These different disorders depend on alterations in

the functions of contractility and tonicity. How far changes in conductivity are operative cannot at present be definitely stated, but it is probable that modifications in the auriculo-ventricular conducting structures, described by Drs. Gaskell, Kent, His, Tawara, and Keith (42), may lead to disturbance of the association and sequence of the movements necessary for the proper closure of the orifice.

Consequences of Mitral Regurgitation.—It is probable that in cases in which very small amounts of blood are regurgitated into the auricle from the left ventricle the consequences are inappreciable. The mechanical results are directly proportioned to the amount of reflux. The immediate effects may be regarded as simultaneous upon the left auricle and the left ventricle. The auricle is distended in proportion to the force of the ventricle and the amount of fluid regurgitated. The auricular wall becomes dilated, and its muscle, subjected to abnormal stimulus, hypertrophied. The left ventricle, receiving during its diastolic expansion an abnormal quantity of blood from the dilated auricle, is subjected to unusual pressure ; the muscle yields and its cavity becomes enlarged. Such increase of capacity is a necessity if the normal supply to the aorta is maintained. In systole it is called upon for more work, in order to deliver an adequate amount into the aorta. Hypertrophy of its muscle ensues, and is a favourable condition. The effect of the regurgitant stream is manifested upon the right chambers of the heart. The current impelled by the right ventricle, which in normal condition should flow unimpeded through the pulmonary vessels, is met by the reflux current from the left ventricle. The capillaries of the lung, the branches and trunk of the pulmonary artery, and the right ventricle itself, are thus subjected to abnormal strain. The effects are hypertrophy of the muscle and dilatation of the cavity of the right ventricle. Hypertrophy of the right ventricle is essentially favourable, for the more vigorous action antagonising the back flow into the left auricle helps the delivery of an adequate supply to the aorta.

The Maintenance and the Failure of Compensation.—If the changes in the cavities and in the myocardium thus sketched out are nicely balanced, a condition of restored equilibrium ensues ; thus a stationary lesion of compensated mitral regurgitation may persist for long periods, the subject thereof not presenting any morbid signs or symptoms. An adverse change, however, may be effected by many causes : the dilating strain upon the left cavities may impair the muscular power of the left auricle and ventricle ; renewed disease of the endocardium may increase the degree of valvular imperfection ; intercurrent diseases may affect the structural integrity of the cardiac muscle, vessels, or nerves ; affections of the lungs may induce direct and mechanical as well as indirect and enfeebling difficulties. The result of any of these interferences is a break of compensation—a failure of the cardiac forces of circulation ; the supply to the aorta and thence to the tissues becomes inadequate, then the muscle of the left auricle and the ventricle becomes more and more enfeebled, their constituent structures degenerate, and their cavities contain more and

more residual blood. The force of the right ventricle now fails, and both right auricle and right ventricle become engorged with venous blood ; the systemic veins are dilated, and the tissues suffer from venous stasis. The hepatic veins (which are in such immediate relation with the inferior cava), being destitute of valves, are especially congested, and their engorgement becomes manifest in enlargement of the liver. As the distension of the right ventricle continues, the right auriculo-ventricular orifice may become so much dilated that the tricuspid valve becomes incompetent to close it ; then the pulsatile action of the right ventricle is communicated to the valveless hepatic veins, and thus to the liver, as well as to the veins of the neck, if the walls of these have been sufficiently dilated to render their valves incompetent. The interference with the general and the lymphatic circulations at varying stages of this period of failing compensation may induce dropsy.

Diagnosis. — The chief sign by which the diagnosis of the insufficiency of the mitral valve is to be made is a physical sign obtained by auscultation—a systolic murmur heard at the apex of the heart, or having a maximum intensity in this situation. It is an essential preliminary that the position and outline of the apex be determined by palpation and percussion. The abnormal sound is often in some degree musical, varying in different cases from a very low to a very high pitch ; in some it may resemble the sound of a whispered "who," in others a musical note of varying pitch and quality, and in no inconsiderable number a shrill whistle. In many it has the sound as of a puff of steam. A characteristic to be especially noted is that it fades off gradually, and does not come to a sudden, abrupt stop. The murmur begins with, or soon after, the contraction of the ventricles ; this may be determined, at the time that auscultation is practised, by the observer placing his finger over a point where the apex-beat is to be felt ; or, if this be impracticable, over one of the carotid arteries in the neck. The bruit may be very short, ceasing at an early portion of the systole, or may be prolonged throughout nearly the whole of the systolic contraction, ceasing just before the second sound. It may wholly replace the first sound, or the dull sound of valvular tension may be heard to precede it, when it "tails off" from the first sound. The murmur may be of very slight intensity, and may be localised at the exact apex, or it may be audible over the whole precordia with maximum intensity at the apex. In some cases it is audible from the apex in a line which extends into the left axilla, and then it often has another area of audibility at the back between the spine and the angle of the left scapula. In other cases the conduction is to the left border of the sternum above the ensiform cartilage, and the cartilages and the interspaces as far as the second left costal cartilage. In some cases the explanation of this conduction of the systolic murmur seems to be afforded by the fact that the disease was chiefly confined to the anterior flap of the mitral valve. It is probable that conduction towards the axilla and the back may indicate an implication of the posterior flap in the disease.

It has been thought by Naunyn, Balfour (2), and Rosenstein that mitral regurgitation may be evidenced by a systolic murmur in the second left intercostal space, not quite close to the sternum but about two centimetres to the left of it; the murmur being due to vibrations communicated by the reflux current to the left auricular appendix. Many considerations seem to render this view untenable. The left auricular appendix, as has been pointed out by Drs. Russell and Bramwell, does not approach the surface at the spot where the murmur is audible; in many necropsies it has not been visible on an anterior view of the heart; when seen it is at least an inch and a quarter to the left of the left border of the sternum, and is for the most part on the posterior aspect of the heart. The whole subject has been reviewed very fully by Dr. Gibson (28). It is more probable that the vibrations of the reflux current, if transmitted to the auricle, would be audible at the back. Duroziez (19) has used this argument to explain the audibility of the murmur of mitral regurgitation at the back :—"L'oreillette gauche placée en arrière contre la colonne vertébrale transmet en arrière le souffle formé à la mitrale." It is most probable that, when the murmur of mitral regurgitation is audible in the second left interspace, it is by means of vibrations communicated to the great anterior flap of the mitral valve, or to the morbid structures in contiguity therewith.

The chief practical difficulty in the diagnosis is that of discriminating a murmur due to mitral insufficiency from one to be ascribed to the influence of the movements of the heart upon the portions of lung in front of it and around it. Cardio - pulmonary murmurs have been described by many observers (Potain (63), Gerhardt, Nixon, Prince, Morton, and Sanders).

The first sound of the heart to the right of the apex and over much of the area occupied by the right ventricle is often observed to be rough under conditions in which cardiac disease has no part. Such rough sounds have been referred to many causes which it seems unnecessary to discuss. As a rule they are readily to be distinguished from murmurs due to mitral insufficiency, because they are not heard at the exact apex or over the situations mentioned as those to which a mitral regurgitant murmur is conducted.

In some cases, however, the difficulties are greater. In order to make the distinction clear, certain steps should be methodically taken. First, *the relation of the murmur to the movements of respiration* should be observed. The cardio-pulmonary murmur is usually much influenced by the respiratory movements; for the most part it is intensified both during expiration and inspiration, especially during the latter; but it often becomes inaudible at the end of an expiration. If, therefore, rhythmical crescendo and diminuendo in the sound of the murmur are heard during the respiratory acts, it is probable, though not certain, that the murmur has its cause in the lung outside the heart.

The *position of audibility of the murmur* must be carefully noted. Cardio-pulmonary murmurs are not heard at the exact apex of the left

ventricle, but over a small area at the level of the apex to the right and to the left. Instead of corresponding exactly to the centre of the outline of the apex of the left ventricle, as does that of mitral insufficiency from organic causes, these murmurs have their maximum from a quarter of an inch to an inch and a quarter away from the point of apex-beat. Above the exact apex there is a doubtful zone, where a precise diagnosis cannot readily be made ; but if the systolic murmur has its site of maximum audibility exactly over the apex, it must be ascribed to intracardiac causes.

The rhythm of the murmur must be determined. A cardio-pulmonary murmur does not take the place of the first sound. The valvular sound is heard, and the murmur is observed to occur subsequently, after an appreciable interval, and to cease before the second sound ; it is manifested during a portion only of the ventricular contraction, and is meso-systolic.

In the next place, *auscultation should be practised in various positions of the patient*. A cardio-pulmonary murmur, as a general rule, is very evident when the patient is recumbent, diminishing in intensity and even disappearing when the sitting or erect position is assumed. In a minority of cases this rule is reversed. It has been shewn by Cuffer that though the bruits which have their causes outside the heart are in the greatest degree modified by changes of position, yet systolic apical murmurs, due to organic mitral disease, are sometimes similarly influenced. Potain says that if the change from the dorsal decubitus to the sitting position causes the complete or almost complete disappearance of the murmur, it can be confidently ascribed to extra-cardiac causes ; the same may be said when a murmur well marked in the erect position disappears on recumbency. On the other hand, it is not true that every murmur which is uninfluenced by changes of position is necessarily organic.

Potain (63) has adduced a great amount of evidence to shew that the cardio-pulmonary murmur is caused by an aspiration of some of the alveoli of the lung produced by the cardiac movements. When the heart is distended in diastole certain portions of the adjoining lung are compressed against the thoracic wall, and the air is squeezed out of them. When the systolic recession ensues the comparatively airless tongue of pulmonary tissue quickly becomes inflated, provided always the muscular contraction is accomplished rapidly.

Estimation of the Degree of Mitral Insufficiency.—When the amount of reflux into the left auricle at each systole is very small, there may be no physical sign to indicate the existence of any lesion other than the systolic murmur having the characters and areas of audibility already described. In the cases in which the amount is sufficient to disturb the normal physical conditions within the chambers of the heart, there are signs which indicate, in greater or less degree, the amount as well as the existence of imperfection. In the attempt to make this estimation, in the first instance the second sound of the heart should be carefully

observed. If, in any case in which a murmur indicating mitral regurgita-
tion is manifest, the second sound, as heard in the second left intercostal
space or the second and third left intercostal spaces, is noted to be
of a sharp, loud, metallic, or tympanitic character, or by its loudness
("accentuation") to contrast with the second sound heard in the course
of the aorta and great vessels of the neck, as well as in the positions
below the third interspace as far as the heart's apex, it must be concluded
that the regurgitant stream, antagonised by the adequate force of con-
traction of the right ventricle, causes abnormal pressure in the pulmonary
artery and the vessels of its circuit. This sign, as Skoda pointed out,
indicates a compensated mitral insufficiency; when the right ventricle
becomes feeble or the tricuspid valve inadequate, the accentuation of
the pulmonary second sound is not so distinctly heard. The observa-
tion of an accentuated pulmonic second sound, with no sign of
pulmonary embarrassment, no abnormality discoverable by ausculta-
tion except the murmur of regurgitation through the mitral orifice,
and no physical signs of dilatation of the muscular chambers of the
heart, will indicate a moderate and not an extreme degree of mitral
incompetence.

Any deviation of the ventricles and auricles from the normal should
be noted and considered. The left ventricle should be investigated by
palpation and percussion. In cases of mitral regurgitation, the apex
may be felt to lift the finger of the observer considerably below the
normal fifth interspace, and in a greater or less extent to the left; so
that it may overpass the vertical mid-thoracic line. The forcible heaving
or thrusting movements of the ventricle constitute a measure of the
degree of hypertrophy of the muscle. In young subjects the ribs and
cartilages corresponding to the area occupied by the ventricles may be
prominent. It is very rarely that a systolic thrill is to be felt over the
apex. The rhythm of a thrill must be carefully noted—one felt near
the apex is nearly always presystolic, and pathognomonic of mitral
stenosis. Determination of the outline of the left ventricle by per-
cussion adds to the information obtained, and indicates the shape and
position of the apex, when these are not perceptible on palpation. The
line of dulness or deficient resonance on percussion, indicating the outline
of the left ventricle, may be found to extend to the left of the mammil-
lary or mid-thoracic line, even as far as the axilla at the level of the
seventh rib, and thence in a line inclining upwards to the level of the
second left intercostal space. The upper limit of deficient resonance has
been found by Dr. Goodhart above the second rib.

At post-mortem examinations, even when there is clear evidence of
much hypertrophy and dilatation of the left ventricle, the latter is
generally observed only as a mere margin to the left of the right ventricle
on an anterior view of the heart; the left auricle is often invisible on
inspection of the front, and only discovered on turning over the heart.
It must be remembered, however, that the conditions during life differ
from those observed after death; the heart-muscle contracts in rigor

mortis ; nevertheless, it is no doubt correct that the left auricle and left ventricle occupy but a small portion of the left border of the cardiac dulness.

In cases in which a notable accentuation of the pulmonic second sound and the physical signs of enlargement of the left ventricle are manifested with no evident deviation of the right chambers from the normal, it may be inferred that, though regurgitation through the mitral orifice may be considerable, the lesion is compensated by augmented force of the right ventricle.

For the due estimation of the extent of the lesion the right cavities must be carefully explored. Palpation may detect a forcible heaving of the right ventricle to the left of the ensiform cartilage. Percussion may indicate dulness extending too far to the right in various degrees in different cases. The dulness sometimes extends to two and a half inches from the median line ; it delimits the right border of the right auricle.

In some cases the right border of dulness does not meet the line which indicates the upper border of the liver at a right angle ; but, from one to two inches above the liver, a sloping line of dulness extends from the auricular border to meet the liver dulness an inch or an inch and a half to the right of the sternum. There is a wedge-shaped area of comparative dulness to the right of the vertical line which indicates the limit of the right auricle. It is possible that this is due to a distension or dilatation of the venae cavae as they open into the auricle ; it is only observed in cases of great dilatation of the right cavities. The upper limit of dulness may reach as high as the lower border of the second right costal cartilage. The extent of the dulness from right to left may be determined by percussion over the first part of the sternum in a horizontal direction ; this line crosses the sternum to the second interspace on the left side. Such a line of dulness over the sternum at the level of the second rib still indicates the right auricle, which may even encroach on the second interspace on the left side. The remainder of the upper limit of dulness is due to the right ventricle and the pulmonary artery.

The evidence of the outline of the heart obtained by percussion must not be accepted without the due estimation of causes of fallacy. Distension of the stomach with air will cause a tilting of the ventricles to a higher plane, and a dislocation towards the right of the right chambers. The size of the right auricle and ventricle fluctuates with the varying turgescence of the liver. Such distension may be protracted and due to a lasting and permanent or temporary and evanescent morbid congestion ; for it is well known that the liver presents great variations in bulk even during brief periods of time. A dilatation of the blood-vessels within the abdomen (that is, in the splanchnic area) also may reduce the bulk of the right auricle and ventricle when there is no obvious change in the volume of the liver. Another cause for reduction in the observed size of the right cavities is expansion of the lungs. In such cases there

are two causes for the recession of the area of dulness indicating
the bulk of the heart; namely, the inflated air-cells of the tongues
of pulmonary tissue overlapping the heart which give rise to a clearer
note on percussion, and the augmented volume of blood circulating in
the pulmonary blood-vessels which reduces the content of the heart-
chambers.

When in a case manifesting the murmur of mitral regurgitation it is
found that the right chambers are persistently dilated, and especially
if physical signs of tricuspid incompetence be present, it must be inferred
that the degree of valvular imperfection is great and the muscle of the
heart gravely approaching failure.

The investigation of the bulk of the liver is also important as a
guide to the estimation of the degree of valvular imperfection in a case
manifesting the murmur of mitral insufficiency. When there are signs
of dilatation of the right chambers of the heart, and the liver is felt as
a distinct mass below the right costal margin, it must be inferred that
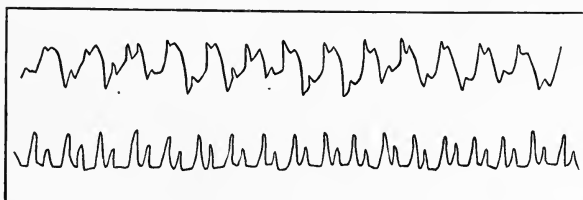
FIG. 43.—Cardiogram and sphygmogram from a case of free mitral regurgitation.

the mitral valve is gravely incompetent—the imperfection is still
greater if the liver be felt to pulsate (*vide* p. 327).

Important evidence is afforded by the observation of the characters
of *the pulse*. If in a case manifesting the systolic apex-murmur and other
physical signs of mitral insufficiency, the hand of the observer applied to
the precordia is sensible of a forcible ventricular contraction, whilst the
radial and other arterial pulses are found to be small and weak, the
inference is legitimate that much of the volume of blood which should
have been delivered into the aorta is lost by regurgitation into the
auricle. The pulse of a slight mitral regurgitation scarcely differs from
the normal : when the lesion is considerable the volume is small and
the pressure may be low. The sphygmograph often shews marked
dicrotism even when the evidence of impaired pressure is not obvious
to the finger. Not infrequently, even when compensation is main-
tained, the pulse presents marked fluctuation of the base line, which
shews that the normal correlation between circulation and respiration is
disturbed.

Irregularity of the pulse is not a characteristic of mitral insufficiency
unless cardiac failure is present.

The *cardiogram* in a case in which there is free mitral regurgitation

sometimes presents special features. There is a pronounced dip or notch
in the upper part of the tracing, giving the summit a forked appear-
ance. It is also worthy of note that the relative durations of the systolic
and diastolic periods, as expressed in the cardiogram, are altered, the
diastolic period being relatively shortened. In compensated mitral regur-
gitation in many cases neither cardiogram nor sphygmogram presents any
notable deviations from the normal.
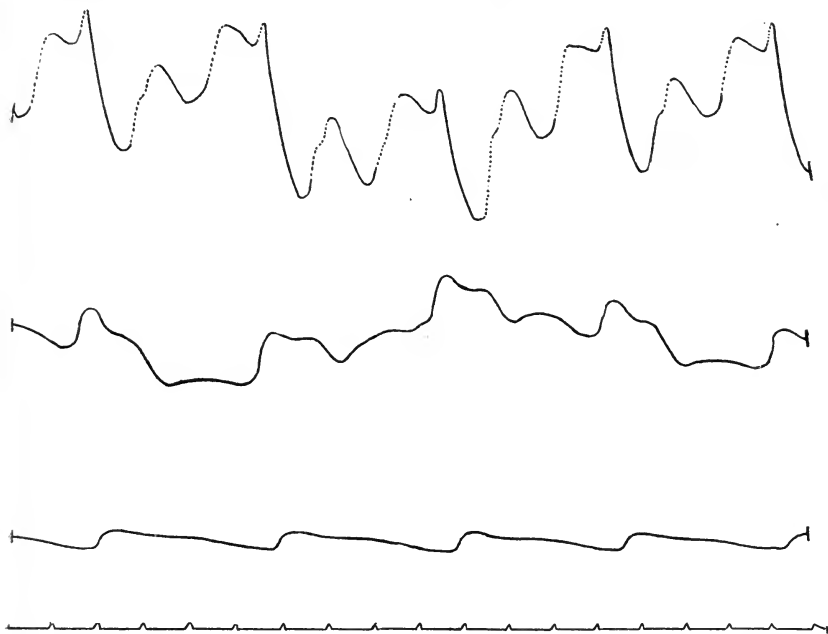
Fɪɢ. 44.—Tracing from cervical veins, apex-beat, and radial pulse, with time-record in fifths of
seconds below, from an instance of pure mitral incompetence.

Clinical Groups of Cases of Mitral Insufficiency.—*Group I. Mitral
Insufficiency the Result of Rheumatic Endocarditis.*—It will be convenient
to consider this group in two divisions : the first of children, the second
of adults.

In children of twelve years of age and under, who have suffered either
from a well-marked attack of rheumatic fever, or from repeated attacks,
or from one attack with subsequent subacute manifestations, it is in the
highest degree probable that the signs of insufficiency of the mitral valve
will be observed. Such insufficiency is, according to Dr. Cheadle, nearly
always due to sclerotic alterations at the left auriculo-ventricular orifice
and to a retraction of the valve curtains, cords, and muscular columns
which are the results of the progressive morbid changes of rheumatic
endocarditis. These, however, are not the only changes in such cases.

Pericarditis frequently coexists; the layers of the pericardium become united, oftentimes throughout their whole extent, by adhesions. The muscle of the heart is inflamed and infiltrated, and rapidly becomes extremely hypertrophied. The whole heart participates in the rheumatic inflammation; there is general carditis, as described by Sturges, the result of which, though life may be prolonged for months and years, is a crippling of the heart while such life lasts. In the course of development of this severe heart disease subcutaneous rheumatic nodules are frequently observed, as Sir T. Barlow and Dr. Cheadle have shewn. Such severe general rheumatic heart disease is rarely met with in children under six years of age; it is most common between the ages of six and twelve years. As a general rule, of the children admitted into hospital for acute or subacute rheumatism 50 or 60 per cent are discharged with valvular disease, the most frequent form of which is mitral insufficiency. This, however, by no means represents the full effect of rheumatic endocarditis as a cause of the valvular imperfection, for the cases discharged without evidence of such disease are often found, after the lapse of months, or perhaps years, during which no rheumatic phenomena have been manifested, to present undoubted evidence of mitral regurgitation, very commonly attended by obstruction. The progress of the changes in rheumatic endocarditis is slow and is not necessarily betrayed by symptoms.

In a considerable number of cases of mitral insufficiency in children no evidence of rheumatism is to be obtained. For instance, in a series of 118 cases of mitral regurgitation, Sansom (73) found an absence of any evidence of rheumatic association in 40. In 8 of these there appeared to be a definite relation in sequence to scarlatina, in 6 to measles, and in 3 to scarlatina and measles. In 13 cases there was no evidence of any antecedent disease to account for the valvular imperfection. Post-mortem evidence shewed that the morbid changes in these were identical with those observed in cases known to be rheumatic.

In the cases in which there is no evidence of rheumatism the child may be brought under notice for a disorder of nutrition—especially wasting and anaemia—or for a disturbance of respiration, such as cough and dyspnoea, the results or concomitants of the heart disease; or for an affection of the nervous system, such as chorea, epilepsy, or hemiplegia. In some of them there is cerebral embolism, the plug being derived from the diseased endocardium. Not infrequently the valvular disease is discovered by accident. No notable discomfort may be caused by the movements of the child in play or on running upstairs; and Henoch says that in many cases the disease is first discovered by the mother observing the violent motion of the heart when she strips the child to give the bath. The evidence points to the conclusion that a form of endocarditis which has the essential characters of the rheumatic may occur in infancy and childhood without any other manifestations of rheumatism. Endocarditis, then, may occur as a solitary expression of the rheumatic disease, as Dr. A. E.

Garrod states. (*Vide* also art. "Acute Rheumatism of Childhood," Vol. II. Part I. p. 650.)

The symptoms observed in childhood during the progress of uncompensated mitral inadequacy vary very greatly. The age of the child has some influence in regard to these. As a general rule, the signs in infants and very young children are chiefly those of inanition,—emaciation, anaemia, and deformity of the thorax. There are in many cases frequently-recurring attacks of bronchitis or bronchopneumonia, cough being a prominent symptom. In children after the age of four years symptoms more directly indicating disorder of circulation become manifest: bleeding at the nose may be cited as one of these. Difficulty of breathing becomes apparent, and in some cases most distressing orthopnoea. Precordial pain and discomfort are severe symptoms in some cases, and these may be associated with lumbar pain. Palpitation may be a distressing symptom. Dropsy is by no means uncommon, but it rarely follows the gradually ascending course usual in the adult; the oedema is either more general, or more variable in the sites of its manifestation. In cases with oedema or ascites, albuminuria is a frequent complication: this may be transient and due to venous congestion, but in the majority of cases it is dependent on the coexistence of disease of the kidneys, and is a sign of dangerous import. In the later stages of the disease vomiting and diarrhoea may be observed as most serious indications; haematemesis occurs in some cases. A marked anaemia, occasional vomiting, restlessness followed by apathy, and partial unconsciousness are symptoms which in many cases mark the weeks or days preceding the close of life.

In the form of mitral insufficiency attended by general carditis the prognosis is bad. The pericardial adhesions and the consequent hypertrophy and dilatation of the whole heart are a constant menace, and prevent satisfactory treatment. On the other hand, an uncomplicated mitral insufficiency in childhood often has a favourable issue; and the results of treatment even when the severe symptoms of threatened failure are present are often very satisfactory. Henoch considers that children recover from rheumatic endocarditis better than adults, and that in them the valve is more likely to regain its structural integrity.

In the *treatment* of mitral insufficiency in the child when compensation fails and the symptoms are those of progressive cardiac enfeeblement —the condition being one of chronic disease uncomplicated by acute rheumatism—the following are the chief points to be observed :—(i.) Rest in the recumbent or in the semi-recumbent position, with the shoulders supported, must be maintained as much as possible. (ii.) Precordial pain and discomfort or difficulties of respiration call for the application of warmth to the chest by warm moist flannels, spongiopiline, or poultices. On some occasions a digitalis poultice may with advantage be substituted for the ordinary linseed-meal poultice : this is made by boiling two ounces of digitalis leaves in a pint of water for ten minutes, about two ounces of linseed meal being gradually added

until the proper consistence for a poultice is attained. The mass is to be spread upon suitable material and applied in the usual way. (iii.) Means for inducing good general nutrition are of the first importance. A child with mitral incompetence is often intensely anaemic. Cod-liver oil, by itself or in an emulsion, or in combination with some of the iron preparations, is very beneficial. In some cases small doses of arsenic, with tincture of nux vomica or liquor strychninae, succeed better than iron. In not a few a plan of supplementary alimentation by nutritive enemas turns the scale towards amendment. One of the best of such enemas is made by shaking together in a bottle one egg, an ounce of hot milk, and an ounce of cod-liver oil, and administering very slowly through a large soft rubber male catheter, with a funnel attached and held at a sufficiently high level, or by an india-rubber enema tube. The administration should be twice or three times daily. (iv.) Cardiac tonics are to be prescribed with judgment. In some cases rest, carefully regulated diet, and the tonic methods just mentioned suffice, and all agents which directly influence the cardiac rhythm are unnecessary or even injurious. Of all cardiac tonics digitalis and strophanthus are of the greatest value; they are especially so when dyspnoea is a marked feature. Digitalis may be given in the form of the tincture in doses of from one to five minims, or the infusion, ten minims to one dram, or the leaves in powder, one-fourth of a grain to half a grain, repeated three times a day. There is some difference of opinion whether the administration should be continuously for long periods or in larger doses with omissions for several days. In some instances digitalis is not well borne, and in children this intolerance is usually shewn by the occurrence of vomiting; it should be omitted whenever vomiting appears. In cases in which digitalis adminis-tered by the mouth seems to be inert, rapid improvement may follow the hypodermic injection of digitalin, $\frac{1}{150}$ to $\frac{1}{100}$ of a grain for a child of from six to twelve years of age. In any case such hypodermic injection should not be repeated for at least forty-eight hours. Strophanthus is of even greater value. It may be administered in the form of Sir T. R. Fraser's tincture (1885 B.P.), of which from one-half to three minims may be used three times a day. In certain cases the hypodermic injection of strophanthin may be resorted to, the dose being from $\frac{1}{200}$ to $\frac{1}{100}$ of a grain for a child of the age just stated. In many cases strychnine in doses of one-half to one minim of the liquor may be employed. As an alternative to preparations of digitalis, strophanthus or strychnine, caffeine citrate dis-solved in water, or in the ordinary saline mixture, in doses of from one to three grains three times a day, may be given. The administration should not be continuous, but for a period of four to six days, with similar periods of suspension; for all cardiac tonics, though tending at first to increase the excretion of urine, by their prolonged action often tend to diminish it. In cases where as a consequence of mitral regurgitation the right cavities of the heart are much dilated—especially when the tricuspid valve is rendered incompetent—digitalis and other cardiac tonics may be power-less for good. Their inefficiency is readily to be explained, for it must

be remembered that their action is on both ventricles, and that they augment the force of the right ventricle as well as that of the left: now unless the drugs employed increase the tone of the muscular ring surrounding the orifice, increased action of the right ventricle means so much the more reflux into the general venous system and further disasters. In many cases where there is such distension of the right cavities (an occurrence which may supervene as an acute phase in a case of chronic mitral insufficiency), the relief of venous pressure by leeching is a most valuable auxiliary to treatment. One or two leeches may be applied to the precordia, and the leeching may be repeated on several occasions at intervals of two or three days. Exceptionally, half a dozen leeches may be applied at the first. Sansom (73) shews that digitalis, which has been powerless for good before the application of leeches, has proved of great service thereafter. Sometimes it is absolutely necessary to go a step farther and relieve the patient by venesection.

Dropsy, in cases of chronic mitral insufficiency in the child, may be transient, and yield to the medicinal treatment already sketched out ; or it may become a far more serious symptom. There may be general anasarca, whilst pronounced ascites and hydrothorax may rapidly take place. In a considerable proportion of cases desquamative nephritis is manifested in the course of the mitral disease. In the treatment of such cases, sponging of the skin with hot water made alkaline with sodium carbonate, the child being afterwards wrapped in a hot blanket, is often a more practicable and efficient measure than the administration of a hot-air bath or a vapour bath. Purgatives, as compound jalap powder, are essential ; at first calomel may advantageously be administered therewith. Saline diuretics are to be combined with digitalis and decoction of broom. The removal of all traces of dropsy in the child is sometimes rapid. In some cases medicinal means fail. As a rule, punctures of the skin of the lower extremities in the treatment of dropsy in the child are not to be recommended ; there is a danger that restless movements may cause chafing and irritation. If there be ascites, paracentesis abdominis should be performed, either with the aspirator or Southey's tubes ; sometimes rapid convalescence follows this operation. Sedatives and medicines to procure sleep must be used with caution, but in many cases they are indispensable.

In mitral insufficiency, the result of rheumatic endocarditis in the adult, we find associations differing from those in the case of children. In adult life the occurrence of general carditis and the implication of the pericardium, endocardium, and myocardium in the rheumatic disease are much less common. In this sense the disease is less formidable than in the child. On the other hand, repeated storms of endocarditis in the adult increase the sclerosis at the mitral orifice and the imperfection of the curtains, cords, and columns ; the thickened fibrous structures tend also in progressive degrees to undergo degeneration and calcareous transformation. The already diseased endocardium may be attacked by pathogenetic microorganisms ; the endocarditis may be malignant. This is especially probable

in women after parturition, and in both sexes when there are dangers of septicaemia : but the disease may arise insidiously without traceable infection.   The causes of over-strain, both physical and mental, which affect the adult, adversely modify the conditions.   Emotions interfere with the rhythm of the heart and tend to disturb the compensation. Severe physical efforts may rupture curtains or cords already diseased. Diseases of various forms may alter the nutrition of the heart-muscle. There are probably many forms of disease affecting the coronary arteries and their branches within the heart ; arteritis and periarteritis occur in many forms of infectious disease, and notably in syphilis.   Arteriosclerosis involves the coronary arterioles (especially in chronic Bright's disease), and the larger branches in the later periods of adult life are especially affected.   The result of such morbid alterations of the walls of the arteries is an impairment of the force of the cardiac muscle with sub-sequent degenerations.   Intercurrent diseases of the lungs, again, may rudely interrupt a compensation hitherto satisfactory.   In some cases causes of inflammatory irritations are imported from without.   In others infarctions or so-called pulmonary apoplexies are both consequences and causes of cardiac failure.   Any considerable interference with the function of the lungs imposes a direct obstacle to the work of the right ventricle. It is the energy of the right ventricle that, by impelling an abnormally large volume of blood through the pulmonary vessels, and thus antagonis-ing in the left auricle the regurgitant stream from the left ventricle, is the effective agency of compensation.

The symptoms in the adult of a failure of the compensatory conditions in cases of insufficiency of the mitral valve are briefly, and in an approxi-mative way chronologically : *difficulty of breathing*, especially upon effort, but also paroxysmally at a later stage ; *cough*, with physical signs of oedema of the bases of the lungs, and often of localised consolida-tions ; *dropsy*, gradually extending from the more dependent portions of the body ; and *scanty urine*, of dark colour and high specific gravity. From all such symptoms and from the epiphenomena of embolism and infarction, pulmonary and systemic, there may be recovery.   When the limits of possible restoration of the powers of compensation are reached, the picture is one of suffering and sadness.   The recumbent position is intolerable, the lower limbs are persistently oedematous and their integuments indurated ; the countenance wears the hue of combined sallowness and lividity, the expression is one of anxiety and of a restless craving for sleep, alternating with a feeble, helpless wandering of mind ; there is abdominal discomfort from a large and tender liver ; the arterial pulse becomes feebler and nearly imperceptible, and by slow degrees, with occasional awakenings to the reality of suffering and distress, life be-comes extinct.

In the *treatment* of a case of mitral insufficiency in the adult, when compensation is failing, rest is of the first importance.   For a practitioner to prescribe cardiac tonics in a routine fashion for patients who manifest morbid heart symptoms is a dangerous error.   Rest, careful dieting, and

judicious purgation may turn the scale towards recovery, even when dropsy, and signs of much venous engorgement of the viscera, have supervened, as in a case described by Vivian Poore. In a large proportion of cases, however, the difficulties are not to be thus surmounted, and recourse must be had to drugs, whose influence is especially upon the forces of circulation ; of these digitalis is the chief. Digitalis may be administered in the form of the powdered leaves, the infusion, or the tincture. One grain of the powdered leaves is equivalent to one-third of an ounce of the infusion and to eight minims of the tincture. The leaves may be administered in doses of half a grain to a grain and a half three times a day in wafer, cachet, or pill, alone or combined with other agents such as mercury, iron, or aloes, or other aperients. The infusion may be given in doses of a quarter of an ounce to half an ounce, or the tincture from 5 to 20 minims.

In many cases the daily administration of digitalis can be continued for long periods, for a considerable number of months at any rate ; but great care must be taken to ascertain that the patient is perfectly tolerant of the drug, and at the outset of this treatment the effects must be noted daily : the treatment should not be continued for more than three or four days without the control of a skilled observer. Digitalis has a complex action. It possibly has a tonic effect upon the pneumo-gastric nerve, whereby its power of moderating and slowing the heart's movements is increased ; but further, it increases the energy of the myocardium by a direct effect upon the muscular mechanism of the heart. It also augments the contractility of the walls of the arteries by an influence upon the muscular coat. The good effects of digitalis are manifested by its so lengthening the diastolic pause that the ventricles become more completely filled, and deliver ampler blood-waves into the general arterial system. The arteries, when moderately contracted, do not impede the blood-flow ; in fact, a larger amount of blood traverses the circulation in a given time. The ventricles emptying themselves more completely, the previously dilated heart diminishes in volume. The beneficial effect of digitalis is also shewn in the production of diuresis. Neither the heightened arterial pressure nor the augmented urinary outflow produced by the drug is, however, by any means constant. Variations of arterial blood-pressure under the action of digitalis have been noted by many observers to be quite independent of the slowing effect upon the heart ; indeed the diuretic results are confined almost entirely to those cases that manifest oedema. It seems probable that the fluid absorbed from the lymph-spaces, drawn within the capillaries on account of the augmented rapidity of the circulation, and carried to the renal capillaries, so stimulates the kidneys as to provoke diuresis. When there is no effused lymph to be absorbed, diuresis does not result ; in fact, the urinary outflow in some cases diminishes even to arrest, and there may be haematuria. Digitalis is contra-indicated when nausea, vomiting, and diarrhoea form part of the symptoms, and when the pulsations of the heart are rendered inordinately infrequent. It is now fully

recognised that the drug produces bradycardia by its effects on the conductivity of the auriculo-ventricular bundle. Dr. J. Mackenzie (54) has given clear evidence on this subject. When the administration of comparatively small doses is continued too long there may be chronic poisoning ; the signs are pallor of the surface, coldness, and, sometimes, attacks of faintness. Certain effects of digitalis may persist long after cessation of its administration. Abnormal retardation of the heart's contractions has been noted by Duroziez for as long as a month after cessation (20). The practical rule should be that average doses of the preparations of digitalis, repeated at intervals of four hours, should not be continued, in the earlier stages of treatment, for more than three days ; then the drug should be suspended for a like period. It is only when a patient manifests a perfect tolerance that the protracted administration should be permitted.

The employment of digitalin is preferred by many physicians, especially by the French. It is to be remembered that the various digitalins vary greatly in strength ; that of Nativelle has about fifteen times the strength of the digitalin of Homolle, weights being equal. Potain (64) prescribes for a case of cardiac failure with dropsy one milligram of Nativelle's crystallised digitalin. This may be administered in one dose, or, if tolerance be doubtful, it may be divided into four or five doses given in as many days. After the administration there is often profuse diuresis. There should be no readministration for many days— the interval may be from ten days to three weeks ; renewed acceleration of the pulse is to be taken as an indication for repetition of the treatment. Digitalin may be administered hypodermically. When satisfactory effects have not followed administration by the mouth, excellent results sometimes follow the hypodermic injection of digitalin in doses of $\frac{1}{100}$ to $\frac{1}{50}$ of a grain.

Strophanthus may be administered in the place of digitalis, caffeine, or theobromine. It is given in the form of the original tincture recommended by Sir Thomas Fraser and embodied in the British Pharmacopoeia of 1885. In every way this preparation is better than that described in the last edition. The dose is from 2 to 10 minims along with 5 minims of tincture of capsicum or 30 of compound tincture of cardamoms. The dose may be repeated every four hours ; the same care in watching effects and suspending the administration at intervals of a few days should be used as in the case of digitalis. Strophanthin in doses of $\frac{1}{100}$ to $\frac{1}{50}$ of a grain may be given. The action of strophanthus upon the heart by the way of the vagus and through the local muscular mechanism resembles that of digitalis ; but it seems to stimulate the contraction of the papillary muscles to a far greater degree than that of the ventricular wall ; Roy and Adami have shewn that on repeating the dose so that the pronounced toxic action of the drug is manifested, the papillary muscles become notably weakened, and their power of contraction may even be annulled. Fraser concludes that strophanthus acts upon the heart more forcibly than digitalis, but on the calibre of the arteries

much less. It has often a very favourable effect upon difficulties of breathing, and, used with care, is an efficient and useful substitute for digitalis, but it is not without its dangers. Its protracted use may cause dyspepsia with diarrhoea and wasting, and there are some probabilities that it may lead to sudden death in the course of its administration for heart disease. It cannot be doubted that the protracted injudicious administration of digitalis and strophanthus—especially in those who absorb these drugs without skilled medical supervision—has often been productive of dangerous and fatal results.

In some instances of mitral insufficiency, the consequence of rheumatic disease, the treatment by digitalis and strophanthus entirely fails ; there seems to be no good effect upon the left ventricle, the right cavities continue to dilate, dropsy increases, and the drugs in combination with ordinary diuretics fail to increase the outflow of urine.

Caffeine or its citrate may be given in doses of from 3 to 5 grains every four hours, but, as in the case of digitalis, it is better that it should not be used for more than three days continuously. It has little action in retarding the pulse or in causing contraction of the arteries ; it has therefore little effect on arterial pressure. Its diuretic influence is decided, and it stimulates the renal epithelium to the excretion of solids. The diuretic effect is found to persist after the suspension of the drug. In some cases this result is coexistent with good and rapid recovery from all distressing symptoms ; but diuresis may occur and persist, and yet the result be unfavourable. The drug very rarely induces insomnia ; it has more frequently been observed that by lessening dyspnoea it promotes sleep. In some subjects, however, it produces agitation, headache, vomiting, purging, and sleeplessness. The combination of digitalis and caffeine may act more favourably than either drug alone, as Sir J. Barr has shewn.

Theobromine, in the form of the sodio-salicylate (diuretin), may be substituted for caffeine. It is to be administered in doses of 15 grains six times in the twenty-four hours. It is freely soluble in water. It has a stronger diuretic action than caffeine, and does not cause nervous agitation and sleeplessness. The diuretic effect is manifested between the second and sixth days of its administration. Uropherin and theocin act in the same way as diuretin ; the relative utility of these drugs is a matter of personal idiosyncrasy on the part of the patient.

In cases in which the right chambers of the heart are much distended *blood-letting* is indicated. A bleeding from the arm to the extent of 6 or 8 ounces coincidently with the administration of cardiac tonics, or subsequently, will often turn the scale towards recovery.

When there is enlargement of the liver and dropsy of the peritoneum, the combination known as Baillie's or Guy's Pill is most valuable. The most useful form for ordinary cases consists in one grain of each of the following :—blue pill, digitalis leaves, squill bulb, and extract of hyoscyamus. One pill should be given three times a day after meals, and along with this combination gentle saline aperients are advantageous. When oedema threatens to be troublesome one of the

most useful combinations is 5 grains of citrate of caffeine, 20 grains of citrate of potassium, and half an ounce of infusion of digitalis. While the salts of the alkaloid and alkali act on the renal epithelium and stimulate glandular activity, the digitalis exerts its influence over the heart and the vessels in such a way as to increase filtration.

The means already indicated, together with the administration of such *purgatives* as produce watery evacuations—one or two purgative doses of calomel are often of service in the early stage of treatment—may suffice to remove all traces of dropsy and to restore compensation. The employment of saline aperients is without doubt of the highest importance, and the method introduced by Prof. Matthew Hay of administering them in concentrated form is most useful.

In other cases when the dropsy does not disappear the *mechanical removal of the effused fluid* may be necessary. Incisions by a lancet or punctures by a needle may be made into the skin of the lower extremities, the limbs being wrapped in flannels or other absorbent material to take up the fluid which copiously drains away ; or the fine trocars and cannulas known as Southey's tubes may be used. In either case the skin should be previously sponged with an antiseptic solution. The former plan is to be preferred in the case of a delirious or very restless patient ; the latter when the patient is tranquil enough to allow the fluid to flow gradually through the fine flexible tubes into the receptacle underneath the bed for many hours. The trocar should be inserted very obliquely beneath the skin ; the opening of the cannula should be at the extremity (and not at the sides), and the flexible exit tube in the portions nearest the inserted cannula should be fixed to the skin of the legs by strips of adhesive plaster ; it should also be arranged so that it does not kink and obstruct the flow. It is best, when the anasarca is considerable, that two cannulas with tubes attached be inserted into each lower extremity. When ascites exists, the fluid within the abdomen may be drawn off by the slow process of draining with Southey's tubes, or by the more rapid process of paracentesis abdominis. When ascites coexists with general anasarca it may be a question whether draining the subcutaneous tissue or tapping the abdominal cavity should be first performed. When the abdomen is not much distended the former should be practised first, for after the draining the intra-abdominal effusion may become absorbed. When the ascites is considerable paracentesis abdominis should take the precedence. Effusions within the pleural cavity should be withdrawn at once.

In many cases of mitral incompetence the arterial pressure is found to be too high, and the principle long ago advocated by Balfour of dilating the arterioles is imperatively demanded. The employment of some of the preparations of ammonia, especially the aromatic spirit, with strophanthus or digitalis is, in this way, of eminent service. It is often necessary, however, to go a step farther, and to use the nitrites. The solution of nitroglycerin, containing 1 per cent in alcohol, may be given in doses of 1 or 2 minims along with tincture of digitalis or tincture of

strophanthus. The nitrite of sodium combines very usefully with many of the alkaline combinations employed in cases of cardiac failure.

Agents for producing sleep or calming nervous agitation are of high importance in the treatment of the failing heart of mitral insufficiency. Amongst modern *hypnotics* which are of use in the management of heart disease trional, in 20 grain doses, and veronal, in 5 to 10 grain doses, are pre-eminent. Without producing any depressing effects they induce calm and restful slumber, from which the patient awakes not merely refreshed but rested. In some cases chloralamide has been useful, and it is always a harmless hypnotic. It may be given in doses of from 20 to 50 grains. A combination of 20 or 30 grains of chloralamide with 30 minims of dilute hydrobromic acid, a dram of syrup of orange flowers, and an ounce of pure water, administered at bedtime, is useful. Another harmless agent is urethane (ethyl carbamate), which is freely soluble in water, the solution having a saline but by no means unpleasant taste. In doses of 15 to 20 grains at bedtime it induces a calm, natural sleep often lasting in a case of severe cardiac failure for more than five hours, the patient being manifestly refreshed on waking. Paraldehyde is a little stronger as a hypnotic. It may be administered in doses of from 60 to 120 minims in diluted syrup or in almond mixture, or in capsules (each containing 40 minims); it has a powerful and unpleasant taste.

In a considerable number of cases manifesting distressful symptoms of dyspnoea and insomnia no agent succeeds so well as morphine, as was pointed out by Sir Clifford Allbutt. By far the best way of administering it in cases of cardiac disease is by hypodermic injection. The solution of the acetate or the hydrochloride, or the solution of morphine and atropine may be used. The first dose should be small—one-sixth or one-fourth of a grain—but this may be increased subsequently to half a grain. Care should be taken that the administration shall not become habitual.

In regard to *diet*, the aliments in the condition of failing compensation in mitral insufficiency should be very simple. Milk is the best of all foods, but in some cases is hardly tolerated. In the gastric crisis accompanying the failing heart there is often a complete disinclination for food. Then peptonised milk or milk gruel may be swallowed in sipping fashion, the patient being never permitted to take a distinct meal or a particle of solid food. In such cases great benefit may follow the administration of peptonised enemas, or the cod-liver oil, milk, and egg enema already mentioned in the treatment of children (p. 396). Brandy, if given at all, should be in teaspoonful doses with milk and wine only. Sherry, marsala, or tokay may be given in jellies. At the subsidence of the crisis, as soon as milk can be well borne, an all-milk dietary, especially if there be dropsy, should be prescribed until convalescence. In cases of great oedema the amount of fluid permitted must be restricted, and it is advisable to withdraw sodium chloride as much as possible from the diet.

The diet and hygiene during the stage of comparative convalescence will be considered with the third group of cases.

Group II. Mitral Regurgitation in Chorea.—In the majority of cases

of chorea a systolic murmur, having the characters which indicate regurgitation through the mitral orifice, is manifested at some period of the disease or throughout its whole course. In a large section of such cases the signs and symptoms are such as to leave no room for doubt that the imperfection of the valve has been caused by rheumatic endocarditis. In many instances of chorea there has been antecedent rheumatism ; the proportion varying according to different statistics—Dr. Pye-Smith gives 30 per cent, Sir Andrew Clark 31 per cent, and Dr. A. E. Garrod 32 per cent. There is a consensus of opinion that about one-fourth of all the subjects of chorea are or have been rheumatic. In many also of those who have personally shewn no evidence of rheumatism there has been a family tendency to the disease. The doctrine has been formulated by Roger that chorea is in all instances a rheumatic affection ; other observers (Stephen Mackenzie, Barlow, and Cheadle) have estimated that in from 45 to 75 per cent of the cases there are sufficient evidences of rheumatic tendency ; it may be concluded, therefore, that in the majority of cases chorea is a phase of rheumatism. It must be allowed that in many of the cases the diagnosis of rheumatism (reposing as it necessarily does on the statements of unskilled observers, with whom as a matter of common experience almost every painful affection is rheumatic) can be by no means precise. If causes of fallacy be excluded we may perhaps take it as a fair working hypothesis that about half the total cases of chorea are rheumatic, and that the endocardial murmurs manifested in these patients are due to structural disease of the valves, the result of the rheumatic form of endocarditis. In this section of the cases the mitral incompetency which is the concomitant of the disease is to be estimated and treated—when any failure of compensation renders such treatment necessary—according to the rules already laid down. The therapeutics of chorea are discussed elsewhere.

Nearly all observers, however, are agreed that some cases of chorea are doubtfully rheumatic. It is well known that shock or terror may be the precursor of chorea : such a cause may operate in a case undoubtedly rheumatic, but there are many cases of chorea in which a sudden and violent emotion preceded the attack in a person who did not shew any sign of rheumatism, or any proclivity thereto. Sir Stephen Mackenzie's statistics shewed rheumatism and fright to be nearly equal, numerically, as antecedents of chorea. Observers, according to Dr. Garrod, are generally agreed that emotional and mental disturbances have a large share in the immediate causation of the disease. As Sir W. Gowers remarks, " The only immediate cause of chorea that can be traced with any frequency is emotion, usually fright, rarely mental distress." The heart affection, in Sir Stephen Mackenzie's statistics of cases of chorea, was associated with rheumatism in 50 per cent ; whilst in 35 per cent no such association was recorded. In non-rheumatic chorea the symptoms and signs of mitral insufficiency sometimes differ from those in the rheumatic cases. In some of these, careful examination for many days may detect no evidence of valvular disease ; then a soft and slightly pronounced systolic murmur,

localised at the position of the heart's apex, may become audible. There is no accentuation of the pulmonic second sound ; the ventricles do not become dilated ; yet the murmur, having its original characters, persists for several years. At later periods it may become completely inaudible. The late Sir Andrew Clark held that the murmurs of mitral regurgitation so frequently observed in cases of chorea disappear, in the great majority of cases, within eight or nine years of the attack. These clinical features greatly differ from those of mitral insufficiency due to rheumatic endo-carditis. The evidence of morbid anatomy completes the distinction. In cases of fatal chorea wherein a soft apical systolic murmur has been observed during life, the left auriculo-ventricular orifice on its auricular aspect has been found studded and fringed with small, firm outgrowths having the signs of papilliform elevations of the endocardium. These outgrowths are firm to the touch, and are not detached by rubbing with the finger. The endocardium is smooth over them. They do not begin, as in rheumatic endocarditis, with a change in the endothelium and an attachment to the roughened surface of fibrinous caps, but they are firm outgrowths shewing fibrous hyperplasia. Their formation is not followed by the sclerotic changes, the widely-spread fibrous proliferation, the retractions of valve curtains, cords, and columns so frequent in rheu-matic endocarditis. On the other hand, they interfere but little with the closure of the orifice in systole, and in process of time, the endocardium remaining quite smooth, they cease to have any pathological significance whatever. It is possible that they may be the immediate results of a sudden over-strain and rupture of the terminal arterioles distributed to the valve structures. The immediate symptoms induced by terror or by any sudden mental shock shew a blanching of the surface of the body, a contraction of the arterioles, a stimulation or over-action of the sympathetic nerve mechanism. The effect on the heart at first would seem to be arrested action, afterwards palpitation. In the case of the delicate arterioles of the endocardium of the valves the result might well be ruptures ;—minute haemorrhages, followed by thickenings analogous to those observed after the experimental production of over-strain in animals (Roy and Adami).

In cases of chorea in which there is no evidence of failure of com-pensation, but only a systolic murmur at the apex to indicate some incompleteness of the closure of the mitral orifice during the ventricular systole, all treatment by cardiac tonics, or by means specially directed to the valvular imperfection, is unnecessary, and probably mischievous. The therapeutic methods adopted should be those for calming the tumult of the nervous system and for ministering to a healthy nutrition.

Group III. Mitral Insufficiency the Result of Dilatation of the Left Ventricle.—This group must of necessity be subdivided. In some cases the dilatation of the ventricular wall is from mechanical causes. This can be traced in the case of disease of the aortic valves, which has caused obstruction, regurgitation, or the combined lesions. For long periods no murmur is heard at the apex, but later the systolic bruit of mitral

regurgitation becomes audible, and the case which formerly presented no such signs, begins to manifest the venous congestion, the rising dropsy, and the forms of dyspnoea of mitral disease. A similar sequence may be observed in chronic Bright's disease with arteriosclerosis. The left ventricle may for long periods shew signs of hypertrophy ; then signs of dilatation are manifested more or less rapidly ; later the murmur and the signs of mitral insufficiency are observed. The ventricle has become hypertrophied or dilated and hypertrophied, from the resistance in the aorta and the peripheral vessels on account of the thickening and contraction of the smaller arteries. The intraventricular over-strain continuing and increasing — because of the augmenting arteriole-obstruction — the left ventricle yields to such an extent that the mitral curtains fail to close during ventricular systole.

In another set of cases there may be none of the ordinary signs of chronic Bright's disease, or of thickening of the walls of the systemic arteries ; and yet, in patients who have manifested no signs of rheumatism or of endocarditis, the physical signs shew dilatation of the left ventricle and finally mitral insufficiency. In some of these it is found after death that there have been arteritis and periarteritis in the vessels of the heart itself ; in others atheroma of the coronary artery of the left ventricle and tracts of degeneration, molecular, fibrous or fatty, corresponding to the area supplied by the branches of the artery. In another subsection, these patients being usually obese and often alcoholic, there is fatty infiltration amongst the cardiac muscular fibres, and the left ventricle yields because of the imperfection of its muscle (*vide* p. 113). In yet another subsection in this group the heart becomes dilated to the degree of incompetency of the mitral valve from a morbid affection of the nervous system. Probably the nervous influences disposing to dilatation of the left ventricle have been too much overlooked. In several cases the complete signs of dilated ventricle and mitral insufficiency have come on in the course of Graves' disease ; these will be considered hereafter (p. 415).

It is obvious from these considerations that dilatation of the left ventricle with mitral insufficiency, apart from structural disease of the valve, may be the result of various and complex morbid states. It must be remembered that these complex morbid conditions may coexist with structural disease the result of rheumatic endocarditis, which has already been discussed. In all such cases the ultimate cause of the ventricular dilatation lies in loss of myocardial tonicity. The subject was investigated experimentally by Dr. Gibson (27), and his results were illustrated by Dr. Keith (41), whilst Dr. J. Mackenzie (53) brought them into relation with Dr. Gaskell's researches.

These considerations must have their due weight in questions of *treatment*. In cases of arterial obstruction in the subjects of chronic Bright's disease, and often in patients after middle life, digitalis and all forms of cardiac tonics fail, or even do positive harm. In such cases good may result from the administration of arterial relaxants, and with these digitalis may often be associated. Balfour (3) considered that digitalis

cannot be safely given in cases of senile heart without a simultaneous unlocking of the arterioles. The cardiac tonic, therefore, should be combined with carbonate of ammonium, or with a nitrite, such as nitrite of ethyl (nitrous ether), nitrite of sodium, or nitroglycerin. In cases in which there is reason to suspect thickening of the walls of the arteries—in the general arterial system, or in the heart itself—a long course of the iodides is to be advised. Digitalis may be also administered for periods of two or three days at long intervals. Trinitrin should be prescribed if any sign of intolerance of the iodides be noticed ; or if these seem to be inefficacious, it may be administered in one-minim doses of the 1 per cent spirituous solution ; or in the form of tablets in which $\frac{1}{100}$ grain of nitroglycerin is combined with chocolate. A combination of amyl nitrite is in some cases a distinct advantage ; for example, nitroglycerin $\frac{1}{100}$ grain, amyl nitrite $\frac{1}{4}$ minim, menthol $\frac{1}{50}$ grain, capsicum $\frac{1}{100}$ grain, with chocolate to form a tablet (Pharmacopoeia of the Westminster Hospital).

When a case in this group shews signs of marked cardiac failure, such as severe dyspnoea and dropsy, complete rest in bed should be enjoined. Before the administration of any cardiac tonic it is well that purgatives be administered. A dose of calomel, three to five grains, is a good beginning ; or the patient, having abstained from liquids for some hours, may take two to four drams of sulphate of magnesium in hot water as recommended by Prof. Matthew Hay. A considerable watery discharge may rapidly reduce the oedema. The patient should be cautioned against rising from bed, or even assuming the sitting position during the relief of the bowels, lest syncope be thus induced. The trunk should be supported by pillows and the bed-pan used.

In cases in which dropsy is not extreme, *massage* may be of great advantage. The muscles of the extremities and of the thorax should be gently kneaded. Abdominal massage should be practised with caution ; to dilate the vessels within the splanchnic area may induce anaemia of the brain. Massage of the extremities aids the venous circulation, quickens the functions of the absorbents, and tends to bring about a more deliberate and efficient ventricular systole.

The manual treatment of Kellgren, described by Cyriax, is often of the greatest advantage, and leads, after recovery has proceeded in some degree, to the possibility of using various exercises.

In the grave conditions of failure of compensation it is best that the diet be exclusively milk, diluted with barley-water or peptonised. Small quantities should be swallowed at a time. Milk is a notable diuretic, and in the dropsical stages it should form the staple diet. All strong extracts of meat, which contain many products of retrograde metamorphoses, are to be forbidden ; but chicken or veal broth and jellies may be permitted in some cases. In the stages of recovery three to six pints of milk may be taken in the twenty-four hours.

When the patient begins to be able to take some walking exercise, and the probability of resuming ordinary avocations comes into con-

sideration, the question of limitation of the ingestion of fluids has to
be settled.   Oertel permits only 34 to 36 ounces of water, including that
contained in the solid food, per diem.   The best proportions of food are
said to be about 1 ounce of fat, $3\frac{1}{4}$ ounces of carbohydrates, and not
less than 5 ounces of proteins.   A cup of tea morning and evening,
about half a pint of claret, from $8\frac{1}{2}$ ounces to rather more than a pint
of water, and a little over 3 ounces of soup, should constitute, besides
that contained in the solids, all the fluid taken during each day.   The
solid diet should be rich in nitrogen—for example, bread 4 to 5 ounces,
meat or fish 6 to 7 ounces, with 5 ounces of chicken or game, one or two
eggs, a little salad, cheese, etc., and $3\frac{1}{2}$ to 7 ounces of fresh or cooked
fruit.   It is always necessary, when employing any methods in which
the amount of fluid is restricted, to watch over the urinary excretion ;
if the specific gravity should reach 1025, the quantity allowed must be
increased.   If this is not done the metabolic functions suffer, the arterioles
tighten, the arterial pressure rises, and cardiac embarrassment ensues.

As compensation is recovered, and during its maintenance, system-
atised muscular exercise is a valuable therapeutic means.   Stokes, in
1854, said that "the symptoms of debility of the heart are often re-
movable by a regulated course of gymnastics, or by pedestrian exercise
even in mountainous countries such as Switzerland or the Highlands of
Scotland or Ireland."   This opinion sounded the note of reaction against
the routine practice of a long series of years of keeping a patient who
presented any sign of heart disease in the most complete muscular repose
attainable.   Supposing that active disease be not going on in the cardiac
tissues, a "coddling" policy, whereby the heart-muscle is kept at a
minimum exercise of function, is contrary to sound physiology and good
practice.   Zander used gymnastics in the treatment of diseases of the
heart, and described the results, which appeared to be very favourable.
The Swedish system for the promotion of good physical development—
the chief exponent of which was Ling—became an important agency for
preventive as well as curative treatment ; the essentials being a forced action
of the voluntary muscles for given periods.   The order proposed by Ling
for these exercise movements was (i.) respiratory, (ii.) lower extremities,
(iii.) upper extremities, (iv.) abdomen, (v.) trunk, (vi.) movement of
lower extremities repeated, (vii.) respiratory movement repeated.   In the
Zander system mechanical appliances were used for the special exercising
of certain groups of muscles.   Oertel in 1884 extended the doctrine and
practice, and advocated, in a regulated and graduated manner, the pro-
motion of vigorous muscular effort in mountain-climbing.   The effort of
ascending a hill is much more potent for good than that of walking on
level ground.   There is an increased flow of venous blood to the right
side of the heart ; the lungs become more fully expanded, the channels
of the pulmonary circulation to the left auricle are more free, and the
volume of blood delivered to the arteries by the left ventricle is greater.
The perspiration causes a reduction in the volume of the fluid blood, and
a relative augmentation of the haemoglobin.   The lymphatics are stimu-

lated to their task of absorption. Many cautions, however, are necessary in the prosecution of this plan of treatment. If the efforts cause unduly rapid breathing, the patient should at once come to a rest and make deep inspirations. The plan is only good when, with the increased muscular effort, there is no considerable increase of the breathing-rate—the lungs must be adequately but not rapidly, imperfectly, and deceptively inflated. No effort must be sudden. It is the sudden over-strain, such as occurs in running to a railway station, that kills. Again, great caution must be exercised in sending cardiac patients to considerable altitudes. Dangerous and fatal symptoms have occurred even at moderate elevations above the sea-level.

The climbing of hills is not to every patient a possible method of treatment. Systematised gymnastic exercises exclude the necessity of hill-climbing. The exercises recommended by Schott are known as resistance gymnastics (Widerstandsgymnastik). The patient, loosely and lightly clothed, is instructed to breathe quietly, and to make certain movements which are gently resisted by a skilled attendant, who uses for this purpose the palms of the hands, without grasping or constricting the limbs. The movements made are (*a*) various flexions and extensions of the forearm and upper arm ; (*b*) movements of the lower extremities, the patient maintaining his position by resting his hand upon a chair ; (*c*) flexions, extensions, and rotations of the trunk upon the hips. A short interval is enjoined after each movement, during which the patient sits down ; the exertion should be only moderate in degree, and should not cause flushing or pallor, or quickened breathing. These resistance exercises are much inferior to passive and unresisted movements.

Much good often results from a course of systematic movements executed without the aid of any skilled attendant. These should be exercises (*a*) of the arms and upper thoracic muscles, (*b*) of the legs both in walking and with the body at rest, (*c*) flexions and extensions of the trunk ; thus movements are communicated to the abdominal viscera. No heavy weights, such as clubs or dumb-bells, should be used, and the muscles of one side of the body should not be exercised disproportionately to those of the other. Deep, slow inspiration should accompany each movement of elevation of any group of muscles, and expiration should occur with the opposite movement. The movements should stop if there is the least feeling of fatigue, or if the patient looks pale or livid in the smallest degree.

The following simple movements, recommended by Dr. G. A. Gibson, constitute the first and best for the purpose :—(1) Standing at "attention," with the hands on the hips, rise slowly on the toes while inspiring slowly and deeply, then slowly sink down upon the heels again with slow expiration. (2) Standing at "attention," with the hands on the hips, slowly move the head back as far as possible with deep inspiration, and afterwards bring it back gradually to the former position while breathing out slowly. (3) Standing at "attention," with the hands on the hips, rise up slowly on the toes with slow inspiration, bend the knees

and slowly sink down nearly to a squatting posture while slowly expiring; rise up again to the upright position on the toes with another deep inspiration and then slowly sink on to the heels with slow expiration. (4) Standing at "attention," with the arms hanging down, raise the arms slowly up, along with deep inspiration, until they are at right angles to the body, then let them slowly fall to the former position with deep expiration. (5) As in the last exercise, but the arms are to be raised high above the head during deep inspiration and then allowed slowly to fall with deep expiration. (6) Standing with the feet 18 ins. apart, and the hands on the hips, rotate the body slowly round in a circle, bending it to the right, then forwards, then to the left, then backwards, and lastly to the original position, all the time keeping the muscles of the calves and thighs rigid. This is to be repeated in the reverse order alternately.

The effect of exercise of the voluntary muscles is an accumulation of blood in their vessels of supply, and a corresponding derivation from congested areas—for example, from the right chambers of the heart and engorged veins. Sir Lauder Brunton says: "The vessels which supply the muscles of the body are capable of such extension that when fully dilated they will allow the arterial blood to pour through them alone nearly as quickly as it usually does through the vessels of the skin, intestines, and muscles together." The conditions, however, induced by muscular exertion are very complex. There are alternate contractions and relaxations, the former compressing the blood-vessels, the latter freeing these channels; concurrently there are increased activities of the absorbents and reflex nerve stimulations. In the movements of the trunk upon the lower extremities another set of factors comes into play. The alternate compressions and relaxations of the abdomen affect the blood-supply to the abdominal viscera. The tendency must be in the main to cause the vessels in the splanchnic area to dilate and so to co-operate with those of the muscles in relieving any turgescence of the right cavities of the heart.

The *use of baths and bathing* in the treatment of ill-compensated mitral insufficiency can only be attempted with caution. In past years there has no doubt been too great fear lest a patient presenting the signs of mitral regurgitation should suffer a chill; thus the ablutions have often been insufficient or injudicious. The use of cool or cold water has been pro- scribed, and possibly hot baths have been too freely indulged in. The effect of a hot bath is evident to ordinary experience—causing dilatation of the vessels of the skin it may induce cerebral anaemia with symptoms of faint- ness. On the other hand, the invigorating effect of the cold tub in those who can bear the shock, and of cool sponging in those who are more susceptible, are matters of common experience. Until recently sending patients to any health resort for a course of treatment formed no part of the therapeutics of heart disease. Beneke in 1859 and 1861, and Groedel in 1878, adduced evidence to shew that the baths of Nauheim, near Frankfurt, in Germany, were beneficial in increasing the force of the

heart and in restoring compensation in cases of valvular disease. Blanc (in 1886) recommended the course of treatment at Aix-les-Bains by douches (temperature about 90° F.), together with skilled massage ; and he cited 52 cases of mitral regurgitation in which this plan was pursued : in 15 of these all signs of disease disappeared, in 21 there was improvement, and in 16 the signs remained stationary. The chemical constitution of the water of Aix-les-Bains has probably but little to do with its therapeutic effect as used externally in these cases. Its chief value lies in its soft, unctuous quality, due mostly to the presence of organic matter (barégine), which, when at the agreeably warm temperature at which it is used, adapts it admirably for the douche-massage. The therapeutic conditions of the employment of the Nauheim waters are more complex. These come from hot springs (temperature 83° to 100° F.), and are charged with saline matters, chiefly chlorides of sodium and calcium, and free carbonic acid gas. In marked feebleness of heart, and generally in the earliest stages of treatment, the patient takes a saline bath from which the carbonic acid has been allowed to escape ; the duration of the bath is six to eight minutes, the temperature of the water being 95°. A rest of an hour is enjoined after each bath. The periods of immersion are increased during the course of treatment to twenty or thirty minutes, and the temperature is lowered by degrees to 85·5° F. The water used is allowed to retain its carbonic acid in varying degrees, as it is exposed for longer or shorter periods to the air, or used as the Strombad foaming with its full content of the gas. Baths specially intended for the treatment of cardiac weakness are also to be found at many spas, particularly at Royat and Bourbon-Lancy, whilst artificial imitations—with Sandow's tablets—are now available everywhere. The effects of the various agencies thus put in force have been studied experimentally by Prof. R. F. C. Leith and others. In regard to these effects, simple thermal baths at 90° F. or under commonly tend to reduce the pulse-rate by five or seven beats a minute. The effect of the addition of sodium chloride to the bath is slightly to emphasise the change in the pulse, and to make the bath more agreeable to the patient ; when the bath is charged with carbonic acid gas (Sandow's effervescing tablets being used) the pulse-rate may be further reduced, whilst the force of the heart's action may be increased ; the pleasantness and buoyancy of the bath are enhanced, and the patient experiences an agreeable sensation of warmth. The result of a bath at a temperature below body-heat is contraction of the cutaneous vessels of the area immersed—higher temperatures cause their relaxation ; the lymph-circulation is necessarily modified, the internal vascular conditions are changed, dilatations of the vessels occur in various regions, and probably there are some rhythmic alternations of dilatation and contraction. Furthermore, there are reflex effects upon the vasomotor and cardio-inhibitory centres. When the bath contains free carbonic acid gas the fine bubbles adhering to the skin protect the body from the colder surrounding water, and constantly impinging upon the surface stimulate the cutaneous nerve-endings.

The effects of the combined treatment by rest, baths, and muscular exercises as carried out at Nauheim are increased strength of the pulse with diminution of its abnormal frequency, decreased rate of respiration, together with fuller inspirations and greater ease and comfort in breathing, and diminution in the size of the dilated heart. There is sufficient testimony to shew that in a large number of cases there has been a great improvement in the subjective conditions. The evidence is not conclusive as to the reduction in size of the heart. From examination of a considerable number of outlines purporting to be those of the heart before and after the Nauheim treatment, the conclusion, urged by G. V. Poore, Sir William Broadbent, Prof. Leith, and Dr. Herschell, that they are the results of a fallacious plan of physical examination, and cannot be held to represent with any degree of accuracy the size and position of the heart, cannot be avoided. On the other hand, there is a very · high probability that in some cases the situation and shape of the heart have become changed, and the right chambers reduced in volume. Careful observations have shewn that the bulk of the heart may greatly change under varying conditions within very short periods of time. In the case of mitral disease, whilst the patient had been at rest, and when no special therapeutic means could be invoked as causes, Sansom (73) observed signs of very considerable variations in the bulk of the heart in less than twenty-four hours. Sir W. Broadbent said : "That a diminution in the volume of the heart may take place under the influence of saline baths and certain movements there can be no doubt, but such diminution is an occurrence which is perfectly familiar to all who are in the habit of noting the changes in the size of the heart under other methods of treatment or from various causes. In a heart dilated from over-exertion, for example, the apex-beat may often be felt to come in for half an inch towards the normal situation, when the patient is simply made to walk two or three times across a room." Not only the position of the apex, but also the outlines of precordial dulness, have been found to vary at intervals during the day. Heitler considers from his observations that there are rhythmic changes in the volume of the heart, the pulse remaining unaffected by these. All these considerations must have their due weight, and too much reliance must not be placed on the evidence derived from the ordinary means of physical examination as to the space occupied by the heart at a given time. Even the employment of the orthodiascope has not been attended by results which are at all definite. The concurrence of signs,—the evidence of rational as well as of physical diagnosis,—however, shews that a combination of judicious bath treatment and physical exercises may be a valuable agency for good in cases of mitral insufficiency with slight failure of compensation. It is a great mistake to send any patient with serious disturbance of equilibrium to any spa such as Nauheim.

One factor in the therapeutics of a health resort must not be overlooked. The change in surroundings must produce an effect upon

the higher attributes of the nervous system—the will, the emotions, and the intellect. It is no slight advantage for a patient to be taken away from the little worries of home to a place where, with clear sky and pure air, there are facilities for systematic self-management, a prescribed and regulated dietary, and the associated hope and faith inspired by the favourable experiences of others. Mental and emotional impressions can strongly influence the trophic nervous mechanism of the heart. It is true that there is a reverse to this picture. Patients are sometimes deceived by false hopes and fallacious arguments; persons, for example, the subjects of mitral insufficiency, well compensated and causing no adverse symptoms, have been persuaded by well-meaning but misguided friends that calcareous incrustations and fibrous thickenings about their heart-valves would by the operation of a certain "cure" disappear as crystals dissolve in water. Long and arduous journeys have been undertaken by those who were totally unfit to leave the comforts of their home, and there has followed a sad awakening from the delusive dream. These agencies are potent for good or for evil, and every case in which the use of them is contemplated must be carefully considered.

Group IV. The Alcoholic Heart.—One of the forms of myocardial weakness leading to incompetence of the mitral cusps is that produced by alcohol. In most instances the left side of the heart does not suffer alone, but the right side and the tricuspid orifice are also implicated. In a considerable number of cases another etiological element is probably at work in the shape of intermittent physical strain. There is undoubtedly in this condition a somewhat complex method of causation, as the alcohol certainly affects both nerve and muscle, and, whether in regard to heart or vessels, the effects produced are of complicated origin.

The morbid appearances found after death vary considerably according to the age of the patient and the extent to which he has abused the alcohol. There may be nothing more than slight dilatation from loss of tone with early granular changes; but, on the other hand, the heart may be found laden with adipose tissue and seared with newly-formed fibrous tissue spreading throughout the walls from the finer divisions of the coronary arteries. Such are certainly the pathological conditions most commonly seen in this country. In other countries, such as Germany, where a very large amount of beer is drunk, there is a very great tendency to cardiac hypertrophy. This condition has been particularly studied by Bollinger, who has found the average weight of the heart of men in Munich to be 370 grams for 61 kilograms of bodyweight. The symptoms belonging to the cardiac condition are breathlessness on exertion with some oedema of the dependent parts. There is frequently a trace of jaundice and sometimes a degree of cyanosis. The arterial walls and arterial pressure vary within wide limits according to the age and other concomitant conditions. There is frequently irregularity and tachycardia. A distinct venous pulsation of a ventricular type may even be seen in the neck. The apex-beat is not uncommonly displaced outwards, but it is sometimes so diffuse as to be difficult of detection

by inspection, and its position can only be determined by palpation. In general the cardiac dulness is increased, and there are commonly murmurs of escape in connexion with the mitral as well as the tricuspid orifice.   Catarrhal symptoms connected with the alimentary tract are common, and the liver is very often considerably enlarged in early examples.   In later conditions the liver may be reduced in size and the symptoms of cirrhosis may dominate the clinical picture.   In many instances there are the symptoms and signs of passive hyperaemia of the lungs, and albuminuria is frequently present.   It is scarcely necessary to add that insomnia, illusions, hallucinations, delusions, tremors, and weakness are often present.

The diagnosis of the condition is, as a rule, facilitated by the nervous symptoms which are present.   The prognosis must be formulated after careful consideration of the complex factors which have led to the condition ; undoubtedly it must be based to a considerable extent upon the duration and degree of alcoholic abuse, but inheritance, environment, and occupation must be duly considered, as well as the physical condition, particularly as regards the nervous system, which is present.   The treatment of the condition must be simply that of cardiac weakness in general, with special reference to the elimination of the poison.   Absolute rest and careful diet, with gentle massage gradually increased, will, as a general rule, produce great improvement.   Of drugs, strychnine is the most useful, and it may be combined with hydrobromic acid, whereby we have a stimulant as well as a sedative influence.   As soon as practicable, the various exercises which have been elsewhere recommended may be employed, and a visit to a spa is frequently of great utility in getting rid of the last traces of the affection.

Group V.   Mitral Insufficiency from Anaemia.—A systolic murmur over the apex of the heart is heard not infrequently in the subjects of the various forms of anaemia ; in some cases it is also audible at the back internally to the angle of the left scapula.   Balfour (2) fully described this cause of " curable mitral regurgitation," and every subsequent writer has concurred in his views.   The first question to determine is whether a bruit having such characters be due to causes operating externally to the heart itself.   Potain describes all the murmurs heard in the neighbourhood of the heart which are causally related with anaemia and chlorosis as cardio-pulmonary ; he finds that they do not begin with the systolic contraction of the ventricle as organic murmurs do, but are meso-systolic (occupying a portion only of the systole) ; that they are soft and superficial, greatly modified by the act of respiration ; that they are influenced by the attitude of the patient, so that they sometimes disappear when the recumbent is changed for the erect position ; and that they vary from day to day.   He considers that chlorosis tends to the production of cardio-pulmonary murmurs by influencing the nervous system, and so enhancing the cardiac excitability.   When in a case of anaemia a systolic murmur is heard at or near the situation of the apex, it is of importance (*a*) to determine by palpation the position of the apex-

beat and by percussion the outline of the left ventricle, and to consider the relation of the observed murmur to the area thus determined; (*b*) to analyse the various signs already noted which differentiate the cardio-pulmonary from the organic mitral murmur. A certain proportion may be found to answer to Potain's criteria of non-organic murmurs. There can be no doubt, however, that in most cases the apical murmur is due to veritable mitral regurgitation; first, because it has the site and charac-ters identical with those due to organic causes, and, secondly, because it may be followed by all the symptoms of failure of compensation in mitral insufficiency. Sansom (73) described an apical systolic murmur arising in a healthy woman after profuse uterine haemorrhage (from fibroids), followed by severe dyspnoea with abundant dropsy, and ending ulti-mately in complete recovery, with the disappearance of all the physical signs of disease.

From the well-known association of fatty degeneration of the muscular fibrillae of the heart with anaemia, it must be inferred that the mitral insufficiency is caused, the valvular apparatus being normal, by the resulting enfeeblement of the myocardium. The incompetence may be from impairment of the muscle of the ventricular wall or of the musculi papillares, or of both. Positive dilatation of the left ventricle has been described by some observers (Goodhart). In these cases the incom-petency of the valve is readily explained by the passive dilatation of the auriculo-ventricular orifice; on the other hand, the ventricle, and the heart generally, have been found by other observers to be abnormally small. Sansom (73) observed cases in which there have been the physical signs of mitral regurgitation in anaemia when the outline of the heart has been markedly smaller than the normal. The regurgitation in such cases may be explained by enfeeblement of the papillary muscles. In fatal cases of anaemia these muscles have been observed to be profoundly affected by fatty degeneration.

The treatment of cases of mitral insufficiency, the result of anaemia, is practically the treatment of the form of anaemia which is the proximate cause. Though there may be very extensive fatty degeneration of the myocardium, there is good evidence that there frequently occurs a "restitutio ad integrum," new and healthy muscular fibrillae being developed. Absolute rest in a sunny, airy room is the first requisite. The good effects of tepid and cool baths in such cases may be briefly mentioned; the use of baths, spongings, and spinal affusions of cool or even cold water has been a routine practice with many physicians in cases of anaemia. The occurrence of a systolic murmur at the apex is no contra-indication to this mode of treatment. The carbonic acid and saline baths have been used very successfully for many years at Schwalbach, in co-operation with the internal administration of ferruginous water, in the treatment of anaemia. The modes in which such baths influence the heart and blood-vessels have been already discussed.

Group VI. Mitral Insufficiency in Graves' Disease and Allied Affections. —Murmurs in the precordial area are heard in a large number of

cases of exophthalmic goitre. In the majority of these the position of the murmur is over the base of the heart, and especially near the pulmonary artery. In a minority the systolic bruit is heard over the situation of the apex. It is probable that in such cases the insufficiency of the valve is due not to endocarditis but to a disturbance of the tonicity of the heart. In some cases of Graves' disease dilatation of the left ventricle has been indicated during life and proved at the necropsy. In others the heart has been found to be quite normal. In some cases of Graves' disease the dilatation is shewn chiefly in the right chambers; the signs of tricuspid regurgitation are attended by well-marked systolic venous pulsation in the neck. The evidence points strongly to the conclusion that the morbid conditions of the heart advance step by step with the exophthalmic goitre, and that there is no pre-existing disease of the heart. The dilatation of the heart is by no means commensurate with the rapidity of its action. In cases of extreme tachycardia the outline of the heart may remain normal, whilst in other cases in which the rapidity of the heart's action is far less, there may be distinctly progressive hypertrophy and dilatation of the left ventricle. It is probable that the insufficiency of the mitral valve, which occurs in a minority of cases of exophthalmic goitre—structural valvular disease being excluded—has a like pathogeny with that which obtains in anaemia. The valve curtains fail to coapt in some cases on account of dilatation of the ventricle; in others because of enfeeblement of the papillary muscles, or faulty correlation between these muscles and those of the ventricular wall. In the treatment of these cases, supposing that there are signs of failure of compensation, the rules already laid down may be followed; but other therapeutic agencies demand consideration.

The treatment of the cardiac symptoms occurring in the course of exophthalmic goitre is notoriously unsatisfactory. The rapidity and irregularity of the heart's contractions in the majority, and the dilatation of the cavities in the exceptional cases, are not favourably influenced by digitalis or any form of cardiac tonic.

The combination of strychnine and hydrobromic acid is frequently of great service in the management of Graves' disease with severe cardiac symptoms; in other cases belladonna, along with digitalis or strophanthus, produces more beneficial effects. The management of Graves' disease is fully dealt with elsewhere, and this is not the place to discuss the relative methods of such drugs as phosphate of sodium, suprarenal extract, or dethyroidised serum. It may, however, be stated that in the majority of cases suprarenal extract is of greater utility than any other remedy. The continuous current is certainly of much value. The current should be weak, not exceeding five milliamperes; the best method of administration is to apply the anode at the nape of the neck, over the vertebra prominens, and the cathode on the groove external to the larynx and trachea. The current should be allowed to pass for five or ten minutes three times a day; the cathode, which may be moved over the skin without lifting and re-applying, being adapted to each side of the

neck alternately. This method of treatment often requires much patience, as months may elapse before definite amendment takes place. It may be added that the continuous current is often of value in cardiac failure from causes other than those just mentioned. Potain and Sansom have both found it to be of great importance in many different forms of failing compensation. In many cases of Graves' disease, in which failure of the heart threatens to set in, it is absolutely necessary to have recourse to surgical intervention (compare Vol. IV. Part I. p. 332).

<div align="right">A. ERNEST SANSOM, 1898.
G. A. GIBSON, 1909.</div>

REFERENCES

1. ADAMS. *Dubl. Hosp. Rep.*, 1827, iv. 353.—1*a*. ALLBUTT. *Practitioner*, 1869, iii. 342.—2. BALFOUR. *Clinical Lectures on Diseases of the Heart and Aorta*, London, 1876, 124.—3. *Idem.* *The Senile Heart*, London, 1894, 55.—4. BARLOW. *Brit. Med. Journ.*, 1883, ii. 509.—5. BARD et PHILIPPE. *Rev. de méd.*, Paris, 1891, ii. 345, 603, 660.—6. BARR. *Liverpool Med.-Chir. Journ.*, 1886, vi. 307.—7. BENEKE. *Arch. d. Ver. f. gemeinsch. Arb. z. Förd. d. wiss. Heilk.*, Göttingen, 1860, iv. 129.— 8. *Idem.* *Deutsche Klinik*, Berlin, 1863, xv. 128.—9. BLANC. *Des affections cardiaques d'origine rhumatismale traitées aux eaux d'Aix-les-Bains (Savoie)*, Paris, 1886.— 10. BRAMWELL. *Diseases of the Heart and Thoracic Aorta*, Edinburgh, 1884, 421.— 11. BROADBENT. *Brit. Med. Journ.*, 1896, i. 769.—12. BRUNTON. *Lancet*, 1894, ii. 895.—13. CHEADLE. *The Various Manifestations of the Rheumatic State as exemplified in Childhood and Early Life*, London, 1889.—14. CLARK. *Brit. Med. Journ.*, 1887, i. 379.—15. CUFFER. *Les causes qui peuvent modifier les bruits de souffle intra- ou extra-cardiaques*, Paris, 1877.—16. CYRIAX. *The Elements of Kellgren's manual Treatment*, London, 1903, 355. — 17. DAVIES. *The Mechanism of the Circulation of the Blood through Organically Diseased Hearts*, London, 1889.—18. DEBOVE et LETULLE. *Arch. gén. de méd.*, Paris, 1880, i. 275.—19. DUROZIEZ. *Traité clinique des maladies du cœur*, Paris, 1891, 316, 485.—20. *Idem.* *Union méd.*, Paris, 1879, i. 615.—21. FAGGE. *Trans. Path. Soc.*, London, 1874, xxv. 64.—22. FORBES. *Treatise on the Diseases of the Chest and Mediate Auscultation*, 4th ed., London, 1834, 591.—23. FRASER. *Trans. Roy. Soc.*, Edin., 1887-90, xxxv. 955 ; and 1890-91, xxxvi. 343.—24. GARROD, A. E. *On Rheumatism and Rheumatoid Arthritis*, London, 1890, 123.—25. GASKELL, W. H. *Journ. Physiol.*, 1883, iv. 43. —26. GERHARDT. *Lehrbuch der Auscultation und Percussion*, Tübingen, 1871, 2nd ed., 187. — 27. GIBSON, G. A. *Edin. Med. Journ.*, 1880, xxv. 979. — 28. *Idem.* *Journ. Path. and Bacteriol.*, Edin. and London, 1896, iii. 32 ; and 1897, iv. 465.—29. *Idem.* *Index of Treatment*, ed. by Hutchison and Collier, Bristol, 1907, 393.—30. GOODHART. *Lancet*, 1880, i. 479.—31. GOWERS. *A Manual of Diseases of the Nervous System*, London, 1888, ii. 549. — 32. GROEDEL. *Berlin. klin. Wchnschr.*, 1878, xv. 137.—33. HAY. *Lancet*, London, 1883, i. 678.—34. HEITLER. *Die Percussionsverhältnisse des normalen Herzens*, Wien, 1891. — 35. HENOCH. *Lectures on Children's Diseases*, 1889, i. 475, New Syd. Soc.—36. HERSCHELL. *Lancet*, 1896, i. 413.—37. HIS. *Arbeiten aus d. med. Klinik z. Leipzig*, 1893, 14 ; *Wien. med. Blätter*, 1894, xvii. 653 ; *Centralbl. f. Physiol.*, 1896, ix. 469.—38. HOPE. *Treatise on the Diseases of the Heart and Great Vessels*, London, 1832, 341.—39. HUCHARD, *Traité clinique des maladies du cœur*, Paris, 1893, 170 ; and *Ibid.* 157.—40. HUNTER, J. *A Treatise on the Blood, Inflammation, and Gunshot Wounds*, London. 1794, 162.—41. KEITH, A. *Lancet*, 1904, i. 706.—42. *Idem.* *Ibid.*, 1906, i. 623 ; and ii. 359.—43. KENT. *Journ. Physiol.*, Cambridge, 1893, xiv. 233.—44. KING, T. W. *Guy's Hosp. Rep.*, 1837, ii. 104.—45. LAENNEC. *De l'Auscultation médiate*, Paris, 1819, ii. 214.—46. LANCEREAUX. *Anatomie pathologique*, Paris, 1871, 233. —47. LEITH. *Lancet*, 1896, i. 757.—48. LING. See Schreiber, *A Manual of Treatment by Massage*, transl. by Mendelson, Edinburgh, 1887, 23.—49. LOOMIS. *Trans. Assoc. Am. Physicians*, Phila., 1888, iii. 180.—50. LUDWIG und HESSE. *Arch. f. Anat. und Physiol.*, 1880, Anat. Abth. 328.—51. LUSCHKA. *Virch. Arch.*, 1857, xi. 144.—52. MACALISTER, D. *Brit. Med. Journ.*, 1882, ii. 821.—53. MACKENZIE, J.

Brit. Med. Journ., 1905, ii. 1689.—54. *Idem.* *Ibid.*, 1905, i. 587.—55. MARTIN. *Rev. de méd.*, Paris, 1881, i. 32.—56. MORGAGNI. *De Sedibus et Causis Morborum*, Venetiis, 1762, i. 208.—57. MORTON. *Med. Rec.*, N.Y., 1889, xxxv. 403.—58. NAUNYN. *Berlin. klin. Wchnschr.*, 1868, v. 189.—59. NIXON. *Dublin Journ. Med. Sc.*, 1886, lxxxi. 473 ; and 1886, lxxxii. 247.—60. OERTEL. *Therapie der Kreislaufs-störungen*, Leipzig, 1884, S. 203.—61. PARROT. *Arch. de physiol.*, 1874, 2ᵉ sér. i. 538.—62. POORE. *Brit. Med. Journ.*, 1895, ii. 1195 ; and *Clin. Journ.*, London, 1893, i. 267.—63. POTAIN. *Clinique médicale de la Charité*, Paris, 1894, 319.—64. *Idem. Ibid.*, 76.—65. PRINCE. *Med. Rec.*, New York, 1889, xxxv. 421.—66. PYE-SMITH. Fagge's *Principles of Medicine*, 1886, i. 958.—67. RENAUT. *Compt. rend. Soc. biol.*, Paris, 1877, sér. 6, iv. 333, 342.—68. ROGER. *Maladies du Cœur, par le Docteur Constantin Paul*, Paris, 1883.—69. ROSENSTEIN. Ziemssen's *Cyclopaedia*, London, 1876, vi. 3 *et seq.*—70. ROY and ADAMI. "Heart-Beat and Pulse-Wave," *Prac-titioner*, London, 1890, xliv. 81.—71. RUSSELL. *Investigations into some Morbid Cardiac Conditions*, Edinburgh, 1886, 56.—72. SANDERS. *Edin. Med. Journ.*, 1897, N.S., i. 522.—73. SANSOM. *Lettsomian Lectures* (1883) *on the Treatment of Valvular Diseases of the Heart*, 2nd ed., London, 1886, 64, 85.—74. *Idem.* "Chronic Endocarditis ; Valvular Disease," Keating's *Cyclopaedia of Diseases of Children*, Phila., 1889.—75. *Idem.* "The Annual Oration," *Trans. Med. Soc.*, London, 1890, xiii. 481.—76. *Idem.* *Internat. Clinics*, Phila., 1894, i. 4th series, 12.—77. SCHOTT. *Berlin. klinische Wchnschr.*, 1880, xvii. 357, 372.—78. SÉE. *Arch. de physiol.*, 1874, 2ᵉ sér. i. 552, 847.—79. SKODA. *Allg. Wiener med. Ztng.*, 1863, viii. 266.—80. STOKES. *Diseases of the Heart and Aorta*, 1854, 357.—81. STURGES. *Lancet*, 1892, i. 621 ; *Ibid.*, 1892, ii. 469.—82. *Idem.* "Lumleian Lectures on Heart Inflammation in Children," *Ibid.*, 1894, i. 583, 693, 723.—83. SUTTON, H. G. *Lectures on Pathology*, London, 1891, 372.—84. TAWARA. *Das Reizleitungssystem des Säugetierherzens*, Leipzig, 1906, S. 9.—85. ZANDER. See von Ziemssen's *Handbook of General Therapeutics*, London, vol. v. 41.

<div align="right">G. A. G.</div>

DISEASES OF THE AORTIC AREA OF THE HEART

By Sir CLIFFORD ALLBUTT, K.C.B., M.D., F.R.S.

IN its causes and pathology aortic disease may be discussed as a whole ; under the head of symptoms some distinction must be made between the effects of stenosis and regurgitation respectively. These effects are often associated, though in very different degrees ; yet, temporarily or permanently, either may occur alone.

Subject.—By aortic regurgitation we mean that in diastole some of the blood driven into the aorta returns to the left ventricle ; when we hear the sound characteristic of this disorder the inference that the aortic valve or orifice, or both, are out of order is almost irresistible. A definite diastolic murmur heard in the areas of the murmur of aortic regurgitation is the surest of diagnostic indications. With aortic systolic murmurs it is not so ; of such signs these are among the least significant. I need not argue that an "aortic systolic murmur" may not be significant of organic disease at all ; and indeed, when significant of disease about this orifice, an alleged stenosis may be more apparent than real ; the murmur may signify no more than a deformity of the part, implying no constriction of the orifice ; indeed a dilatation of the orifice, or of the contiguous part

of the aorta beyond the orifice, is more frequent. It is too much the custom to speak of aortic stenosis in all cases of organic disease of this orifice attended with a direct murmur; aortic obstruction is not a much better term, and we shall do well to speak of a direct or systolic murmur only, until by consideration of all its features these relative dimensions can be estimated in the particular case. Notwithstanding, it is probable that in a sense all disease about the orifice is in some degree obstructive, as it is apt to hinder the normal play of the elastic and muscular components, and to increase fluid friction.

Causation.—The causes of the diseases of the aortic area of the heart (omitting congenital malformation, which is dealt with in another article, p. 276) are chiefly three; namely, infectious diseases, mechanical strain, and the ordinary atheroma of senescence.

Infectious Diseases.—Of these, rheumatism is the chief; syphilis comes second; the poisons of other infections, such as diphtheria and influenza, fall more frequently upon the muscular structure of the heart than upon its valves or orifices. Acute endocarditis has been dealt with already, and Dreschfeld has described a case in which infective endocarditis fastened upon a ruptured aortic valve. In its liability to disease, and in the nature of it, the aortic area of the heart is so bound up with the aorta itself, and especially with the first portion of it, that no inconsiderable part of the present subject is contained in the Articles on " Diseases of the Arteries " and " Aneurysm " respectively. This common liability of the orifice and of the near parts of the aorta above it is seen especially in the cases of syphilis and of atheroma. Indeed, whether the aortic valve is ever attacked by syphilis primarily and more or less exclusively, or only in common with or consecutively to a belt of the suprasigmoid aorta, is still a matter of doubt. Apart from the presence of the *Treponema pallidum*, pathological histology has not yet enabled us, by inspection, to rely upon any differential characters of syphilitic disease; although in many cases, and in most recent cases, a fairly safe opinion may be given. During life, and especially for the determination of the course of therapeutics where the history is inconclusive, Wassermann's serum test, if it be simplified in method (Schütze, Danielopolu), may prove of much service. Gout and its associates, such as plumbism, seem to produce lesions not distinguishable from " atheroma," under which easy-fitting name their agency may be included.

By far the chief cause of aortic disease in persons under middle age is rheumatic fever; as is atheroma in those over this time of life. Rheumatic fever falls first, and as it were by preference, upon the mitral valve; when the aortic valve is implicated it may suffer with the mitral, but generally after it. Usually, in my experience, it creeps, with a contrary movement to atheroma, from the anterior mitral curtain to the adjacent aortic cusp, which may thus be the only cusp to suffer directly; or the inflammation may invade the other cusps also, usually in less degree. Dr. Mackenzie has noted occasionally after the appearance of a mitral murmur, and before the appearance of an aortic diastolic murmur, an increase of the interval between the auricular and the

ventricular systoles ; as if due to extension of carditis from the mitral
area onwards to the tract of His.   But attacks of rheumatic fever upon
the aortic valve primarily, and even exclusively, are not unknown,
especially in men ; in some such cases it may be either an extension
of the supravalvular aortitis prone to occur in this disease, or of
pericarditis.   I think that I may have seen it thus limited even
in women, but in them, as in children, this singularity is very rare.
The following proportions of sex in rheumatic valvular disease were
calculated by Dr. Hartley from St. Bartholomew's Hospital :—aortic
regurgitation, males 83 per cent ; mitral regurgitation, sexes equal ;
mitral stenosis, females 58 per cent.   Dr. Norman Moore in 63 male and
37 female cases under his own care found that in 9 the aortic valve
suffered alone, and all these were males.   With the mitral valve, I need
not say, the converse is the case ; in the large majority of cases of
ordinary severity rheumatic fever maims this part of the heart without
implicating any other valve.   But in other cases an apparently simul-
taneous implication of both areas, or at any rate the rapid succession of
inflammation in them, together perhaps with pericardial and myocardial
symptoms, suggests that the inflammation is primarily universal, and
virtually independent of extension by contiguity.   Yet the invasion of
the aortic valve by rheumatic fever being in women so much rarer than
in men ; and in general experience, I think, aortic lesion is added to
mitral so predominantly in men, that, whether the aortic mischief
be synchronous or propagated by continuity, we are led from the
double valvular disease to infer the not infrequent intrusion of some
factor other than the rheumatism.   This factor may be mechanical
strain.   Some prevalences of concurrent mitral and aortic disease
after rheumatism we may regard indeed as confirmatory exceptions ;
such, for instance, as the cases of women engaged in labours harder than
those usual in the sex—in women who have worked in the fields, in
washerwomen, in women employed in brick-making, or on the banks
of mines.   After making due allowance in such cases for exposure to
weather, there seems to be a greater prevalence of aortic mischief after
rheumatic fever among labouring women than among women who have led
lives of less muscular stress (*vide* p. 428).   I have not found that either
in alcohol or syphilis we have factors to invalidate such an interpre-
tation ; but to pursue the subject much farther would be to trench upon
Endocarditis.   I have alluded to invasion of the aortic area by pericarditis.
Ferrio, who has studied this question in Bozzolo's clinic, has adduced strong
evidence of such extensions, both inwards and outwards.   Acquired aortic
disease in children, say under the age of ten, is among the rarest of clinical
events : as in the acute rheumatism of women, the mitral valve is usually
affected first ; if the aortic be involved, it suffers later.   In the chorea
of young subjects, whose muscular efforts in health are fitful, not con-
tinuous, disease of the aortic valve alone is a most unusual event ; of 250
cases Sir W. Gowers found aortic regurgitation in 2, and obstruction
in 1.   Now on a careful analysis of the symptoms in these rare cases

of solitary aortic lesion, Ferrio argues that pericarditis had preceded or accompanied the aortic lesion. In the reverse order of events rheumatic endocarditis may set up pericarditis sicca around the roots of the great vessels. Cases of temporary murmur, due to pressure of pericardial effusion, were of course excluded. I have repeatedly called attention to Pawinski's cases of aortitis with angina pectoris, arising from pericarditis around the root of the aorta.

The predominance of rheumatic inflammation on the left side of the heart is often explained in like manner ; namely, by the larger variations of blood-pressure, which fall more hardly on these valves than on those of the right side ; and so it may be, yet it is not easy to explain such a preference. Are we to assume that before the rheumatic attack in these persons, muscular labour had already produced some impairment of structure ? This would seem to be a grave charge against the physical uses of the body ; and the proposition, on the face of it, unreasonable, if so be that without the rheumatism no harm would have come of them. And on a remote suspicion of such a deterioration are we to discourage all exercises beyond nursery games ? Short of strain, one would antici- pate that vigorous work would enhance nutrition, and thus fend off rather than invite the approach of disease. On this problem the studies on the elastic properties of the arterial wall by Roy, M'William, and Herringham and Wills should be consulted.

Syphilis is directly concerned in the causation of many cases of aortic disease ; and even when the lesion is not obviously gummatous in form, or the parasite not apparent, the history, and the clinical and pathological features are usually suggestive enough. The part of syphilis in arterial disease will be shewn by Dr. Mott (p. 562). This specific lesion of the aortic area is now common knowledge, as are also the morbific effects of the poison upon the whole arterial tree. Dr. Parkes Weber (127) con- siders that syphilis is no infrequent originator of ordinary atheroma. A somewhat acute, cushion-like, more or less annular lesion in the suprasigmoid aorta of a person of or under middle life is pretty certainly syphilitic ; and in not a few cases the symptoms betray such a lesion almost unmistakably. There will be fewer oversights in particular cases during life if the suspicion that an aortic lesion may be syphilitic is always with us. Even if we can elicit no evidence of an infection, the inferences from the story of the case, or from associated changes and relics elsewhere on the body, will generally bring us to a moral certainty. We know that a comparatively young man of otherwise healthy habit does not suffer from local disease of the aortic region of the heart unless it be in consequence of some extraordinary muscular stress, or of rheumatic fever, or of syphilis; if then such muscular stress and rheumatism be dismissed, we fall back upon syphilis, as we do with a like assurance in the case of aortic aneurysm in such a person. The inference is often justified by the effects of specific treatment ; and by prompt treatment we may dispel the disease while confined to the supra-aortic area, and save the valve. Waning of the first sound, and perhaps Mackenzie's auriculo-

ventricular delay forbode its advances. In obscure cases, as I have said, Wassermann's test should be undertaken. The following case, one of no uncommon kind, illustrates these remarks :—

Dr. Pye-Smith reported a case of a man, aged thirty-two, who died with heart disease, the physical signs being those of aortic obstruction and regurgitation. Rheumatism and chorea were excluded. Atheroma was improbable, owing to the comparatively young age of the patient, who was, moreover, not subject to laborious work. After death there were no signs of rheumatic or infective endocarditis, but a patch of recent aortitis and a deformity of the valve. The lesion was soft, injected, with a swollen crescentic margin suggesting the advancing edge of a secondary syphilitic eruption of the skin ; there was no atheroma. The only other evidence of syphilis was a fibroid condition of the testicles, though this was not very marked. He suggested that the syphilitic aortitis had spread to the valve and so produced the disease in question (70).

Atheroma.—For a full discussion of the nature and fashion of this disease, or common result of several diseases, the reader is referred to the article on " Arterial Degeneration and Diseases," p. 582. But here we may ask if disease of the aortic orifice sheds any light on the origin of this insidious and especially, if not peculiarly, human lesion. Does labour play an exclusive or predominant part in the origin or determination of the change ? The evidence on the question is conflicting. It is true, no doubt, that atheroma is found more or less exclusively on the left side of the heart, the side of extreme variations of stress ; it is true that if it occurs in the pulmonary artery it is in cases of high pulmonary resistance ; it is true that peripheral arteriosclerosis is very common in labouring men ; but it is also true that atheroma may be the ultimate issue of arterial disease of whatsoever origin—rheumatic, syphilitic, mechanical, and so forth. Every physician will admit that atheroma is as likely to be found in the aorta of the elderly lady who has spent her life in trotting amiably about the parish, as in her husband who has ridden for his falls, felled his own trees, and stumped about after his birds from his boyhood. On the contributory effect of muscular exertion, of which I shall say more presently, we may note now that as life advances—say after the age of thirty-five—the aorta is altered by the slow substitution of fibrous for elastic tissue, and by the imperfectly repaired damage of molecular strains, which the inferior fibre is indeed more able to resist, but is less able to recover from. Such initial lesions we see in the yellow streaks at the root of the aorta, at the bifurcation of the larger arterial branches, and so forth. The co-operation of toxic causes accelerates and perhaps modifies the process ; and in some persons the vessels seem more vulnerable than in others. Yet although in and after middle life an aorta has pretty surely altered thus in elastic quality, and fibrous tissue has displaced the elastic, or succeeded it, in large measure, the vessel to the naked eye may appear normal ; there may be no obvious "atheroma." Indeed, before atheroma becomes visible, the aorta may thus become deformed in outline. Had we the means of following histological changes during life, we should

probably see reason to make some distinction between this increase of connective tissue and atheroma ; but clinically we can only become aware of such changes by results in the later stages of vascular disease.

Atheroma, in the areas which it occupies or selects, is by no means constant, nor approximately uniform ; although usually well marked in the arch of the aorta, where tensile stress is high, it is not by any means confined to areas which are affected by the main stresses of muscular exercise, or to parts where tone is least and tension most.   On the contrary, it is one of the marvels of practice to find it now in one district, now in another ; and within them it is patchy.   If in one necropsy atheroma is abundant about the heart and thoracic aorta, in another the heart and its orifices are fairly normal but extensive patches of atheroma are discovered in the abdominal aorta, or in the peripheral areas of the arterial tree.   In one body the cerebral vessels are like branched corals ; in another, with atheroma enough elsewhere, the cerebral vessels may be fairly clear of it.   Such contrasts are discussed in other articles ; they may depend in part upon the diversities of functional activity in different persons, in part we may have to take a step back to various diseases of the vasa vasorum.

Again, if one chief cause of atheroma of the heart and aorta must be mechanical stress, yet this stress may be due, not to athletic exercise, but to that more persistent high arterial pressure of constitutional origin which may be established as well in the squire's wife, with her easeful habits and gouty inheritance, as in the squire himself who day by day works off his excess of meat and drink in the fresh air.   Animals, which have exercise enough—muscular stress enough, many of them—enjoy a comparative freedom from atheroma.   Domestic animals are fed by their owners, and usually fed economically.   It would seem then that, toxins apart, atheroma may be due to strain, but less perhaps of muscular exertion than of persistently enhanced arterial blood-pressures.   In cases of stenosis we may see how this alteration may protect the aorta from atheromatous lesions.   It would seem that it is not the occasional high blood - pressures of muscular labour, but the more persistent high pressure due to luxus - consumption relative or positive, to goutiness, especially in its non - articular forms (for the frank articular form of gout leads less surely to high arterial pressure), to lead poisoning, and possibly to certain products of metabolism engendered or retained in ageing organs or tissues, which produce the degenerative changes which invade the aortic region of the heart.   And this morbid hyperpiesis may be maintained by a powerful heart even in old age.   We see frequently in the post-mortem room, yet still with some surprise, how readily the heart even of an old man may take upon itself no puny hypertrophy.   It is no unusual thing to find a big heart, and one big with no bad stuff, in old persons subjected in later life to increased blood-pressure, or to aortic stenosis ; and this even when the coronary arteries have undergone no inconsiderable measure of deterioration.   In such cases the aortic valve, though practically always thickened—as under

the stress of mitral stenosis the tricuspid valve will thicken—and often somewhat deformed, is generally competent against regurgitation. In senile atheroma a systolic murmur, if not continuously audible, may usually be awakened by a short and easy gymnastic, such as waving the arms. Indeed it may thus be produced in almost all old people. As the heart is then often quite competent, this form of aortic disease is rarely of itself the immediate or even a proximate cause of death ; it is but an incident in the course of a general cardiovascular involution, which was described almost as well by the older pathologists, before the advantages of ausculta- tion, as by ourselves.

This general vascular decay may set in comparatively early. I have seen two cases in the last two years, in persons between forty and fifty, of universal (so far as observation by palpation and auscultation could extend) and extreme calcifying atheroma without high blood-pressures, present or past. In both the general features were those of precocious old age. In neither was there any notable history of infection.

Muscular Strain or Violence.—The effect of bodily exertion in pro- ducing disease of the heart, which was apprehended by Morgagni, had again, in the newer study of the effects of rheumatism, been overlooked until attention was recalled to the subject by Peacock, Myers, Da Costa, Seitz of Zürich, Sir James Barr, and others, including myself. That muscular exertion is among the causes of aortic disease, and may deter- mine the occurrence of aortic regurgitation, is now admitted. If, in- deed, a man under forty-five years of age, in whom there is no evidence of premature decay or of syphilis, presents symptoms of aortic regurgita- tion without mitral disease, we may suspect that effort or violence in one way or other contributed to its occurrence. [*Vide* Art. " Over-stress of the Heart," p. 193.] Sudden muscular stress may damage the aortic valve, even to the point of rupturing a limb or limbs of it ; the posterior cusp, it is said, most frequently. A cusp may split from the free border across, or may be torn at the base from its attachment. The accident is not very uncommon, and the cases on record are so many as to make it unnecessary to accumulate examples. A good collection of cases will be found in Dufour's thesis. Heller records a remarkable case of violent effort in which an aortic cusp was forced inwards, and a pouch driven out from the wall of the aorta above it. The author considered that there was no syphilitic factor in this case, but I think (from his own paper) that syphilis was a contributory factor ; though, as Heller was one of the earlier observers of cardiac syphilis, it is hazardous to differ from him. Among the symptoms was intense severe angina pectoris due, in my opinion, to the deep suprasigmoid lesion. And—what has been noticed in necropsies before (Rokitansky, Fränkel)—the torn aortic area in the six months' interval had attained to some histological healing, including the investment of a new intima. Such an accident may happen likewise to the mitral valve, though far more rarely (Habershon's case) ; indeed we read of simultaneous rupture by violence of both these valves ; or again, of rupture of both an aortic cusp and the left ventricle

itself, as in the classical case, reported by Bouillaud, of an old woman, no doubt of degenerated tissues, who threw herself from a third-floor window. Peacock, in his Croonian Lectures in 1865, adduced 17 cases of rupture of the aortic valve, one or more of the aortic crescents being thus ruptured. In my own experience blows or crushes have resulted sometimes in aortic aneurysm, more often in rupture of the aortic valve. I have referred to Dufour's list of such cases. Indeed rupture of an aortic or mitral valve may be produced experimentally by smiting the walls of the thorax in animals, or with a fully charged heart in the cadaver, as many pathologists have demonstrated ; among them Chauveau and Marey, Potain, Duroziez, Barié, François-Franck, Rosenbach, and Dufour. It is scarcely irrelevant to allude to a case of mitral stenosis, under my care (in the Leeds Infirmary) in which, after the closest inquiry, we confidently attributed the lesion to the kick of a horse on the cardiac area. The patient was a young man, and the symptoms were some months in declaring themselves ; yet the connexion between antecedent and consequence seemed conclusive. Heidenhain of Greifswald, who also has studied these cases, agrees that, with or without injury to the ribs, or even without obvious external bruising, an external blow may (*a*) rupture a valve in the heart, may (*b*) damage or rupture the cardiac muscle, or rarely (*c*) by insidious endocarditis produce a stenosis, as in the case of my own just quoted. Sir Samuel Wilks has recorded a case of a youth, aged nineteen, in whom a blow on the chest ruptured the posterior cusp of the aortic valve from its free margin to its base ; a small deposit of fibrin had begun to form on the raw edge. I suppose such an accident might happen in boxing. A case in a lad of sixteen, in which the heart itself was ruptured by a blow on the chest which caused no external bruise, was reported by Dr. Groom of Wisbech ; the preparation is now at Cambridge. Dr. Lawton Roberts records a similar event in a child whose heart had been reported as normal at St. Thomas's Hospital a few months before. Deganello's patient, a healthy man, aet. thirty-six, was crushed between a cart-wheel and a post. He lived a few days, and died with pulmonary oedema. The healthy cusps had been torn away ("arrachement") from a healthy aorta ; both ventricles were dilated. Cases of this kind may give rise to litigation, and to medico-legal questions of no little difficulty (Bernstein). In the course of such inquiries the physician will do well to consult the papers of Peacock, of Barié (who gives 38 cases of traumatic aortic injury from the records of the nineteenth century), and the more recent essay of Sinnhuber. Drs. Calwell and Mark published a case in a man, aged twenty, of clean history, and accepted three weeks previously for life insurance ; he fell from a scaffold, and was much bruised. On re-examination, ten days later, besides a (double) aortic murmur, water-hammer pulse, throbbing arteries, and enlargement of the heart to the left, a mitral murmur was noted also, and attributed to the sudden dilatation. In the history of some of these accidents the distinction between outer and inner stress cannot be made ; but

probably in all the mechanical process is similar, the external blow violently compressing the thoracic cage. We may conceive of a violent concussion of the distended aorta just at the moment of the short pause ; the glottis being closed in deep inspiration, and the thorax rigid. Moreover, by the muscular compression the carotid and subclavian arteries may be gripped, and, between the crura of the diaphragm, even the aorta itself. Potain argues that if a blow, such as a jockey received who was heavily thrown so that his chest struck the ground, cause, as in him, the rupture of an aortic cusp, the heart at the moment of the blow was in systole, and the aorta distended. In cases of rupture of the mitral valve, on the other hand, of which Potain records two examples, he argues that when the blow fell the heart was in diastole and the ventricle full.

It must not be assumed, however, that in every case of sudden and distressing failure of compensation a valve is ruptured ; the heart or orifice may yield suddenly, with symptoms and signs perhaps little less severe. In this case prognosis, which must depend upon the whole story, may be more favourable. Of such probably was Dr. Marshall's case of a man who, while running for an omnibus, heard a murmur arise in his chest, but with no immediate sense of injury. The murmur proved to be one of aortic regurgitation, and the patient died of gradual heart-failure. We must not forget that in the case of an external wound cardiac disease may be due to the supervention of an infective endocarditis.

Clean or jagged slits in the intima of aortas apparently healthy are more curious than very rare. For instance, Huber narrated a case of such a rent ("eine kleine Langriss") in a healthy young man, aged twenty, in whom otherwise the aorta and heart were quite normal ("Aortenwand ganz normal"). It followed a terrible fright. A diffuse aneurysm rapidly followed, and he died within the day. The rift in this case was at the upper part of the arch. In Dr. M'Weeney's case the aortic arch (with the left pulmonary artery beneath) was torn across, three-quarters of an inch beyond the left subclavian, by a fall from a scaffold. In Weyrauch's case, a male aged thirty-seven years, without any history of syphilis or of strain, there was a sudden onset of anguish, followed by a pulseless condition and death in five hours. The necropsy shewed haemopericardium due to total transverse rupture of the aorta with sharp edges. The aortic intima was bright and smooth, and if there was any atheroma, there was certainly none near the rent. The aorta was not dilated, and there was not any evidence of syphilis. In a case of mine, a very healthy boy, aged thirteen, during very severe exertion a rupture of the aorta into the pericardium took place a little above one of the cusps of the valve. Many such cases are reported (compare p. 639).

It is more difficult to estimate, or to apprehend, the part of muscular effort or strain in the production of aortic regurgitation of insidious origin. In 1870 I wrote that such acute strains as mountain-climbing and the like were apt to tell rather upon the right heart, the chronic effects of years of labour upon its left side. With this opinion Roy agreed, and, broadly speaking, I still entertain this opinion (*vide* p. 241). When, however,

a vigorous and fresh-complexioned physician of some thirty-five years of age, carrying a heavy patient on a sudden emergency up a flight of stairs, felt a sense of something having given way inside his chest, and became suddenly breathless and oppressed ; when thereafter a murmur of aortic regurgitation was heard, which murmur continued to the end of a life prematurely cut short by the disease ; when, moreover, no trace of syphilis could be detected, or even suspected, either by himself or his medical friends, we are almost bound to suppose that it was by the sudden stress alone that the valve was strained and ruptured. One of Prof. Anderson's three cases was in a young man, aet. nineteen, who on lifting a great weight, felt the snap in his chest, and a double aortic murmur was established. Unfortunately no necropsy was obtained. In a second case the man was aet. twenty-four, but there was a history of syphilis. In the third there was no syphilis nor other irregularity or defect of health ; and after death, besides rupture of a cusp and small dilatations in its sinus, there was no marked evidence of atheroma. But the man was fifty-five years of age. Dr. Cautley found aortic regurgitation in a city clerk, aet. thirty-four, after making Saturday records on a bicycle to Brighton. Two years before he had had influenza. In Dr. Theodore Fisher's Bristol cases (sailors, etc.) syphilis was probably a considerable factor, though Dr. Fisher thinks a healthy valve may be ruptured (see also Bramwell (17)). The doubt about the problem is if this accident may thus be produced in an aorta and valve previously quite sound ; yet even in favour of this proposition there is much strong evidence. To the experimental evidence I have already referred (*vide* p. 425). If the patient survive, even for some days only, vegetations may form so rapidly as to obscure the anatomy. However, to emphasise the duty and yet the difficulty of exact appreciation in such cases, I will quote one more case, published by Dr. Lauriston Shaw, in a young man who, by a violent effort against the lurching of a ship, ruptured the valve. Dr. Shaw was at first convinced that the case was one of sudden rupture by mere strain ; and so it stood in my notes till, on the revision of this article, Dr. Shaw kindly informed me that the patient in former years had had an attack of chorea, which he had forgotten. So the valve which was forced by the effort may not have been previously intact. In Dr. Hartley's case the man was to all appearance healthy ; but he was forty-seven years of age, and twenty years before had been infected with syphilis.

To an intermediate class belong cases of traumatic aorti-valvulitis. For example, a young, slightly-built housemaid of healthy stock presented the ordinary signs of aortic stenosis without any other lesion ; no sign of rheumatism, chorea, influenza, or other infectious disorder was to be seen or heard of ; she told a clear tale of something suddenly giving way in her chest, with pain and distress, while she was lifting a heavy bed, from which moment she became incapable of exertion. We could not avoid the conclusion that during this effort an acute aorti-valvulitis was set up in healthy parts with consequent

constriction. This patient has been in Addenbrooke's Hospital repeatedly, in the first instance at any rate under Prof. Bradbury's care ; and once in St. Thomas's under Dr. Hawkins, who most kindly wrote to me about her. During University examinations she has been examined by many physicians, and the view here given of the causation of the mischief has been unanimously accepted. The signs were those of stenosis of the aortic orifice ; the acute symptoms those of angina pectoris. She has now recovered apparent health, but a harsh systolic murmur, a thrill, and an enlarged left ventricle remain. Peacock has stated that aortic disease is to be found in young women servants subjected to straining efforts before they are fully grown. When, therefore, in a person the subject of aortic regurgitation, we learn that there is no definite story of a sense of injury on a particular occasion ; that the oppression came on more or less insensibly ; that the patient had not been in the way of syphilis, of gout, of alcoholic excess, or of some other cause of arterial degeneration, but had followed a laborious employment, we may fairly presume that muscular stress, or stress and decay together had gradually impaired the valve to the point of insufficiency. The difficulty lies in the interpretation of cases of sudden valvular failure in which latent toxic factors may have preceded a moment of conspicuous muscular effort. Senile decay alone does not usually cause aortic regurgitation, but disease of the aorta with implication of the orifice, betrayed by a direct murmur. But when we find that regurgitation occurring in persons under fifty years of age, of the laborious sex, and especially in those who have been engaged in heavy toil, we cannot but suppose that muscular stress, if not the sole or always the chief agent in the event, is at any rate a potent determining cause.

If then not infrequently, yet almost exclusively in men, we discover that aortic insufficiency occasionally establishes itself in patients under the age of senile degenerations, free from evidence of syphilis or other infection including rheumatism, or of sudden rupture, we reasonably assume that the disease is attributable to the accumulated effects of muscular stresses recurring at longer or shorter intervals over many years. But, if a man of irregular habits, and deteriorated tissues, describes to us the symptoms of sudden rupture of the aortic valve, we shall as reasonably infer that an effort, inadequate to rupture a healthy aortic valve, had sufficed to rupture a valve already impaired.

Dr. Seymour Taylor in a private letter reported to me three such mixed cases, in which regurgitation was due to the stress of effort upon tissues affected by syphilis, malaria, and plumbism respectively. The curious point was that in all these the diastolic murmur was "musical," and in all was "B below the staff" ; presumably in each the aorta was of the same proportions. The quality of the note also was approximately the same. As all three patients got along fairly well, the sudden change may have been a small rent or perforation in a diseased cusp.

It seems, then, that in the causation of aortic insufficiency due to muscular strain, we may formulate three classes : namely, a few cases

of rupture of a healthy valve; cases of sudden or chronic forcing of a valve previously impaired by some constitutional poison, such as syphilis; and cases of chronic forcing of the valve by the importunity of continual muscular strains, none of which alone was sufficient to break down the valve, but all of which, by molecular rather than massive lesions, probably by repetitions of slight attacks of valvulitis, contributed gradually to break down the resistance of the part (*vide* Roy and Adami). I may repeat that the condition of the tricuspid valve in protracted cases of mitral stenosis is a good example of chronic valvular impairment due merely to mechanical stresses. We may note in passing that, in respect of prognosis, it is important to know if the injury to the valve is being counteracted by tissues otherwise healthy; and, if so, for how long this is likely to continue.

It has been alleged that prolonged acceleration of the heart, as in Graves' disease, may produce the valvulitis of strain; but unless the sum of work done in unit of time be considerably increased, which is not usually the case, such a result is not to be anticipated.

Nervous Stress.—These influences are fully considered under "Over-stress of the Heart" (p. 225).

However true it may be that sudden emotion or prolonged grief may invalidate the chambers of the heart, such evidence as we have, physiological and clinical, seems to indicate that the aortic machinery at any rate is subjected to no exorbitant stress thereby; perhaps rather the contrary. But it is probable that the nervous system is intimately concerned in the maintenance of compensation.

Pathogeny and Morbid Anatomy.—Whether the heart of labourers and athletes, under normal and relatively normal conditions, undergoes primary hypertrophy is one of the most important problems which meet us at the outset of this part of our inquiry. The answer to the question is not yet final; but the affirmative opinion is gaining ground. Whatever books may repeat, it is no easy task to appreciate a moderate hypertrophy of the left ventricle, for the sources of error are many. Violence of impulse is by no means directly related either to the volume of the heart and its content, or to the blood-pressure; the "heaving quality" of the impulse may be hard to appreciate in degrees of hypertrophy so early as we are now contemplating, and a slight displacement of the apex is no less difficult to ascertain, the form of the chest, the volume of the lungs, the height of the diaphragm and other landmarks being far indeed from constant. In slim, long-chested young men, with wide costal interspaces and relaxed vessels, a thumping or uncovered heart is too often mistaken for hypertrophy or dilatation; and if perchance a haemic or "pulmonary" murmur were heard also, a false diagnosis of aortic disease might be given. If the hypertrophy be attended with a disproportionate dilatation, the heart cannot be described as even quasi-normal. Unless the person under observation be an inordinate drinker of fluids, alcoholic or other, there seems no reason to anticipate an increase of the mean ventricular output over periods of

days or even of hours : if, however, the sum of the conditions of resistance be higher than in ordinary men, hypertrophy may be anticipated ; still even in this case cavity-enlargement need not take place. The heart has to deal with large fluctuations of output in persons whose muscular stresses are not extraordinary. We have seen that in untrained men prolonged exertions may make themselves felt (p. 205) in an uncompensated dilatation ; but perhaps in sprint runners, putters of weights, wrestlers, and the like, in whom sudden repeated straining efforts, with 30 and 40 per cent rises of pressure, bear a large proportion to more ordinary exercises, the mean blood-pressure will probably be enhanced, perhaps considerably ; the maxima are so high and of such frequent recurrence, that the remarkable intervals of low pressure and infrequent pulse in such persons while at rest (p. 200) may not quite restore the sum total to the mean. If this be so, simple hypertrophy will follow, and the axiom will be fulfilled that in mammalia the heart-muscle stands in definite proportion to the mass of skeletal muscle.

The researches of Myers, Da Costa, Thurn, Fräntzel, and others, on hypertrophy of the heart found in men submitted to physical stress, were made chiefly upon soldiers (*vide* p. 235), but in these men contingent conditions have to be considered : omitting drink and syphilis, many ill-fed, untrained, half-developed recruits are (or then were) clad in ill-fitting clothes, girthed with belts and breast-straps, loaded with 20 lbs. and more of weapon and kit, and sent on long harassing marches. In civil life we see the muscular or neuro-muscular evils which flow from like causes, and how tedious may be the recovery from them. Effects of this kind are discussed in the article on Over-stress of the Heart (p. 235). We have heard little from physicians of the Navy of cardiac hypertrophy ; drink and syphilis are far from unknown, but sailors are clad in easy dress, and are not "trashed about." Dr. Guthrie Rankin, however, does state that in seafaring men, in whom violent efforts, exposure to weather and neglected syphilis are common, aortic disease is frequent ; although on the other hand acute rheumatism is almost unknown among them.

It is said that in hard-worked animals, such as foxhounds, greyhounds, and racehorses, simple hypertrophy of the heart, unassociated with cardiovascular disease, is met with. Yet Sir John M'Fadyean, of the Royal Veterinary College, writes to me, "I have not formed the opinion that an amount of muscular tissue notably above the average is ever found in the heart of a horse or dog as the result of great muscular stress, but that hypertrophy of the left heart is always the result of some morbid condition of the valves or of the arteries. . . . If muscular effort were a cause of simple cardiac hypertrophy, it should be almost the rule in 'bus horses, and such is certainly not the case." Colonel F. Smith is practically of the same opinion ; though he admits occasional cardiac hypertrophy. Upon these opinions we shall reflect that these animals have relatively short lives, that the proportions of work, rest, and feeding have been very accurately determined by commercial experience, that in the build of a horse the heart and circulation are originally developed

in correspondence with a very large bulk of voluntary muscle, and have relatively little engagement in other functions; and finally, that the horizontal position is more favourable to the circulation.

The load factor of the heart, the ratio between its mean and its maximum work, is ample; as Cohnheim used to impress upon us, the heart has an astonishing "reserve capacity." If by partial ligation of the aorta the resistance be increased to three or even four times the normal mean, the arterial blood-pressure will not fall; although for this balance the left ventricle has to do three or four times its ordinary work.

If some hypertrophy of the left ventricle be a quasi-normal consequence of extraordinary muscular exertion, at what degree does its excessive vigour begin to menace the integrity of the adjoining section of the aorta or the orifice itself? Roy and Adami noted (*vide* Vol. I. p. 800) that "when the aorta of a dog was suddenly and greatly constricted, and consequently the pressure in the proximal portion of the vessel greatly increased, the plasma of the blood was forced into the cusps of the aortic valve, and vesicles of lymph made their appearance on its under surface, in that region where fibroid thickening is most frequent in cases of chronic high arterial pressure." It is in this way probably that in mitral stenosis sclerotic lesions of the tricuspid valves are established. If these intermittent high aortic pressures are not compensated by peripheral dilatation, and by sufficient periods of low pressures during rest, the wear and tear about the aortic orifice may be considerable; and it is here that even in comparatively young persons we find those yellow streaks, or stream lines, which are the first inscriptions of an invading atheroma.

Provisionally, then, we are led on the whole to the opinion that to muscular exertion, unless very sudden and excessive, and attended perhaps by fixation of the chest-walls with arrest of breathing, and by some moment of differential pressures in ventricle and aorta of which we know little— moments in which, as we have seen, a valve may be directly ruptured— some other factor must be added to bring about aortic lesions in men of young or early middle life. This factor may be a proclivity, natural or acquired, to constitutional high blood-pressure; or, on the other hand, by some debilitating or toxic cause, such as syphilis, anaemia, or "misère," the normal tenacity of the vessels may be so reduced that ordinary blood-pressures are relatively high. A patient of mine, who had certainly done all that he could to strain his heart, if by physical stress it could be done, died of dilatation ("true aneurysm") of the aorta, a result put down unhesitatingly by his friends to over-exertion; yet he and I were aware that a syphilitic infection had occurred many years before, and that not a few evidences of the infection, among which were transient symptoms of encephalic arteritis, had from time to time betrayed its persistency. The great vessels had been impaired by specific arteritis; the muscular stress did but accelerate the evil. If we are to form a definite opinion of the part played by muscular stress in the causation of aortic disease, with or without the intermediation of hypertrophy of the left ventricle, we must weigh with it in the particular case all other factors

which may have conspired to the same end. It is then in a highly complex sense that we may speak of aortic disease as the result of excessive arterial blood-pressure. When we turn to patients over forty years of age, we have seen that, even in the absence of toxic causes, a substitution of fibrous for elastic tissue, while fortifying resistance, may yet so narrow the limits of elasticity that when these by some heavy stress are exceeded, the resulting strain may be irreparable. It is in this phase of life that both aorta and ventricle are liable to permanent deformation; after the age of forty, however, labour is usually less, not more severe or impulsive.

Peter, Traube, and later authors such as Huchard, have insisted upon a distinction between aortic disease originating in the heart itself, such as that of rheumatic valvulitis in young and otherwise healthy persons, —cases in which the cardiac affection is in its initiation a local disease, —and implication of the heart in those more general constitutional changes, such as syphilis or a less specific arteriosclerosis—wherein the heart disease is but one feature of a general disease. These divisions have been distinguished by such names as " cardiopathy," "arteriopathy," " cardiosclerosis." The distinction, if occasionally useful, has been made far too formal; these infinite variations of nature deride our logical devices. Whilst primary cardiac disease tends to generalise itself, conversely constitutional disease derives much of its peril from the cardiac factor; the series start, it is true, from opposite points, but they meet and overlap; so that, unless it be in extreme cases, in the interpretation of which we are not likely to go astray, the distinction is usually too artificial to be of much service. It is too readily assumed, for instance, that arteriosclerosis, even in the cardio-aortic area, is necessarily attended with considerable impairment of the myocardium. Albeit such contrasts, obvious as in one sense they are, may have a practical value in the study of origins; and occasionally may influence prognosis and treatment in particular cases. The experiments on valvulitis of Roy and Adami, already quoted, throw some light on the process by which blood-pressures, relatively excessive—I say relatively excessive, because, of course, a hypertrophied left ventricle does not by any means necessarily imply a positive increase of arterial pressures—set up in a more gradual way opacity and condensation of the valves; and as the aortic valve suffers so may the aorta (97), and an atheroma of strain may invade no small part of this vessel. Whether, then, the initial injury be such as this, or a rheumatic valvulitis, or a syphilitic, the valvular lesions may merge into a common form of atheroma; and if so, the line between the aortic area of the heart and the aorta itself being no barrier to the extension of disease, the aorta dilates, its elasticity is impaired, its walls are diseased, and the coronary arteries become themselves less pervious, or are blocked at their orifices. The earlier milky opacity of the valve thickens into a denser and more puckering sclerosis, extending to the fibrous ring. With the disabling lesions secondary to the contraction of these cicatrised tissues we are but too familiar : induration of the ring, or of the infundibulum below it, may lead

to simple smooth constriction ; or with concretion, contraction, puckering, calcification, or cohesion of the cusps, to more irregular deformities. Thus the valve may become incompetent, or the orifice contracted ; or these results may be concurrent.

Subaortic stenosis is usually of the more uniform fibrotic kind ; but it may be atheromatous or syphilitic. It is apt to extend into the mitral area, or perhaps to arise in it, and to set up a murmur, if not a regurgitation, in the mitral mechanism. Prof. Osler thinks that this form of stenosis is always an upward extension from the mitral area ; in many cases this is so, but I have seen specimens, and possess one, in which it is confined to the aortic seat, or in which the upper mitral curtain had been reached by a progressive lesion from above. In certain somewhat rare instances an infundibular or subvalvular ("annular and subannular") stenosis coexists with a separate stenotic ring at the valvular level.

The following remarkable case of twofold constriction, annular and subannular, is published by Drs. Langwill and Shennan. The patient was a poorly-developed lad of nineteen. He complained of pains in the chest on exertion, though he had worked as a foundry labourer till four weeks before admission. A strong systolic thrill was felt at the base, and a loud systolic murmur was audible five inches from the chest. The chief cardiac disease found by Dr. Shennan at the necropsy was as follows :—

Right auricle.—Normal ; tricuspid orifice, 1·2 in. *Right ventricle.*—Nothing particular to note. *Left auricle.*—Endocardium somewhat thickened. *Mitral valve.*—Cusps slightly thickened, particularly inner cusp. Musculi papillares small, and fibrous at apex where they join the chordae tendineae. *Left ventricle.*— Walls hypertrophied ; cavity 3 in. long ; thickness of walls varies from 1 in. to 0·5 in. There are a few narrow fibrous bands stretching across the cavity, at whose points of attachment to the wall there is marked thickening of the endocardium from old endocarditis. On passing the finger up towards aorta, it passes through a fibrous ring—0·7 in. diameter—about 1 in. below the aortic opening. This is continued on the ventricular surface of the inner mitral cusp. In this position, and extending upwards from the ring on the lower surface of the postero-external aortic cusp, is a narrow band of comparatively recent vegetations. These cover the lower surface of all the aortic cusps, which also shew fibrous thickening and contraction—cone-diameter of the opening being 0·7 in. Above the valve the aorta dilates slightly—1·2 in.—but in the second half of the transverse part of the arch begins to contract, so that at the upper part of the descending aorta the cone-diameter is 0·6 in.

A similar case, also in a young patient, has been published by Dr. Smart.

Mere stenosis of the aortic orifice, without regurgitation, is said to be a rare disease. Prof. Osler and Prof. Hamilton speak of it as beyond their experience ; Fagge and Pye-Smith as "most rare" ; Fräntzel as "ein seltener Herzfehler." The condition is relatively infrequent, but surely no extremely rare result of chronic aortic-valvular atheroma and

calcareous petrifaction, or again of the mere fibrosis just mentioned. Stenosis usually results from long chronic disease ; and if thus gradual, life may continue, in favourable circumstances, until by petrifaction a dense floor is built up, and the aperture reduced to a chink, the size of a crow quill or less. Regurgitation is thus prevented ; the coarctation of the part may work against it. It is true the more uniform fibrous condensation of the aortic ring with diminishing calibre is rare ; but two pure cases of it I observed for many years, and obtained post-mortem verification ; and in St. George's Museum there are a number of specimens of well-marked aortic stenosis, in many of which the aorta is to all seeming healthy. Indeed, in spite of the high velocity of the " choke-bore," the stenosis may protect the aorta. It is in these cases of fibrous coarctation without atheroma that the effects of aortic disease upon the left ventricle are most clearly seen ; for in them hypertrophy takes its simplest form. In so far as the aortic orifice is narrowed, the inner surface of the left ventricle is protected from " recoil " ; but the contraction-volumes must be increased.

Thickening of a cusp of the valve may lead to the formation of a spur, or vegetation, which, by intruding into the area of the two other shrunken cusps, may accidentally prevent regurgitation. Cases have been described in which such excrescences by their cup-like form seemed to assist a defective valve or cusp to maintain some competency ; but from such rough edges and points " vegetations " are apt to sprout, and to form fringes on the free ventricular edges of the valve, rarely on its aortic aspect, whereby friction is increased and extended. Moreover, consequential chronic inflammatory changes operate on the endocardium, if the diseased valve brushes it, on the valvular structures themselves, and on the corresponding aortic surfaces. Below the valve, streaks or " ripple-marked " thickening of the endocardium on the septum or elsewhere, due to the recoil wave of aortic regurgitation, have been demonstrated by Dr. Glynn and other observers. Hamilton reminded us that, in disease of the aortic valve, it is rather the base of it which is the seat of the mischief, the cusps may even be free ; in the mitral it is the edge of the cusps and their substance which suffer first.

By the above-mentioned adhesion, concretion, and contraction of the limbs of the valve I have said that the orifice may be so narrowed as to convert it into a slit, funnel, or chink. In a case in Addenbrooke's Hospital, under the care of Prof. Bradbury, the adherent margins had united so closely that the blood forced its way behind one of the cusps through a sinuous pipe or crack which, even on minute inspection, almost evaded detection. There was no regurgitant murmur during life, nor was any backward drip detected by Prof. Kanthack's post-mortem tests. In such cases calcification is usually far advanced in the ring as well as in the valve. Many similar cases of extreme constriction have been described; for instance, by Dr. Foxwell.

A grave feature of atheromatous cases is that these changes involve, sooner or later, the orifices of the coronary arteries ; so that the

heart, instead of enjoying that increase of nutrition which its greater work demands, and which at first a hypertrophied ventricle probably supplied, may, after the first stage of the malady, receive by this route considerably less than its normal nourishment ; yet, notwithstanding this deprivation, the muscle may retain considerable functional value, and in some cases, indeed, has been carefully reported as normal. The apparent anomaly is probably explained by the very slow rate of the lesion and readaptation, and by the comparative inactivity of decrepit persons. Thus time is given for the enlargement of other channels of myocardial nutrition such as the veins of Thebesius.[1] In stenosis a prolonged systole is to the gain of the coronary circulation.

It need scarcely be said that the presence of " vegetations " and of other detachable fringes on dog-eared cusps is a matter of far more than local importance, as by them embolism may occur.

Ulceration of one or more of the limbs of the valve is always a perilous process. When dependent upon micro-organisms, and we cannot say how secretly they may enter in, the process may be terribly destructive, as the records of infective endocarditis give us too much reason to know : on the other hand, decay or perforation may be very gradual, and not always due to infection ; in chronically diseased valves the disintegration may be merely mechanical. Perforation of a segment is said to betray itself by a piping quality of the regurgitant murmur. Other rasping or "musical" qualities of these murmurs are attributed to the projection of spurs or shreds of segments which, fluttering or vibrating as the tongue of a reed, give peculiar qualities to the sounds. It is commonly supposed that murmurs may be generated as the blood runs over a roughened surface, as a brook murmurs over pebbles ; this assertion must be taken with considerable reserve : to produce a murmur the column of the blood must be broken, and this a merely mammillated or corrugated surface does not effect unless the eminences be such as to set up vortices around or behind them. The common notion that murmurs may be generated in a rough aorta without any contribution from the valves or orifice ("frottement de la colonne sanguine contre les rugosités"), is improbable, and is not supported by experience. If, in the absence of any cause in the valve, such murmurs be noted during life, they are to be attributed rather to dilatation of the aorta, wherein fluid veins would form between the tarrying external and the more axial layers of the issuing blood. We meet with many cases of advanced and uneven disease of the inner surface of the aorta in which no systolic murmur is audible, at any rate while the patient is at rest.

Again, that there is more than the satisfaction of an anatomical curiosity in the endeavour to fix the incompetence or the obstruction upon this or that limb of the valve, I am indisposed to believe ; no leaf or stump or diseased segment can hamper the access of blood to a coronary artery, unless, of course, it so adhere to the wall of the aorta, or

---

[1] Dr. Beddard has kindly referred me to the article by Pratt on "Nutrition of the Heart" through the vein of Thebesius in the first volume of the *Amer. Journ. of Physiology.*

the mischief so extend from it, as to block the mouth of the vessel permanently. That the deformation of one particular limb of the valve should affect the coronary circulation more than another is impossible; within this area there cannot be differential pressures. That the propagation of a regurgitant murmur, in this direction or that, can indicate the limb of the valve affected, or chiefly affected, is hydrostatically improbable and is not borne out by experience. I have said that in rheumatic fever, in the first instance at any rate, the inflammation usually creeps from the anterior mitral to the adjacent aortic cusp, which is thus affected either alone, or first, or worst; but this cannot be revealed to the stethoscope. In the large majority of cases of simple rupture of a healthy or comparatively healthy valve, one limb only is torn; but a few cases of the rupture of two cusps are on record. The cusp, as we have seen, may be rent on the free edge, but is often torn from its base, at its attachment to the wall.

Of "gouty valvulitis," of a primary kind, after the manner of rheumatic valvulitis, and apart from the chronic subinflammatory and degenerative changes in the aorta resulting from abnormally high arterial pressure or other intercurrent causes, we have no definite knowledge, either pathological or clinical. Certainly we have no pathological criterions of specifically gouty lesion.

A fibrotic or other slow morbid invasion of the aortic area, especially such as we see in gradual stenosis, is prone to implicate the bridge of Gaskell and His, whereby the conductivity of the myocardium may be more or less gravely impaired. This in greater or less degree one sees not very rarely in elderly persons, and the heart will begin to betray it by retardation of rate. There is, as I have said, some evidence that pericarditis penetrating to the valve area may injure it and the tracts of Aschoff permanently by inflammation or cicatricial stenosis (p. 420).

Whether these chronic changes in and about the aortic orifice lead to regurgitation, or to stenosis without incompetency, crucial as the distinction is in clinical medicine, pathologically is a matter of local accident. To test the competency of an aortic valve by means of a column of water, a test which is more useful, it is true, in the post-mortem room than one might have expected, is insufficient in a doubtful case, unless the height of the column of water be equal to the maximum aortic pressure—say of 150 mm. Hg; and even then the water may escape from the coronary arteries. Practically all the water can do is to bring the valves into apposition; the competency of both valve and orifice together must be guessed at. However, the opinion of the pathologists is, that the valve, if perfect, is mechanically sufficient without any shouldering up. In stenosis, indeed, the morbid state of the fibrous ring is such that a muscular cushion could be of little service.

Insufficiency of the aortic valve may come about, though very rarely, not from defect in its own quality, if any, but from dilatation of the aorta, whereby the sectional area of the orifice is passively enlarged. Some

intermittent or temporary aortic regurgitant murmurs may be thus explained. Barié, a careful and experienced observer, has reported thirteen cases of aortic regurgitation from widening of the orifice, without disablement of the valves. Vierordt also states that in weak dilated hearts dilatation of the aortic ostium may cause "relative Klappeninsufficienz." Persistence of the second sound is, of course, no criterion of a competent aortic valve. Records of temporary aortic regurgitant murmur are increasing in number, but still need cautious interpretation. If such cases be followed up, the regurgitant murmur will probably be found at no distant date permanently established, and therefore no doubt valvular ; as in Sir Hermann Weber's very interesting case (p. 469). It seems proved that dilatation of the aortic ring, tough as it is, may occur, but I have never happened to meet with such an increase in the sectional area of this orifice as, without disease of the valve, to permit of regurgitation ; not a few specimens of the kind are, however, to be found in museums. Beneke, quoted by Prof. Osler, calculates that "the aortic orifice, which at birth is 20 mm., increases gradually with the growth of the heart until at one-and-twenty it is about 60 mm. Of this size it remains until the age of forty, beyond which date there is a gradual increase up to the age of eighty, when it may reach from 68 to 70 mm. Thus at the very period of life in which sclerosis of the valve is most common, there is a physiological tendency toward the production of a state of relative insufficiency." But when on the point before us, I turn to Prof. Osler himself, I find that "relative insufficiency of the sigmoid valves due to dilatation of the aortic ring is a rare condition" ; he adds, "Indeed I have myself never met with a pure instance of the kind, in such cases I have always found the valve segments involved with the arterial coats." Rosenstein, in Ziemssen's *Encyclopaedia*, had never seen incompetency by dilatation only. Moxon, however, and Balfour, held that "relative insufficiency" may occur. As aortic insufficiency is not eminently a disease of old persons, but rather of persons about or under middle age, we readily notice cases of aortic regurgitation due to senile arterial disease,—I have now such a case under my occasional observation, —yet we know that the prevalent effect of aortic disease in the old is "obstructive." Again, although in elderly persons, and in younger men the subjects of syphilis, we meet with considerable and even enormous dilatations of the aorta, yet even in these cases aortic regurgitation does not generally appear, unless there be disease of the valve itself also ; the orifice, at any rate in and after middle life, seems prone rather to harden than to expand to the point of insufficiency.

However, that regurgitation, permanent or temporary, may arise directly out of a mere dilatation of the aorta and its orifice, though a rare event, seems to be no longer doubtful ; but in such cases the incompetence is probably nominal. In Tigerstedt's opinion the semilunar valves are efficient under conditions of considerable relaxation, and experimental evidence shews that in case of slight defect the limbs of the valves aid each other by mutual readjustments (pp. 5 and 434). Dr. Newton

Pitt, who has investigated this point, verified and added to the records of cases of regurgitation by dilatation of the first portion of the aorta, without defect of the cusps. Dr. Pitt gives 8 cases—4 from post-mortem records, 4 from specimens in Guy's Hospital. Of the 4 necropsies: in (i.) the first portion of the aorta was much dilated, but the cusps could scarcely be called efficient; in (ii.) the first portion was also much dilated and the cusps stretched, they were not shrunken nor diseased, but made shallower by tension; in (iii.) the cusps were "fair," but taut and did not close the orifice; in (iv.) M. aet. 29, the aorta was much diseased and dilated (syphilis?), but all the cusps were normal. In all 4 a loud to-and-fro murmur had been noted. The museum cases were much to the same effect. Barié says that when in dilatation of the aorta in arteriosclerosis the regurgitant murmur arises, a "tympanitic" second sound persists. A case described by Dr. Whitby in a patient with stenocardial oppression, suggests that high arterial pressures alone may force the valve:—M. aet. 59; no alcohol, no tobacco; pulse 65; "tension markedly high"; aortic second sound very loud at apex and base. Blowing diastolic murmur down sternum. An attack of influenza with pneumonia supervened. During the fever (101°-103° F.) "the pulse became continuously compressible," and the aortic murmur wholly vanished. That this was not due to a weakened ventricle became evident, as with convalescence and renewed appetite the high pressure was re-established, but without the murmur which, during Dr. Whitby's time of observation, did not recur. The stenocardia, however, pointed to disease in the aortic area. Edwards in a careful survey of the evidence, concluded that relative valvular insufficiency, though very rare, does nevertheless happen. Hamilton and Byers record a case of it in which both aorta and valves proved to be practically normal in form. Dr. Keith writes to me that "the subaortic musculature, although neither circular nor even semicircular in arrangement, does during systole so contract as to render narrow the subaortic lumen of the ventricle; but, as the valve is competent, whether the heart be in systole or diastole, their action seems to be entirely mechanical." But, he adds, in advanced arteriosclerosis this musculature, especially in the upper part of the ventricular septum, is extremely prone to atrophy—much more so than the rest of the heart; so that from a thickness of 12-15 mm. it falls to 5-10 mm. Thus he suggests, as has been suggested before, that the aortic orifice can be widened by ventricular dilatation, so that in arteriosclerosis aortic incompetence may occur with a sound valve. A small aneurysm, intrapericardial, or just above a cusp, may, of course, by deformation of the orifice, set up relative incompetence. These conditions are, however, so rare, that in current diagnosis a diastolic aortic murmur is presumably indicative of valvular defect.

Sometimes, as Corrigan shewed, on examination of the aortic valve after death, from whatsoever disease, its segments are found atrophied; the flaps are thin, and not infrequently fenestrated in a line parallel to the free edge, and above the line of contact. These conditions are not necessarily morbid or mischievous; and fenestrations if on

the apposed margins of fairly adaptable cusps, do not give rise to regurgitation.

Aneurysm of the parts about the valve is dealt with in the article on Aneurysm (p. 620).   Morbid growths, polypus, and the like about the orifice, are pathological curiosities.

The effect of aortic disease on the other valves and orifices has been studied by Hamilton. Aortic regurgitation, as he remarked, is often "anticipated in its injurious results on the other orifices by its own peculiar sources of mortality."   From his measurements, however, the following results appear : namely, that, unless in addition to the incompetence of the valve the aortic orifice be dilated, "the effect upon the size of the other orifices is nil ; if, however, the aortic orifice be dilated, a general distension of all the other orifices is apt to follow." "Constriction of an incompetent orifice, then, exerts a salutary effect "—so far, that is, as stress on the other orifices of the organ is concerned.   I have put it broadly already that positive stenosis and regurgitation stand thus in some inverse functional relation.   Such is the capacity for re-adaptation in injured parts, we should expect the aortic mechanism to preserve a mediation as exact as may be possible to degenerate tissues, between the altered diameters of ventricle and aorta ; whether it be that or this cavity which is dilated.

In diseases of the aortic valve, as of other parts of the heart, our attention may be too much given to murmurs ; the working calculation which we have to make is the effect of the lesion on the chambers ; for by their efficiency the organ stands or falls, at any rate for a time. In early pure stenosis the left ventricle may approach that mythical type "concentric hypertrophy," and by it the chamber may be protected from the recoil which, in aortic regurgitation, is said to be very damaging. In regurgitation, especially if attended, as it is wont to be, by dilatation of the aorta, the ventricle is at least as much dilated as hypertrophied. The pathogeny of this coincidence has been much discussed, and it is the general opinion that this dilatation is due to the recoil of blood from the aorta upon the wall of the ventricle during diastole. Besides the resistance-head in the arteries, that fraction of the force of the systole which is stored up in the diastole of the still more or less elastic aorta is expended not only upon the forwarding of the blood, but in some part also upon the inner surface of the ventricle, with a physiological effect we shall note presently. It is often taught that the dilatation is due to the filling of the ventricle from two sources ; but, if we neglect some loss of energy in vortices, it cannot matter whether the cavity be filled from two sources or from twenty ; the matter is not one of the accessibility of blood only, but of the resultant stimulus or stress of intra-ventricular pressures. The aortic pressure is so much greater than the auricular that this may count for comparatively little ; yet the resultant pressure is not the sum of the two, and the resistance of the aortic stream, being greater than that of the auricular, will head this back more or less, perhaps partially closing the mitral valve. From experiments

upon animals it seems that, on suddenly produced insufficiency of the aortic valve, the aortic pressure may be so great as even to rupture the unprepared ventricle. But in a case of sudden rupture, by violent effort, of the posterior pillar of the lower mitral cusp (vide *Lancet*, 1908, ii. 175), with intense dyspnoea and death in a few days, the blood-pressure records were no more than 130-140 mm. The distress felt on rupture of the valve in a straining man may be due to distension of an unprepared ventricle. In more gradual disease, however, tone and " reserve capacity " sustain the arterial pressure till the ventricle can grow up to the new call upon its strength. If it rupture it will give way at its weakest point ; but to speak of the regurgitating stream " impinging on the inner surface of the apex of the left ventricle," and " of re-peated blows of a jet of blood disabling the ventricle," is to regard the cavity as if it were the pan of a water-closet, and to lose the conception of the heart and arteries as a plenum. The heart is to be regarded as a screw, with tone and reserve driving-power both dynamic and static at command ; and if so even without the valve the work may still be done. Indeed for an indefinite interval, during which the patient is often unaware of any defect in his circulation, the work often is well done. The failure comes about partly because re-adaptation can never be com-plete (p. 441), partly because of the periodic excesses of pressure of aortic blood over auricular ; were the auricular blood impelled under a pressure more equal to that in the aorta the valve might be more dispensable. The dilatation is due, then, to continuously excessive aortic pressure. If from the ventricle of a frog, beating in a tonometer under a supply of blood from a pressure-bottle at varying heights, curves are taken to measure the volumes of the ventricle, as long as the pressure from the bottle remains constant, so long the line of the volume at diastole will stand remarkably level. But if by raising the bottle we increase the pressure slightly, the diastolic line immediately sinks, shewing greater capacity ; even though the height of each systole may be as before (Gaskell). The distensile force being greater, the corresponding increase in diastole follows a well-known physiological law (*vide* Tigerstedt (121)). How far, however, this distension may be also physiological and adaptive we shall consider presently.

Dilatation accompanies hypertrophy, as Roy, Starling, and others have clearly put it, because, although a loaded may do more work than an un-loaded muscle, the amount of contraction (that is, the height of the lever) is less. The cardiac muscle may be more tense, and the contraction, therefore, more powerful, but not so as to occupy all the increased length of the muscular fibres ; thus some dilatation remains, the residual blood is more, and the output less. On the next diastole the heart is overfull, but even under this increased stimulation only the normal inflow may be sent out : thus arterial pressure is kept up, but the stimulus and the work are increased, and hypertrophy will follow. Not only has some of the output to be lifted again, but also the inertia of the relapsing blood has to be converted into forward motion. It is often said that the heart

attains a larger bulk in aortic regurgitation than in any other disease ; even to 2-3 cm. at greatest thickness, and 1-2 cm. at apex; and to weights up to or occasionally exceeding 30 ounces.   The papillary muscles grow much larger and flatter.   This is true ; but in chronic Bright's disease the " cor bovinum "—the " heart of a pantophile," as Voltaire called that removed from Diderot's body—may attain to a still greater bulk.   It is usual to speak of this enlargement as a compensation of the defect it counteracts.   There is no great objection to this expression if it be remembered that it is a figurative one ; all we know is that within limits dynamic is followed by static increase ; it is not in physiology only that function takes form in structure : yet if in respect of one factor the difficulty is thus postponed, the re-adjustment cannot be universal ; a broken concert must bring evils somewhere in its train ; were it not so we should have another system as efficient— that is having as wide a potential—as the normal, as that developed on evolutionary lines : " which is absurd."   We shall not say then that " Nature provides a compensatory hypertrophy to the walls of the heart in order that, etc." ; but avoiding teleological connotations, we shall regard the hypertrophy of the ventricle simply as the static expression of increased function in a certain direction—that is, a larger contraction-volume—whatever the consequences.

The dilatation of the ventricle is, then, of two kinds, however diffi-cult it may be to discriminate them in particular cases ; namely, the adaptive and the atonic.   In the mode whereby the ventricle opens, as it were actively, to receive the larger loads, there need not be—at the best there is not—any loss of tone.   The cavity, by some vital endow-ment beyond elasticity, unfolds to receive the excessive quantum without any rise of intra-ventricular pressure, or push against the left auricle.   In animal experiment, to touch the inner wall with the catheter, without interfering with the valve, will cause such an expansion.   It is true no doubt that, in animal experiment, on such ventricular expansion, even if only regulative, arterial pressures fall ; and so far experimental and clinical facts seem to be contrary.   Von Basch finds the reconciling factor in arteriosclerosis, as itself a cause of rising pressures.   But the facts are true in stenosis without arteriosclerosis, as I verified in one case of pure stenosis without arterial disease; and also in the kinds of arteriosclerosis I have distinguished as toxic and decrescent, kinds in which pressure is not necessarily or usually much enhanced, if at all.   My interpretation is— as in many such ambiguities—that in disease, as compared with experi-ment, we have also the important factor of time—of changes gradual enough to solicit the corresponding re-adaptations.   To maintain a larger mean diameter may imply, with the hypertrophy, a reinforcement of tone ; but, as the argument proceeds, we shall see that when in fatigue tone begins to fail, the other or passive mode of dilatation supervenes.

In what fashion otherwise does this adaptation break down ? Speaking generally, the harmonic constituents of the rhythm cannot come again into perfect tune.   All muscular overgrowth may be

transitory, for extraordinary conditions are less stable than the normal; the normal arrangement is that which has proved most stable under prevalent contingencies. The hypertrophied biceps of the file-cutter is said to fail after a certain number of years. Again, disproportionate increase of one part of a system alters the relations of all reciprocating parts, and the system begins to rock: in swift passenger-ships hypertrophied engines mean a shorter life for the ships. Thus the excessive inertia of too big a heart will wear associated parts, not all uniformly reinforced. We have seen that, near the heart, the aorta under the immoderate stress is strained; it dilates, and atheroma, the effect of strain, will be found above the valve, where it may readily implicate the mouths of the coronary arteries. From the first perhaps these arteries, like the rest of the tree in aortic insufficiency, suffer under the excessive percussion and the extreme variations of pressures. I do not attach much weight to differential pressure, as in the main the muscle is nourished, not by throwing blood at it, but by its own metabolic activities. Moreover, the sinuses of Valsalva, so long as the parts are whole, support the blood at the mouths of the coronaries. The respective gain and loss between systole and diastole varies with the cardio-motive energy, so that, although recent researches tell us that in regurgitation diastolic pressures are often more than maintained, yet areas of the cardiac muscle may come short of blood; fatigue is cumulative, and fibrous tissue, which is more economical to feed, and has a high elastic coefficient but diminished resilience, supplants the active muscular fibres (Dehio and others). The Leipzig school of Curschmann, Albrecht, Krehl and others has demonstrated to us the frequency of unsuspected areas of indolent subinflammatory or necrotic changes in the myocardium in valvular diseases; and attributed to them very justly a considerable part in the cardiac failure at later dates. Such lesions are probably the chief factors in comparatively early breakdown; but if they be, as often they are, in relatively small and scattered spots or patches, I am disposed (on my own investigations with Kanthack) to believe they heal soundly by scar-fibrosis, and, if so, leave the heart but little, if at all, the worse. In many a case of old valvular disease the heart-muscle proves to be substantially sound. If the aorta be unhealthy to begin with, these disintegrations come about so much the sooner, as the storage of energy in aortic diastole becomes less and less. Mere nervous re-adjustments are but of temporary value; when all nervous connexions have been severed the reserve is still considerable.

Then there is the effect of a persistently large residuum of blood in the left ventricle on each contraction. If in health there is always some residual blood—some margin, however minute, between contraction-volume and output,—how much more must this be the case as the work of the ventricle, distended under high pressure, is increasing beyond limits, as the cube of the radius of curvature. This consideration alone, when we recollect the cumulative effects of fatigue and the many incidental causes of atony of the heart, may go far to account for the failing of stability. On the

other hand, by the expansive vasomotor reflex in aortic insufficiency the ventricle is relieved by a low peripheral resistance, whilst in stenosis it works against a high resistance ; the output must then be far larger in the former, as indeed the upstroke of the sphygmograph partly indicates.   A protraction of systole in stenosis, if any, means longer working hours at the expense of rest.   Again, in the normal state the blood-pressure falls in the ventricle suddenly, in the aorta gradually : in regurgitation it falls suddenly in both cavities ; in insufficiency the pulse-rate is accordingly more frequent.   Experiments upon animals, even if so gradually performed as to establish cardiac hypertrophy after aortic regurgitation, shew that even in them—as unquestionably in man—individuals differ widely in potential of cardiac reserve and resistance.   Yet in all cases the normal heart (under experiment) can achieve more under artificial increases of arterial pressure than the hypertrophied heart with aortic regurgitation ; it is only within narrower limits that these hearts can cope with artificially increased arterial pressures as well as the sound ones.   We have noted already that the hypertrophied myocardium is altered in texture ; more or less fibrous tissue is to be found in it.   This fibre may serve a useful purpose in supporting the tone of the muscle, as its elasticity is higher ; but as it lies within much narrower limits, a shorter radial extension will strain it.   That dilatation is the feature of insufficiency rather than of stenosis would indicate that mere residual blood is not the predominant factor in dilatation which is usually supposed ; it is when tone and cardiomotive energy are beginning to fail that residual blood becomes so grave a condition of dilatation.   Dr. Gossage states that tone may fail while other cardiac properties are constant, or some even enhanced.   In regurgitation abatement of negative pressure in the ventricle may be of some disadvantage ; and, finally, in this state stresses tell on the ventricle when this muscle is relaxing, in stenosis when it is contracting ; so in this respect also when regurgitation and stenosis occur together, stenosis may have some protective effect.

However, if, by some transient cause such as acute rheumatism, the aortic valve be injured alone, and not severely, in the midst of a healthy body, we may not detect, even after long persistence of the defect, any implication of other parts, except a condensation of the mitral cusps under the effects of the ventricular hypertrophy.   Ultimately, however, if life be not cut short by a syncope, the mitral valve will give way under increasing dilatation, and the patient become exposed to mitral rather than aortic consequences.   It has been said that forcing of the mitral orifice with moderate regurgitation gives relief to the overwrought arterial circulation ; such an effect it may have for a while, but it is the opening of one more of the gates of death (*vide* art. " Mitral Disease ").

The pathological changes in the arteries, due to the excessive stresses of the heavily beating heart, have not been carefully compared, in uncomplicated cases in young persons, with the arteriosclerosis of later life and of obscurer causation.   The research is well worth making.   In regurgitation the thoracic aorta itself assumes a somewhat sinuous curve from the

commencement ; this may be due to cardiac displacement, for its coats are said, in these young cases, not to deteriorate for some little time. Every householder has heard the water-hammer sound on sudden closure of a cock.   By these concussive stresses the pipes are soon injured, and, to meet them, engineers put in large margins of strength.   Now cast-iron pipes which, like rigid arteries, do not absorb these oscillations, suffer more, so that now a mild steel is used which, like resilient arteries, recovers form without strain.   But it is not to be supposed that this consequential arterial disease is an important factor in any rise of arterial pressures.

In senile aortic disease, emphysema and other cognate modes of decay too frequently increase the burdens of the patient's latter days.

STENOSIS.—**Symptoms and Signs.**—The invasion of stenosis, as of regurgitation, is often latent.   Even if organic, aortic systolic murmurs signify no more than a deformation of the orifice, whose sectional area may or may not be diminished.   In many cases in which this area is positively diminished the valve is also incompetent, and the case is no longer a simple one.   To understand stenosis we must study it in its unmixed form ; especially in those rarer cases of fibrous constriction which, creeping on for many years, begin at no very advanced time of life.   For instance, Mr. X. became aware of an oppression in the chest.   A direct aortic murmur was found with hypertrophy of the left ventricle.   The patient was about fifty years of age, of correct and domestic habits ; he had never suffered from rheumatism, his life had not been anxious or laborious ; there was no history nor evidence of syphilis.   Nor was there any sign of kidney disease or of general arteriosclerosis ; apart from the aortic defect, his arterial system seemed to be no older than his years.   Sir William Gull came to meet us, and often I recall him as he described with his finger an imaginary cardiographic curve—the portentously long upstroke, as the heart heaved like the back of some imprisoned monster ; then the curt diastole with faint second sound ; the protracted pause, as if the heart were slowly gathering itself together for another effort ; the slow deliberate rhythm, some forty in the minute, in which each reluctant beat, stout as it was, seemed as if it might be a last effort.   In these cases the muscle-fibres are slower in reaching the position of full contraction.   In certain experiments (Gael and Ludowitz and others) the protraction ranged from 7 to 30 per cent (more in rabbits than in dogs).   Apart from any invasion of the bundle of His, the slowness of rate necessary to compass as much output as possible was well illustrated in another case of this kind, reported by Dr. S. West, in which the pulse was 30 ; and in another by Dr. Parkes Weber (128), in which the heart's beats became so slow as to give rise to syncopic attacks ; but as this fibrous thickening, while it slowly contracts the orifice, is prone to invade Aschoff's tract, the bradycardia in these cases may have been due in part to tardy conduction.   In another pure case of the same kind which I watched for some years, a fibrous

stenosis (verified by necropsy), for many years (twenty years at least) presented the signs of stenosis only, the pulse-rate retrograding slowly. During this stage the health did not suffer ; the heart was large and the work well enough done. But during the last ten years signs of invasion of the bundle of His gradually appeared, when the ventricular rate fell to 30, 20, 12, and even to 8. As the pulse receded below 30, the whole Stokes-Adams series was manifested.

The cardiac output in old persons may be much less than in a vigorous middle-aged subject such as Mr. X. ; nevertheless, as we have seen, even the hearts of old people can attain to no inconsiderable amount of hypertrophy ; the old woman referred to on page 434 had a heart of 24 ounces, and of sound muscle. The channel in many senile cases is so strait that the velocity necessary to keep up an aortic pressure sufficient even for the narrow life of old folks must be enormous.

Although it is true that the left ventricle does not dilate in stenosis as it does in insufficiency, yet it is untrue to say that it does not dilate at all ; for the residual blood becomes considerable ; the auricle probably

Fig. 45.—Sphygmogram. James D., aet. 46, rheumatic fever at aet. 7. Loud systolic murmur in aortic area ; no diastolic murmur, no second sound. P.M. No incompetence. (Graham Steell.)

gains a little in strength to meet the increased pressure in the ventricle, but the contraction-volume of this chamber also is excessive. Restricted as the output may be, by increase of velocity the arterial pressure is maintained ; and as the pulse is usually infrequent, the systole is not only strong but also absolutely, not relatively, protracted. It seems almost certain, as Dr. L. Hill has stated, that the length of the cardiac systole may be of very various proportion to the rest of the cycle, but beyond this we have little information. I appealed to Dr. Lewis on the subject, who kindly informed me that although this point had occupied his attention a good deal, hitherto he had been baffled in his attempts to obtain quantitative knowledge. The net output may be less, but the arteries will contract upon the smaller content. The aorta, therefore, if dilated, is not dilated as a direct consequence of the stenosis ; but by a dilatation of the ascending arch the reduction of the abnormal velocity through the narrowed aperture—the *vena contracta*—would be promoted. This point has not, I think, been investigated.

The second sound will vary with the state of the tissues around the orifice and of the valvular segments ; if these be hardened, or the muscular cushion attenuated, the sound may have the "parchment" character ; but it will always be short, as the blood-pressure above them has not a high maximum ; and, unless the vessel be drawn nearer to

the sternum, it will not be loud, because the sectional area of the orifice is diminished ; and by certain condensations of the parts it may be muffled.   By this waning of the second sound at the aortic base we may distinguish stenosis from cases of high pressure with systolic murmur. The sound may indeed become inaudible, as it did in J. D. (Fig. 45), and yet may reappear.   The contrast between the big heart-beat and the small pulse is often remarkable, and in this respect stenosis is contrasted with regurgitation, wherein the pulse - wave is of brief duration — "collapsing"—but has a very high maximum.   In pure stenosis we have with a large heaving left ventricle an infrequent and tardy pulse, a low maximum wave unaffected by raising the arm, and peripheral arteries contracted upon a smaller content.

The murmur of stenosis may be heard widely, and over more than the area of the heart and larger arteries ; it is often loud at the apex, and in the interscapular region over the aorta.   It may be extremely difficult, in some cases perhaps impossible, to decide whether a certain systolic murmur is of mitral, or of aortic, or of mixed origin.   To this point we shall return presently.   Weiss and Joachim, by registering the vibrations, found that the maximum of a mitral systolic murmur begins instantly on the systole, that of an aortic systolic a trifle later ; occasionally, as we shall see, much later.   With this our aural impressions agree.

The character of the murmur varies to some extent both in quality and in order.   Sometimes its sound-vibrations are attended with others less numerous, not rapid enough to cause a sound but perceptible to touch, as a *thrill*.   Such coarse vibrations need not indicate extreme stenosis, but rather suggest some reed-like formation.   The cusps are too thick to float in the current.   In mitral stenosis the thrill is about the apex, in aortic stenosis usually about the base, but it may be more distinct at the apex.   A thrill perceptible over a large part of the cardiac area is present in open foramen ovale ; occasionally in pericarditis ; and when, as in arteriosclerosis, the vessels are rigid while the heart itself is vigorous and the blood-pressure is maintained ; but, in the absence of aneurysm, a thrill more or less limited to the base is almost pathognomonic of aortic or pulmonary stenosis, whether alone or in combination with other disorder.   A thrill depends a good deal upon the vigour of the heart ; with a strong heart and a low tone of the vessels it may be felt in the arteries of the neck.   Accordingly we expect that the murmur also will, in part at least, be compounded of slow sound-vibrations ; whether "musical" or not, it will be noisy— sawing, rough, or harsh.   If the ventricle gives way under its toil, the murmur may grow softer, possibly even to extinction ; and mitral regurgitation may add its own systolic murmur to the discord.   Or the disease by extending to the mitral may cripple it ; or under digitalis the murmur may alter in quality, as the pulse may even quicken in rate ; then again, as the pulse slows down, the harshness of the murmur may return.

As regards the order of the murmur, it is generally a moment later

than a mitral regurgitant, and seems sometimes indeed, if alone, to take a postsystolic rhythm; a delay probably due to a prolonged prosphygmic period. In a certain private patient, the first moment of systole, as judged by the carotid, was free from murmur; then followed a very brief murmur, and instantly thereafter a second sound clear of murmur, though not normal in quality. I may repeat that the second sound tends, in contrast to that of high aortic pressure, to diminish. Anacroty may be noted in this rhythm. I am interested to find that Vulpian also reports such a murmur so placed :—

A woman, aet. forty, suffered severely from acute rheumatism; two years later she presented herself with mitral regurgitation revealed by the ordinary signs. At the base a roughish bruit was also heard; this basic bruit was placed "between the two normal sounds." The murmur was heard also at the mid-precordial region, and upon the localisation of a rough short systolic murmur. The pulse was regular, small (rate not given), "et un peu concentré." The ascending sphygmographic line was "ill-marked."

Vulpian was bold enough to diagnose thereby a contraction "sous-aortique," "une lésion de canalisation . . . à une certaine distance au dessous des valvules aortiques." He does not give his reason; it may lie in the increase of velocity as the ventricle contracts. Such a late element of the murmur may be due to fluid veins within the arch of the aorta, for a similar effect may be heard occasionally in high arterial pressure without narrowing of the orifice. To speak generally, however, the murmur is a long and rising one, occupying the whole of the first phase up to diastole. A presystolic murmur in this place has been attributed to stenosis, as by Lemoine (quoted by Sansom). Sansom characterised it as a murmur coincident with the earliest ventricular effort; but in stenosis the prosphygmic interval is abnormally prolonged, and may indeed become perceptible to the touch.

The propagation of the murmur from the right second costal cartilage depends much on the stage and conditions of the disease. If the murmur be loud—it may be loud enough to be heard at a distance from the chest—its area of diffusion will be considerable, both about the basic region and towards the periphery in the arteries. Thus it may gain an ascendancy over other murmurs, and quite possibly, by interference-vibrations, alter or even resolve them. When stenosis is extreme it is said that the murmur may fail to reach the carotids; as the walls of these vessels are not so relaxed as in regurgitation, they vibrate less readily.

I have alluded to equivocal aortic murmurs, more or less simulating those of mitral disease. They may be divided into those suggestive of mitral regurgitation, and those suggestive of mitral stenosis. These will be dealt with under aortic regurgitation; of those which simulate mitral regurgitation, four examples were brought forward by Dr. Dickinson in 1897. In them, although after death the aortic orifice in each was found in advanced stenosis, a systolic murmur was heard at the apex,

such that mitral regurgitation could not be decisively excluded.. These murmurs, as observed by chance in hospital wards, I have been wont, too confidently perhaps, to refer to the aorta. In two cases in which I hazarded this opinion it was borne out by necropsy ; there was no mitral insufficiency. In one the murmur at the apex was musical, and a musical murmur is generally aortic ; in another the murmur was audible an inch away from the patient's chest, and a murmur so audible is surely aortic. In a third case a thrill was palpable at the base. Aortic systolic murmurs are audible behind ; but in the cases I have seen such murmurs were not confined to the axillary and infrascapular regions, but were audible universally—*passim* not *ordinatim*. If there be no insufficiency to reduce the prosphygmic interval, the aortic murmur starts slightly later than the mitral, and the aortic is a rising, the mitral a falling murmur. Again, in uncomplicated cases, the pulse is not " mitral " in behaviour ; the radial volume may be small, but the rhythm and output are uniform, and the small vessel may be of good tension. Again, a systolic murmur of aortic origin is generally propagated into the carotid, a mitral murmur is not ; and by mitral insufficiency the first and subclavian tone is reduced. Even with twofold murmurs of equivocal kind, the quality at base and apex is often different enough to enable us to discriminate the direct murmurs in each situation. " *Pulsus tardus* " and an anacrotic tracing would testify to aortic stenosis, though it would not exclude a coincident mitral incompetency of moderate degree. In atheroma a murmur of stiffened machinery, without characteristic general symptoms, is often, it is true, of relatively little importance ; but the question whether, at a declining stage of aortic disease, mitral regurgitation is or is not setting in, is a grave dilemma. Then probably the late modification of the murmur, the history of the case, and the course of the general symptoms will not fail to guide us to a correct diagnosis.

The pulse, in moderate degrees of aortic stenosis, may not shew any abnormal features ; big as the heart is, the pulse may begin to diminish in volume, yet, as contrasted with a mitral pulse, it is regular and sustained in all positions. As the stenosis increases the waves will become more infrequent and tardy. As the systole is protracted both limbs of the curve will become more gradual. In the aortic direct murmur of arterio-sclerosis, without constricted orifice, and with a comparatively open periphery and a powerful heart, the ascending limb will be steeper, so that the delay will lie not with the arrival of the wave, but in the completion of the "plateau."

Sphygmographic Signs.—The principal feature is then the tardiness of the curve, not on the systolic limb only, but upon both ascent and descent. The anacrotic and the bisferiens pulse have engaged attention from Mahomed's time to the present day ; and it has often been said that the anacrotic pulse (Figs. 46, 47), which in a well-marked case may be per-ceptible to the finger, is so definite a peculiarity of aortic stenosis as to be pathognomonic of the condition. Characteristic it is, but we find the

pulse anacrotic in so many different modes of cardio-arterial disease that it is safer to say that it indicates some more common factor which in aortic stenosis tends to predominance. Moreover, transitional curves are obtainable between anacrotic, bisferiens, flat-topped, and high-pressure pulses (Lewis). Furthermore, anacroty is often not consistent in the same case. Dr. Graham Steell, to whom we owe many careful observa-

Fig. 46.—Anacrotic pulse, sloping upstroke ; apex of curve formed by tidal wave ; ill-marked dicrotic wave. Man, aet. 29, with rheumatic history, loud systolic murmur and thrill in aortic area. (Graham Steell.)

Fig. 47.—Florence ——, aet. 28, rheumatism. Loud systolic and diastolic murmurs in aortic area. Death from cerebral embolism. Anacrotic pulse with bigeminal or alternating rhythm, possibly due to digitalis, although none taken for two days. P.M. (Graham Steell.)

Fig. 48.—Anacrotic pulse-tracing from case of mitral stenosis without aortic stenosis. P.M. (Graham Steell.)
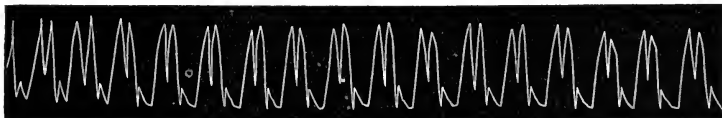
Fig. 49.—Margaret G., aet. 25 ; rheumatism, aet. 14. Exemplary P. bisferiens. R. radial, double beat plainly felt ; L. radial, ordinary tracing of aortic incompetence. Loud systolic murmur and thrill in aortic area ; diastolic murmur. P.M. Stenosis of aortic orifice with incompetence of valves ; no explanation of difference between radial pulses. (Graham Steell.)

tions on the subject, kindly allows me to reproduce some of his tracings of the anacrotic and of the bisferiens pulse. Dr. Steell recorded the behaviour of the pulse in four cases in which this formation was interpreted by necropsy. He concludes as follows : " Three of the four cases bore out the belief that the anacrotic pulse is a valuable sign of aortic stenosis, provided the physical signs correspond. The fourth case taught, however, that pathognomonic value must not be attributed to this pulse, inasmuch as other conditions besides aortic stenosis may

produce it (Fig. 48). Moreover, in cases i. and iv. the pulse was not constant in this character ; in case iii., however, unalterableness of the pulse was a striking feature of the most definite case of all, inasmuch as it was the least complicated. Such unalterableness of the anacrotic pulse is probably of great diagnostic value, although it may be rare."

Sansom came virtually to the same conclusion ; he emphasised the deduction that a persistently anacrotic pulse means disease of some kind, whether it be an aortic lesion or a persistently excessive arterial pressure ; and that in case of doubt an anacrotic pulse might signify a systolic murmur at the base of the heart to be organic. That the anacrotic wave is of central origin is pretty certain. To illustrate this, I insert the following curves (Fig. 50) by Bayliss and Starling. I may repeat that in aortic stenosis, at the first moment of the opening of the valve, the blood issues readily ; but as the stenosis throttles the wave the increased velocity of the blood is often more than counterbalanced by the slackening current and rising pressure in the aorta, so that the farther delivery becomes slower and more laborious ; though, so long as the heart is strong, the pulse is regular. The common factor which determines anacroty [1] seems then to be that during systole the flow from the aorta to the periphery is at a slower rate than that from the ventricle to the aorta. In aortic stenosis the current issuing from the choke-bore is of a relatively high velocity ; that from the aorta to the periphery, however, slackens as the blood occupies the relatively large continent. If, as I have suggested, an important factor in falling velocity is dilatation of the ascending arch, anacroty may be produced by such dilatation, and sometimes I have conjectured that the initial wave might be due to contraction of the auricle upon an overcharged ventricle. In high arterial pressures, on the contrary, peripheral resistance raises the aortic pressure against the ventricular ; in a yielding heart then, for instance in a later stage of chronic nephritis, aortic pressures gain upon the lagging cardiac energy ; for in arteriosclerosis it is, of course, the wave, not the current, which is accelerated.

Sallavardan asserts that anacrotism is an artefact, produced by a degree of pad-pressure which reduces the first limb of a bisferiens pulse below the second. Increased pad-pressure may make a slight anacrotic wave more evident (Lewis) ; but I have found that in high-pressure pulses, without stenosis, even an apparent anacrotic form is not easy to obtain so long as the heart is coping with the resistance. Dr. Lewis has demonstrated how, by raising the peripheral resistance (*e.g.* in the radial by compression of the hand or of the abdominal aorta), a genuine anacrotism can be produced. At a moment of check to the flow the lever is ahead of the wave. A gradually increasing obstruction, positive or relative, in front of a wave must flatten its top, and this is a sort of prolonged anacroty. Dr. Lewis reminds us that the aortic ring may

---

[1] Anacrot*ism* or dicrot*ism* means not the phenomenon itself, but, more abstractedly speaking, the conditions or process of which it is a character. Thus in French we read of dicrot*ie* and dicrot*isme* respectively.

soon become too rigid to mould itself to the stream, and if so will thwart it. We should expect, then, to meet with anacroty when, resist-
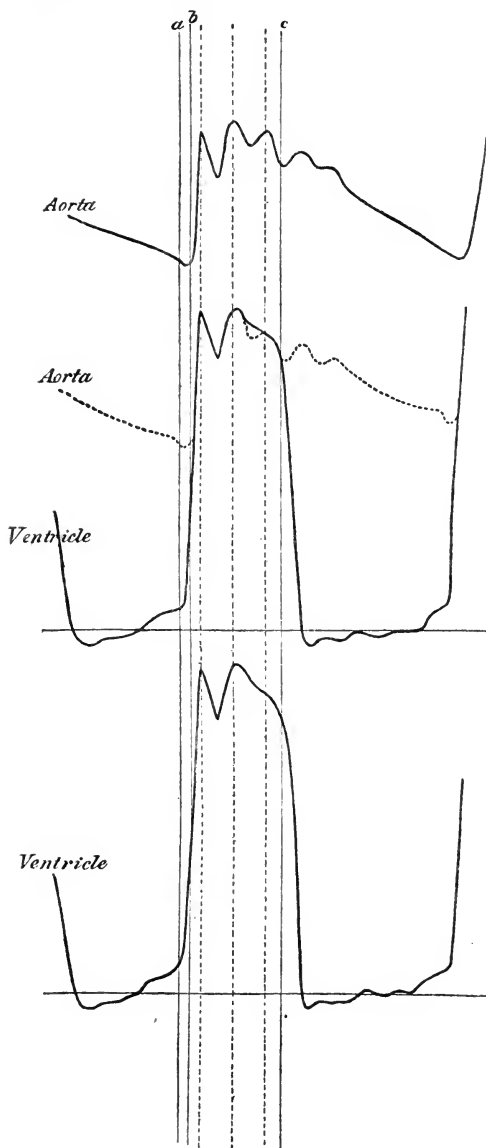
FIG. 50.—The first two waves of the ventricular plateau coincide exactly with the first two aortic waves; the third corresponds in position, but is rather higher on the aortic curve. The descent of the two curves corresponds to the point of closure of the aortic valves (line *c*); the ventricular curve continues to descend; the aortic curve is raised again by the dicrotic wave. The interval *a* to *b* is the prosphygmic interval ("Anspannungszeit"). (Bayliss and Starling.)

ance being normal or plus, the heart is failing (Sansom, Lewis). If, therefore, in such a case we note anacroty without stenosis, we may

forbode thereby a failure of compensation. On the whole, then, as Sansom surmised, anacrotism is of no good omen. It is interesting to add that von Frey (quoted by Lewis) noted it in a heart escaping from vagus inhibition.

Of the pulsus bisferiens (Fig. 49) Dr. Steell says, that although cases of stenosis are often associated with regurgitation, it is not easy to find material on which to make conclusions regarding pure stenosis yet on the authority of Mahomed and others, we may assert that the pulsus bisferiens is consistent with pure stenosis. In Mahomed's two cases of pulsus bisferiens there was some regurgitation also ; but in Steell's and Lewis's cases the phenomenon was unequal on the two sides, in one of them mainly unilateral ; the other radial assumed the character very occasionally and imperfectly : a careful examination of the arteries concerned afforded no explanation of this peculiarity. In two other cases, moreover, the phenomenon was manifested in the one on the right side, in the other on the left. The pulsus bisferiens will be considered again under the head of regurgitation. Dr. Steell concludes thus :— " In stenosis we are unable to explain the mode of production of these pulses ; and I do not think we are warranted in affirming that either the anacrotic or the bisferiens pulse is the direct result of aortic stenosis ; both pulses are found, however, so often in association with aortic stenosis that we cannot deny them diagnostic value ; of the two the anacrotic pulse probably possesses the greater diagnostic value."

Pain, distress, disturbed rhythm, oppression are not frequent features of gradual aortic stenosis or obstruction about the orifice, unless the supra-valvular area also is engaged in the subinflammatory process, as is frequent in syphilis of the part, and not uncommon in senile atheroma ; then angina pectoris is prone to appear in its minor or in its major degree (*vide* p. 174).

Dyspnoea.—In aortic disease dyspnoea is not a frequent nor a prominent feature, as in mitral disease ; and this for obvious reasons. Indeed this symptom would suggest some holding up on the venous side.

There are not so many eccentric symptoms in stenosis as in regurgitation ; there is less tendency to gastric disturbance, to headache, to cough (cough in such cases points rather to dilatation of the aorta), or to faltering of the mind and memory.

Diagnosis.—Latent, in respect of symptoms, as stenosis of the aortic orifice in its earlier phases usually is, on the other hand I need not reiterate the warning not to assume this deformity in every case of basic systolic murmur. We have seen that even in persons of advanced years, in whom in all probability such a murmur does indeed signify disease about this area, it is not to be assumed that the aortic orifice is straitened. On the other hand, in them an atheromatous lesion is not to be set aside on the absence of systolic murmur, unless a testing effort fails to reawaken it. In atheroma I have heard the murmur with the reinforced beat after an extra-systole, which during the ordinary rhythm was inaudible. But systolic murmurs about the base of the heart are of

the commonest of clinical events : in young persons they are usually due to temporary changes in the blood or blood-vessels, in the elderly to atheromatous disease, which may be so slow as not to interfere much with their quieter lives ; and in not a few persons a soft systolic murmur, varying with the respiration, is insignificant ; it is often produced or intensified by deeply held expiration, and is attributed to some compression of the pulmonary artery. I have read, as a quotation from Berghe (a Dutch physician), that in pregnancy or arteriosclerosis an artery on the thoracic wall—in the second or third space—may so enlarge as under the stethoscope to produce a systolic murmur. On shifting the instrument an inch or two, of course it ceases. We have, then, in the first place to decide whether in a given case an "aortic systolic murmur" is of no significance, or is of the kind known as haemic, or is due to atheroma, or to other chronic arterial disease such as the syphilitic. Murmurs due to the acuter kinds or phases of disease are discussed in the article on "Acute Endocarditis" (p. 269). When in a syphilitic or rheumatic case the valve is menaced the second sound wanes ; in atheroma it alters in clang. To the left of the sternum down to the third rib its tone is of course mixed with that of the pulmonary valve.

In deciding whether a given aortic systolic murmur be haemic or organic, dynamical or statical, neither age nor sex is conclusive. A young woman may suffer from aortic stenosis of a fixed organic kind, without regurgitation ; and even this without any history of rheumatism, chorea, or other constitutional disease. There may be no definite signs of anaemia, no venous hum, no characteristic blood-change, little change of intensity with respiration or on varying her position ; and the first cardiac sound may be supplanted. In anaemia a murmur may be loud, or even harsh, and in stenosis the murmur may be soft ; but a sawing quality, especially if associated with a thrill, strongly suggests organic disease. In stenosis the apex of the heart is usually displaced, if but a little, in the vertical direction ; and the cardiac impulse is not merely forcible, not merely vehement, but steady, long and heaving. Protraction of systole under ordinary pressures is almost decisive.[1] Some overaction of the left ventricle may be perceptible, and yet the cardiac dulness scarcely increased transversely. A substernal oppression may make itself felt on exertion, or even during rest, which differs altogether from the painless and more panting dyspnoea of anaemia ; and the oppression may amount to pain, and run into the left arm. Again, although Bright's disease be not present, nor general arterial disease, the pulse may be anacrotic ; this feature of the pulse and a long plateau would set aside that rarer lesion, static pulmonary stenosis, and especially if the history indicates that the disease is not congenital. As to the

---

[1] As protraction of systole might not only be important in diagnosis, but for prognosis also, as, *caeteris paribus*, it would mean a corresponding overdraft on the cardiac reserve, and by innutrition might shorten again, I endeavoured to make some calculations concerning it. As I failed to obtain any certain data, I appealed to Dr. Lewis, who most kindly supplied me with a series of memoranda to shew that there are as yet no trustworthy measurements of cardiac systole. The fractions of time are too subtle for our instruments.

alternative of mitral origin, propagation into the carotids, and perhaps down the aorta behind, with a waning second sound, and the position and time of a thrill, are important distinctions, even if we suppose a mitral reflux insufficient to cause symptoms of venous retardation ; the murmur, again, though loud at the apex, may wane towards the axilla. An aortic systolic murmur may, however, emerge at the apex with some change of quality.   We have noted that a rising aortic often starts a moment later than a falling mitral systolic murmur (p. 446).   Then as to positive stenosis : within an orifice of not less than normal dimensions may the murmur be due only to a spur of a diseased valve in the current ?   May the hypertrophy be accounted for by undue peripheral resistance ? or again, may the case be one of aneurysm, for instance of a sinus of Valsalva ?   This alternative cannot be eliminated ; but against it would be a hypertrophy of the left ventricle, which is not a feature of this kind of aneurysm.   In a case of aortic regurgitation a prolonged systole with a diminishing volume of the radial pulse might signify progressive stenosis.

Can a systolic murmur be generated in the aorta without disease of the orifice ?   I have watched so many cases of large aortic dilatation, to their close in death, in which neither a systolic murmur nor any other murmur ever appeared, that I cannot assert that murmurs arise in the absence of implication of the orifice.   That vortices must form as the blood passes into a larger channel is certain, and that they should set up murmurs is likely ; yet in all cases of organic systolic aortic murmur which I have followed to the post-mortem table the orifice has presented disease sufficient to have caused it.

Finally, Rosenstein, following no less an authority than that of Traube (*vide* Guttmann, New Syd. Soc.), asserts that in aortic stenosis the ventricular impulse may be weak, or even imperceptible ; in positive stenosis with open coronaries this opinion is contrary to my own experience, and to that of many others ; although it may not be always possible to distinguish between cases in which a murmur is generated at the orifice without constriction in the positive sense and those of stenosis in the literal sense.   In estimating the size of the heart the positions of lung and diaphragm are not always allowed for.   If there be any power of response in the heart at all, it seems inconceivable that an increase of resistance such as we contemplate should fail to produce hypertrophy, and that it does so is a matter of common observation ; it is in these cases, indeed, that it comes nearest to the "concentric" form.   In the cases in which hypertrophy is absent the pulse is not tardy.   We do not get in aortic disease that globular heart which, large as it may be, does often fail to convey a concise impulse to the exploring touch. A high diaphragm, such as we often see in heart disease with a dis- tended gastric pouch, thrusts the heart upwards and backwards, so as to conceal the left heart more than the right.   Even on the *x*-ray screen a high diaphragm may cover much of a really large left ventricle.   If a systolic murmur is heard in the aortic area, if the pulse is 70 or over,

if there is no hypertrophy of the left ventricle, and the systole is not protracted, I should say either that the disease of or about the orifice, if any, has not the effect of stenosis, or, in the alternative, that the nutrition of the heart is failing.

Prognosis.—It is said that the forecast of aortic stenosis is of all heart diseases the least unfavourable. No doubt this is true if we bulk together all organic murmurs heard at the aortic orifice; but this is a pell-mell classification. We have said that well-to-do old ladies may lead tranquil lives, up to fourscore years or more, with systolic aortic murmurs of a quarter of a century's standing; as we see such persons with arteries reduced to coral, yet also living the length of the human span. In such cases the atheroma is rather that of senile degeneration than of high blood-pressures. I remember an old lady with jerky carotids whose radials had been as tobacco-pipes for fifteen years, who still pursued the unbroken tenor of her existence with no more to trouble her than a slight dry gangrene of the toes which had left her lame half a dozen years before. In these patients, however, the demands of life are of the narrowest and the lightest; the expenditure is almost nothing. Straitened as the orifice may have become, a hyper-trophied ventricle still drives an attenuated stream of blood through a tiny channel at a velocity, perhaps, of some four metres per second; so that the blood-column in the aorta is sustained for a long time at a pressure compatible with such a life. And if the mouths of the coronaries be obstructed very gradually, alternative paths of nutrition open out. Thus when we regard such cases, and those again in which the aortic mischief sets up corrugation rather than strict stenosis of the orifice, the prognosis does seem better than in that next best disease, mitral regurgitation. Yet if there be no aortic regurgita-tion, and if the relations of cubic capacity of ventricle and aorta be but little disturbed, there is, of course, a mechanical disadvantage. In the case of projecting spurs and rigid angles the fluid motion becomes so far discontinuous; and "dead water" dragged along is separated by surfaces of discontinuity from the more axial stream, so that energy must be absorbed. A division may be made which would shew aortic stenosis in a less favourable light; thus if we take patients under fifty-five years of age we shall find the prognosis much worse. The majority of such cases are rheumatic or syphilitic; the stenosis is more frequently stenosis proper, and consists in fibrotic inflammation about the ring and the valve which sooner or later may impair the myocardium, or attack the coronary orifices. If the pulse become more and more retarded, we may fear the masked process is invading the tract of Kent and His. Many of us have published cases of aortic stenosis thus terminating in bradycardia, as in Dr. Parkes Weber's case (128). Some prolongations of the a.-c. interval may, however, depend on other causes. Moreover, young life is more exacting, the patient is not becalmed in senile serenity. Much depends, of course, on the rate and kind of the process; but my impression is that when a person in young

or middle life begins to suffer overtly from acquired aortic stenosis the condition is a grave one. If the case be syphilitic, and taken in time, the results may be satisfactory. In such a case, in a gentleman aged thirty-six, whom I saw with Mr. Wilkins of Wickhambrook, very active aortic mischief, betrayed by dilatation of the ascending arch, rasping systolic murmur and stenocardia, after seven months of rest and steady specific treatment the murmur disappeared ; and except that careful percussion still revealed some extension of the aorta and slight general arterial thickening, no fault could be found with the circulatory functions. Unhappily, however, the light reflex and the knee-jerk had disappeared, and some gastric crises had occurred. The duration of the latent period of aortic stenosis is of course hard to forecast ; the mischief may be detected by chance, and it may be but a part of disease elsewhere. If as to the replenishment of the arterial system it is comparable to mitral stenosis, the intervention of the capable left ventricle does much to modify the consequences. The final phase may be by dilatation and rise of pulmonary and venous pressures ; but the usual mode of death is, by a more acute exhaustion and dilatation of the left ventricle ; sometimes very acute, as in the case of the boy (mentioned by Sir R. D. Powell) who, while running, succumbed and died suddenly. At the necropsy extreme aortic stenosis was discovered for the first time.

The stenosis of children and youths due to congenital defect of heart or aorta is described in another article.

Treatment.—*Vide* p. 483.

REGURGITATION.—Like stenosis, regurgitation also is often furtive in its invasion ; the cases are many in which the signs and symptoms of this disease are found without apparent cause. We have seen that aortic regurgitation does not appear in the ordinary course of events in elderly and atheromatous persons ; the ordinary result of atheroma in this area is a systolic murmur. But we have seen also that regurgitation is generally accompanied by a systolic murmur, though, as we may infer from the volume of the pulse, usually not indicative of positive stenosis.

Whether the mischief be due to past rheumatism (*vide* Acute Endocarditis), to strain, to syphilis, or to atheroma, the symptoms and signs are much alike. But in the two last cases we expect to find, and we generally do find, by other signs and symptoms, that the cardiac disease, urgent as it may be, is but a part of widespread arterial disease, with its own incidents and issues (*vide* " Diseases of Arteries," p. 609).

Symptoms and Signs.—*Pulse.*—Aortic regurgitation is sometimes perceptible to the patient himself, or to an observant friend, by the characters of the pulse, a very prominent and peculiar feature of the malady ; or the disease may first betray itself by vertigo, palpitation sometimes vexatious, and by the substernal oppression (larval angina) which has already been mentioned under stenosis.

It is commonly said that in aortic regurgitation the arterial pressure is low, and this in face of evidences of tensile strain witnessed in equal degree perhaps in no other disease. This unquestionable tensile strain is due to the stresses of the hypertrophied left ventricle upon arteries conspicuously large and slack. Under other conditions, as in Bright's disease, high as the mean pressures may be, the arterial tree is constricted ; and thus the maxima and minima are not widely apart. If in a normal arterial system, by active vaso-dilatation or abatement of tone, we find a wide divarication of the maxima and minima, this is temporary and the vessels are unharmed ; it is otherwise when, as in aortic regurgitation, the relaxation is both extreme and persistent. In this case tensile strain, acting both longitudinally and transversely, widens and lengthens the vessels, tending to split them across or along ; the vessels relaxed by reflex adaptive mechanism, no longer adapt the continent closely to the content ; the extremes of volume are far asunder. An equable circulation changes towards the form of discontinuous discharges, as if from a catapult. The systolic pressures, in an uncomplicated case in a patient under aet. fifty, range about 180. The well-known tracing of the radial pulse in regurgitation, usually with a hook at its summit, shews the violent percussion of a large output into a relaxed periphery, and as sudden a descent without plateau—the pulsus celer. Of itself this signifies no more than a large output and open periphery ; but in aortic regurgitation it is associated with a comparatively high diastolic abscissa (Fig. 51) (Lewis, Hugh Stewart). To attribute the steep gradient to the defect of the valve is erroneous ; at any rate there is no direct proportion between them. Even in acute cases, such as the traumatic or syphilitic, the vasomotor adaptive dilatation is instantly established. There is a large output at a low resistance. The hook in Dr. Lewis's opinion is not an artefact. The pulsus celer is not altered by the mere supervention of mitral regurgitation, if the left ventricle be still vigorous. No long series of observations is needed to ascertain

Fig. 51.—Margaret E., aet. 38 ; rheumatic fever at age of 17. Typical, pulse of aortic incompetence shewing exaggerated percussion-wave and steep tidal wave. (Graham Steell.)

if, in a number of cases, the mean pressure is higher under these extremes than in an equal number of cases of hypertrophy of the left ventricle without regurgitation ; nature has given us the information in the consequent state of the arterial tree ; in the lengthened and dilated vessels, in strains which, even in young subjects, issue in general arterial disease, especially in the parts most exposed to the driving of the big ventricle. These results are sufficient to assure us that, whatever the diastolic phases, in aortic regurgitation the mean arterial tension is excessive ; though, as it is far less buffered by tone, some deduction must be made in this respect. By elongation the arteries are thrown into curves ; and, as these are straightened at each diastole, the vessel is then thrown out of its bed with a visible and palpable jerk. The wife of such a

patient told Watson that for some time, on taking her husband's arm, she had felt this uncomfortable jarring.

Whether in a normal peripheral artery, such as the radial, the pulse should be visible is a matter of doubt. In some thin people, in whom a fine skin allows the radial artery to be seen, the pulse is perceptible to the eye, especially if its tone be slack. The beat is made visible by the tension of the skin over the vessel; were the vessel without a dint its pulse would probably not be visible. In arteries such as the temporal, which are without much cushion, elongation takes place more readily; and in men still young and healthy the temporal is often thus thrown into curves which reveal the pulses clearly enough, even to the eye. After the stress of aortic regurgitation has been continued for a longer or shorter time, all the arteries exhibit this jarring impulse—"danse des artères," starting out of their beds with each pulsation. "Sometimes the whole of the patient's body," says Watson, "nay, his very bed, is shaken by the strong shock of the heart during its systole."

The characters of the pulse are well known. The gifted physician to whom we owe most of our knowledge of this subject has given a memorable description of it. Corrigan compared it to the "water hammer," a toy in which water, imprisoned in an exhausted tube, falls, on every turn of the tube, from end to end with a thud. With such a thud the charge of blood is shot along the open arteries. In many cases this jerkiness is well seen in the tonsils. On raising a limb—the arm for instance—to a vertical position, the jerky character of the pulse becomes more apparent; so that if in a case of the kind these characters are not obvious in the horizontal limb they will appear when the limb is raised. Indeed the pressure in the peripheral vessels may fall to such an extent that under these conditions the pulse may disappear in them; though the disappearance is often due to some constriction of the vessel at a higher point, as at the flexure of a joint, or by the fold of a garment. On raising the arm of the patient while he has an overcoat on the radial pulse may vanish; on removing the dress the pulse in the same position may persist. It is popularly but too readily assumed that this character is directly attributable and proportional to the ventricular reflux; but this condition is probably less concerned in it than the reflex flaccidity of the arteries. It is by no means always present; a certain degree of vaso-constriction, such as digitalis might produce, abates it. On the other hand arteries throb in Graves' disease, in "pulsating" abdominal aorta, in hot baths, and so forth.

In cases of extensive arterial sclerosis, or at any rate of sclerosis of the radial and brachial arteries, the stiff walls of the vessel do not collapse with the sudden ebb of the pulse-wave as a comparatively normal artery does. Nor, indeed, can the arterial diastole be so well marked. Yet, unless stenosis be present, the patent and often stiffened and stretched arteries will vibrate or jar; a jarring or purring in

the carotids, abdominal aorta, contorted brachials and other accessible
vessels which, even in the absence of a systolic aortic murmur, will be
perceptible by a light touch of the finger, if not by the eye.   Some
authors have anticipated that with the loss of the support of the aortic
valve dicroty would wane or disappear ; indeed in the degree of its
persistence a prognostic test has often been sought, though sought in
vain ; for in most cases, even in the more advanced, we find a dicrotic
pulse in a tracing, if not with the finger ; the subject needs further
investigation.   Dr. Lewis has shewn that records of dicroty, if near the
base line, are soon lost to the sphygmograph.   The notion of Janowski
and others, that dicroty in aortic regurgitation signifies "mitralisation,"
is rightly contested by Dmitrenko and Geigel.

Eichhorst states (Vol. I. p. 40), and produces three cases to prove
that any difference in the tracings taken from the horizontal, vertical, and

FIG. 52.—Aortic incompetence without stenosis.
Man, aet. 29, with history of rheumatism at the
age of 20.   P. bisferiens. P.M. (Graham Steell.)
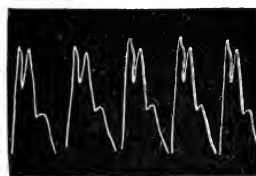
FIG. 53.—Bisferiens pulse in a case of
aortic incompetence without stenosis.
P.M.   (Graham Steell.)

dependent arm respectively, are no more than instrumental artefacts.
But surely the truth is that the differences are not of a kind which lend
themselves to sphygmographic comparisons.

It is the function of a healthy heart and healthy vessels to pro-
mote at each beat the maximum of blood-displacement with the minimum
alteration of pressures.   At each beat the heart should leave that
portion of its energy in the elastic arterial coats which, given out again
between the pulsations, would convert the intermittent pulses into a more
continuous flow : now it is plain that in aortic regurgitation we have
the converse of this—the maximum of pressure-disturbance with the
minimum of blood-translation.   More of the systole is left behind at
each successive point, so that the continuous flow, normally not reached
until the area of fine vessels, in aortic regurgitation is not reached at all.
Thus, in a well-marked case, in no part of the arterial tree does the
flow become continuous, not even in the capillaries ; and Quincke's
"capillary pulse," although not peculiar to aortic regurgitation, is very
characteristic of it.   Yet even in health, if the arterial tone be very low,
the capillary pulse may be seen.   A former pupil, a quite healthy man,
demonstrated to me the capillary pulse in his own person ; he told
me that it was habitual in him.   Dr. Waller also says it may be
detected in many normal persons, though in its extremer degrees it is
characteristic of aortic regurgitation.   Like the character of the pulse

so this capillary wave is not attributable so much to the mere mechanics of the aorta as to the extreme vasomotor relaxation which usually ensues upon the valvular affection; presumably to accommodate as much blood as possible before the backlash. On the other hand, in many cases of this disease the capillary pulse is not notable. Now and then in a case of aortic regurgitation, even of considerable degree, it does not appear, or it is suspended; in such cases capillary pulsation is invisible. So digitalis, by toning up the arterioles, is said to abate it. The readiest way of obtaining the reaction at the bedside is to press upon one of the patient's finger-nails with the point of a pencil; in a good light, and with the limb a little elevated, the pulsation generally becomes distinct. Or it may be shewn by pressing a glass slide upon the mucous lining of the everted lower lip; or the skin, on the forehead or elsewhere, may be rubbed until, as it reddens, the pulsing comes into view. It may be seen also in the vessels of the retina, even in the veins, by the direct image; or in the areola around an eruption, such as urticaria. The same thing may be observed in slack vessels in other states, as, for instance, in Graves' disease. If, in a case of double murmur with considerable regurgitation, the capillary pulse be not perceptible, or be indistinct, we may surmise a considerable factor of stenosis.

Delay of the Pulse-Wave.—In the first edition of this work I gave some considerable space to this question, as it was then attracting no little attention both practical and theoretical. Sir William and Sir John Broadbent had declared emphatically, as Fagge and others had done, that in aortic regurgitation the cardio-radial interval is—or often is—prolonged; and since then not a few physicians of repute, especially Tripier and Dr. W. Broadbent, have supported this opinion. Some, indeed, have gone so far as to found on the degree of delay a gauge of the degree of the heart disease. Upon the basis chiefly of ordinary clinical experience I was holding a contrary opinion; but I hesitated to oppose a clinician so sagacious and with a memory so richly stored as Broadbent. Moreover, I was shaken by some tracings taken at the moment of writing by Dr. Paul Chapman. As time went on, however, I was unrepentant, for the closer my attention the less could I detect of the alleged peripheral pulse delay. I am no longer occupied in a large hospital, but I see too many cases to suppose them all to be exceptional. It is true no doubt, that, *caeteris paribus*, the larger and laxer the vessel the slower the propagation of the wave; so that our prepossessions might be in favour of some delay, not in aortic regurgitation only but also in other cases of slack arteries; *e.g.* in febrile states, or Graves' disease, the distance between aortic valve and radial being taken at 80 cm. Many fractions quoted are so small as to be imperceptible to the most sensitive finger; and even a delicate machine, unless applied with rigorous precision, which is very difficult to ensure, will fail to record them. As in aortic regurgitation the aorta and ventricle become virtually one chamber, the prosphygmic interval is shortened or abolished, a systolic murmur is more immediate, and, as without differential pressures

there can be no differential times, the aortic diastole should be coincident with the first contractile effort of the cardiac muscle. If so, the radial pulse cannot well be delayed beyond the time of the velocity of the wave. Let us turn to the measurements of the interval; most of them have been taken, as were Tripier's and Dr. Walter Broadbent's recently published curves, not from the carotid but from the heart. Now in working to fractions of a second a precise benchmark is essential. But the apex-beat—upon the moment of which the measurement thus depends—is, even in health, not very easy to fix, and in a cycle so diverse as the rolling and recoiling impulses of a tumid left ventricle boxed up in the chest, peculiarly difficult, if not impossible; a demur, by the way, not to be forgotten in criticising many other cardiac observations. On some chests the first part of the cardiogram is auricular. This is often very evident in mitral stenosis. In any case we must calculate not from apex movement but from the *opening* of the aortic valve. And in thoracic cardiography, it is very difficult, even during the same experiment, to keep the conditions constant. I could not but decline, therefore, to accept cardio-radial computations, even if inspection of such curves did not also in themselves seem to fail of conviction. The carotid wave does indeed give us a better benchmark; but in the published carotid-radial tracings I find no evidence of abnormal delay. Dr. Broadbent admits that he cannot demonstrate any carotid-radial delay; certainly no delay which the finger could detect, or which might not be swallowed up in concomitant variations. Still, as of late years I had worked more with manometers than with the sphygmograph, I referred the matter to Dr. Mackenzie, who replied : "I have taken an enormous number of tracings of radial, carotid, and apex beats in aortic regurgitation, and have never in any single case detected radial delay." This opinion is borne out by the observations of François-Franck and d'Espine, and by some of Dr. Chapman's curves. It is rather in mitral regurgitation, which may abate the first stress upon the aortic valve, that we should expect any delay of the radial pulse.

The pulse during the more stable phases of aortic insufficiency is regular but, as contrasted with stenosis, is usually somewhat accelerated, and is of greater amplitude. Indeed, aortic regurgitation is the only heart disease in which the pulse is enlarged. The acceleration is, in a recent lesion, perhaps for some weeks, considerable; and a reduction of frequency is a sign of amelioration. Accordingly, in later stages the rate again becomes excessive. The rhythm is of similar importance. Trivial as in a healthy heart an intermittence of the pulse may be, in the disease before us it is of grave significance; almost as grave as it is in pneumonia or other infective disease. An occasional intermittence, of the extra-systolic variety, is of no ill omen; but, if recurrent, it too often indicates dilapidation of the heart. A faltering or dropped beat, such as pulsus alternans, is a far more serious event in aortic regurgitation than in stenosis, or in mitral disease : as in "fatty heart," it indicates a failing heart, or at any rate a heart needing

protection by the vagus.   Other forms of irregularity are warnings of
like omen.   In the aortic insufficiency of cardio-arterial disease,
irregularities occur earlier than in that of younger persons with a sound
myocardium.

    If the stethoscope be laid with the lightest pressure on an artery of the
size and position of the carotid or subclavian, an abnormally loud "tone"
may be heard on its diastole, and not infrequently on its systole like-
wise—"the double tone"; this is not peculiar to aortic insufficiency,
and may be heard in health, if the vessels be slack (pulsus celer).   It
is important to remember that such a tone, single or double, is also one
of the notes of aneurysm.   A slight increase of pressure produces a
murmur, as we know; but if a murmur be generated at the aorta, this
first arterial tone is supplemented by murmur, without the use of
pressure.   In aortic insufficiency and in stenosis the second tone in the
carotid usually wanes.   The murmur of aortic insufficiency may itself
be heard in the carotid, but by no means always; the conditions of
its propagation thither are of no clinical moment, but it serves
for an occasional discrimination of an aortic from a mitral diastolic
murmur.   But the sudden collapse of toneless arteries gives rise to
signs which are perhaps something more than curiosities; one of
these is the sign of Duroziez.   For the most part murmurs are
produced in the arterial system by the passage of the blood into
a wider channel; if then in health, pressure be made on an artery,
say with the edge of the stethoscope, these conditions are fulfilled,
and a murmur is set up.   In aortic regurgitation this phenomenon
is intensified, because in this state the slackened vessels vibrate more
readily, as may be conveniently observed in the femoral or brachial
artery.   But, as Duroziez pointed out, there is something more than
this : the artery may give out not only this single murmur, a murmur
of its own diastole, but a murmur on its systole also—a double murmur
which in the normal state cannot be obtained.   We have said that
a tone is produced by the diastole of a normal artery near the heart,
such as the carotid, to be heard on light contact of the stethoscope;
but here we are discussing not the tone, but a murmur artificially
produced by stronger pressure (the word "bruit" unfortunately means
either tone or murmur); and in the case of aortic regurgitation this
murmur may be followed by a second murmur generated on the arterial
systole or collapse.   The causes of this murmur are not precisely
known, but consist in differential pressures above and below; it is
not dicrotic, it may be a "recoil" murmur as surmised by François-
Franck, though a positive backlash of the current is not to be
supposed.   To get the double murmur clearly, Potain directs us to
press on the artery with that edge of the stethoscope which is
farther from the heart, so that the whole wave, if it be a recoil, passes
under the base of the instrument.   For its production there is a "most
favourable point of pressure," a degree between too light a pressure and
obliteration of the vessel, a degree to be discovered in each case during

the examination.   Another method is to compress the vessel with the finger, and to place the stethoscope on it above this point.   It is said that the second murmur—Duroziez's murmur—dies out as compensation fails ; if so, it is more than a mere curiosity.   Personally I have often elicited Duroziez's murmur in aortic regurgitation, but not always ; nor am I sure of the reasons of the variation, though I suspect they lie in the state of the arterial coats.   In a recent case of aortic regurgitation with vessels still unimpaired I heard it very definitely. Moreover, as Vierordt says, Duroziez's phenomenon is not quite peculiar to aortic insufficiency.   It can be obtained occasionally under other atonic or relaxed conditions, as in fever, when the arteries are largely vibratile and their diastole sudden ; and in aortic aneurysm ; in Graves' disease, and after administration of amyl nitrite : what it tells us is that the arterial diastole is very brusque and the systole "collapsing." Arteriosclerosis therefore tends to reduce it.   These arterial sounds have been carefully studied by Litten.

Physical Signs.—*Inspection and Percussion.*—Of the signs of dilatation and hypertrophy so much is said elsewhere that I can touch only on special points.   The enlargement in aortic insufficiency may be enormous, and in cases of any duration is unmistakable.   In aortic disease the heart maintains much of its conical form, and the dulness occupies a long diagonal from the third rib ; in mitral disease it takes a more globular or purse-like shape.   In persons under middle age the cardiac area may become prominent, as the cyrtometer will shew.   In childhood this alteration of shape may become conspicuous in the stretched skin and widened spaces.   In slighter hypertrophy the heaving impulse will be more readily appreciated if the ear be laid directly upon the wall of the chest ; or if a light wooden stethoscope be grasped by the ear-piece and the knob applied to the chest.   In slight regurgitation hypertrophy may presumably exist with but a nominal degree of dilatation ; and the less the element of dilatation the more "triangular" is the apex area ; the superficial area of dulness is extended downwards and outwards a little beyond the impulse, but does not extend so much transversely.   It is only by inferential reasoning that we can decide in a particular case how much of the dilatation is primary and adaptive, and how much secondary and due to atony.   Although a dull area, corresponding to mechanical or vasomotor dilatation of the aorta, may occupy the region of the manubrium sterni, and transgress it an inch or two to the right, ventricular dulness may not be enlarged to the right ; or not at any rate until in protracted cases the chambers of the heart are involved in a common defeat.   Signorelli states that in normal persons the first four thoracic vertebrae give a clear high pulmonary sound, not very intense ; the rest a deeper and intenser sound.   If, however, the first six give the same sound the deficiency of resonance over the fifth and sixth signifies enlarged auricles, especially the left, and so perhaps mitral disease ; over the seventh and eighth it signifies enlarged ventricles,

especially the left (or of course pericardial effusion). Dulness on the first two or three signifies enlargement of the aorta. Emphysema may baffle these tests. François-Franck points out that the apex itself may be "dicrotic"; the first shock due to the reflux, the second to the propulsion of the blood. The late Sir W. Broadbent gave us the useful warning not to mistake the systolic recession of intercostal spaces, due to atmospheric pressure acting upon the space left by the diminution of a large heart, for a sign of adherent pericardium. In cases of arterial disease the observer will not forget that causes of hypertrophy may have been in operation for an indefinite time before the establishment of regurgitation. In such cases the hypertrophy cannot be taken as a direct measure of the insufficiency, which may be a recent and inconsiderable accident, the chief part of the changes being attributable to the common causes of both; however, whether recent or of long standing, cardiac enlargement in later life, considerable as it may be, does not reach the dimensions of cases in younger and sounder persons. It is in the aortic insufficiency of the young, due almost always to rheumatic fever, that the huge hearts are found which lift life forward for many years.

Sounds of the Heart.—Whether there be positive stenosis or not, aortic insufficiency is generally accompanied by such changes in structure or function about the ostium as to give rise to a direct murmur also. It is probable, for instance, that a murmur may be produced by the collision of the outward with the regurgitant current. In cardio-aortic disease, such as syphilis or atheroma, a diastolic without a systolic murmur rarely happens, as the regurgitation is usually a later incident in its course. If in strain or rheumatic injury the murmur of regurgitation may be heard alone, at any rate for a time, yet even then the first sound is seldom pure. At the apex the first sound is usually prolonged, especially if there be coincident stenosis; and it takes a more "booming" quality as the hypertrophy increases. The direct murmur is usually, but not always, carried well up into the carotids, perhaps with a thrill also, so that their diastolic tone is supplanted by the murmur; sometimes it is carried even to arteries of the third magnitude, radials or tibials; or on the contrary neither sound nor murmur may be audible in them. A tone like a cardiac first sound may be produced by the diastole of a slack carotid. The right carotid is better for auscultation than the left; but the signs in the two sometimes differ. At the pulmonary cartilage, if the mitral valve be effective, the second sound is unchanged; unless, indeed, the artery be thrust nearer to the wall of the chest, when the sound will seem accentuated. The second aortic sound in aortic regurgitation usually persists more or less; although under uncertain conditions, not by any means parallel to the gradient of the advancing disease, it varies widely in intensity, or may even vanish. It is said by some clinicians that any persistence of tone is due to the pulmonary valves only; but that this is not generally the case is proved by its not infrequent propagation into the carotids. By others it is argued that if the second aortic sound coexist with a

regurgitant murmur there must still be a substantial area of closure, whether by fractional parts of the valve, by the aid of surrounding parts, or by the establishment of a degree of stenosis, which if moderate may be conservative. My own impression has been that persistence of the second sound was a subordinate detail ; for it is caused, not by the valve slapping to under the blood-column, as the cusps, or scraps of them, tighten under the vortices above, but. by a vibration of valve, orifice, blood, and aortic wall together. The cusps vibrate not of their own mere tension under the blood but in it, and their vibrations are damped by the viscosity of this fluid. The disappearance of the second sound may be of ill omen, but it is certainly not clinically correct to state that persistence of the second sound is inversely as the regurgitation. However, since the date of the first edition of this work a series of experiments by Ferrannini has added some important data, though we must beware of identifying confidently the results of. rough experiment with the creeping inroads of disease. Ferrannini, for this purpose, divides the long pause into four spaces, which he names respectively protodiastolic, protomesodiastolic, mesodiastolic, and telediastolic ; names which I may abbreviate as p., pmd., md., td. He produced in mammals a long series of lesions of the aortic valve, under proper precautions of method. If the lesion was tiny, or but a fenestration (p. 438) or small perforation, no murmur was generated ; but, as the valve thickened or slightly altered, the second aortic sound lost quality. The slightest lesion, if sufficient to produce a regurgitant murmur, produced it as p., but this was very soon protracted into md. or pmd. And thus far the second sound persisted, though diminished in proportion to the degree of the lesion ; p. or md. indicated slighter lesions than pmd. As an increase in lesion extended the murmur from p. (which was the least frequent length) or md. into pmd., the second sound did often vanish. Conversely, on some healing of the wound, a pmd. murmur would retract into md. or p. Practically td. never occurred alone, nor indeed was often heard ; which agrees with Henderson's division of the cardiac cycle into systole, diastole (filling), and diastasis (the moment of rest). Broadly, then, in these experiments, the degree of surviving second sound did seem to be inversely as insufficiency. It is not without practical interest that among the effects of experimental aortic regurgitation a disposition to aneurysmal dilatation at the apex has been noted.

As compensation fails, of course, all heart-tones become less audible ; and, if mitral regurgitation supervenes, the tension of the aorta slackens and the first sound wanes accordingly. To distinguish a tone in the presence of a murmur, the device of slightly withdrawing the ear from the stethoscope, so as to make the murmur more distant, must not be forgotten. These finer distinctions of quality are to be sought with a light wooden stethoscope ; the binaural tube is a coarser instrument. Dactylic, anapaestic, and gallop rhythms are not very characteristic of aortic disease, and are discussed in other chapters. The propagation of an aortic regurgitant murmur into the carotids is said to signify a large lesion,

especially if the second sound be inaudible. But again I would demur that these phenomena depend upon many variables, and differ with the formation of chest, states of arterial wall, cardiac energy, and so forth. Many of these points of physical diagnosis need verification ; but, after all, sounds and murmurs make but a part of diagnosis, and in quickly beating hearts such points become inappreciable. In the varying states of the chambers of the heart we have other and better measures of degrees of disability.

Murmur of Regurgitation.—This murmur may be due in some part to gravitation ; but suction of the ventricle, aortic recoil, the return from a smaller to an enlarging cavity, and the encounter of the mitral affluent are the main coefficients. If it be true that this murmur is usually definite and its meaning inevitable, it is none the less true that, with the exception perhaps of mitral stenosis, it is the murmur most frequently overlooked ; and not by pupils only, but also by experienced physicians. Sometimes the murmur lurks in unexpected places ; sometimes its quality is so soft and evanescent that a quick ear is required for its detection, especially if a rasping systolic murmur precede it. If it be both faint and in its site aberrant, it may escape even a practised ear, at any rate for the moment. If in rheumatic fever, or other infection, a systolic murmur appear, a very careful search must be made in all areas of the left ventricle for a regurgitant sound ; the patient, if convenient, sitting erect. It is often audible in the axilla down to the seventh and eighth spaces, and may not only be soft and distant, or lurk in strange places, but be aberrant in time also. It may occupy fractional parts of the diastolic period, and not always the initial part. Like a murmur of mitral stenosis, it may be perceptible to the ear only in the middle, or middle and final third of the long pause ; and if discovered accidentally in this rhythm, before the advance of secondary changes, may deceive even the elect. Physiologists are not yet quite agreed about negative intraventricular pressures. To me it seems that, when venous pressures begin to rise, as blood pours in, a negative pressure must be very soon annulled. Yet possibly, as the venous pressure is relieved, a second moment of negative pressure may arise, to be occupied by a fractional murmur of regurgitation. One such case I remember which divided three hospital physicians in opinion. One inferred mitral stenosis ; two held out for aortic regurgitation. Dr. Douglas Stanley described an interesting case of this kind at a branch meeting of the British Medical Association. A diastolic murmur arose immediately on diastole, a murmur of aortic regurgitation, well marked at the base ; but at the apex was heard a mid-diastolic murmur, rougher than that of the base, and not heard, as such, outside the mitral area. After death the aortic valve was seen to have only two cusps, and these were involved in vegetations. The mitral parts were healthy. A diastolic murmur heard in the carotid must be aortic, though, unfortunately, it is not always carried into this vessel. To seek an aortic regurgitant murmur at the second right cartilage is very properly to note it where it is most isolated

from other sounds; but, as this may not be, and very commonly is not, the seat of its maximum intensity the search must also be made everywhere within the "sphere of influence" of the left ventricle.

Loudness of murmur is no indication of severity of lesion; the contrary is rather to be anticipated, at any rate in systolic murmurs. For a loud murmur generally signifies a vigorous heart, and even a refluent stream, returning at a high velocity, may set up more active veins in the ventricular content than a larger return at a lower velocity. Indeed, if a murmur, previously loud, fall in intensity we may be apprehensive of failing velocity. Moreover, a short murmur soon muffled may well mean that the regurgitation is so ready and voluminous that aorto-ventricular equilibrium is reached almost at once. Sudden ruptures of the valve, which call out the utmost cardiac reserve, often give rise to very loud murmurs, audible over a large area of the chest; sometimes audible to bystanders, sometimes even to the patient himself. In the presence of stenosis, other things being equal, a regurgitant murmur is louder, as the velocity of the refluent current is greater. In these cases a comparatively small and fairly sustained pulse is associated with a sawing double murmur. A jet returning through a perforation of a limb of the valve is said to be attended with a piping or mewing sound (p. 435); it has been compared to the chirping of chickens. A cardiac murmur, direct or regurgitant, audible without contact with the wall of the chest, is probably always aortic—an inference sometimes of diagnostic value. A regurgitant thrill is sometimes perceptible. That by any quality of its own the murmur of mere valvular disease can be distinguished from that due to a local atheroma I can hardly admit.

Sewall of Denver has investigated the behaviour of all cardiac murmurs under increasing pressures of the stethoscope. He says that murmurs of aortic stenosis audible at the apex disappear under pressure there, and are herein distinct from mitral regurgitation. Also that, great dilatation of the aorta or aneurysm apart, the murmurs of aortic regurgitation may be reduced or annulled by pressure at the base but not at the apex. "Inorganic murmurs" at the base, he says, can all be obliterated by pressure; and by the same means used in the second right interspace close to the sternum the normal second sound can be stopped, unless the aorta be so dilated as to be in contact with the wall of the chest. I have never succeeded in thus suppressing a static aortic murmur, though the pitch of it may seem to change. I say "seem" because we must not forget that by the pressure we alter the medium of conduction.

The student is often directed to track a murmur to its origin by shifting his stethoscope along the surface of the chest from one area to another, in order to note where one murmur dies and another is born. This is a misleading device, or one to be used only by cautious observers. A murmur, like a river, may run underground in part of its course; and as the media of conduction differ from place to place, one and the same murmur, as the stethoscope travels, may so alter,

as the structures about it vary in conductive capacity, as to appear twofold. Many an alteration thus arises as the observer slips the instrument diagonally upwards ; a murmur heard at the apex disappears to reappear at the aortic cartilage, and thus a murmur generated at the aortic orifice only may be regarded as indicative of two lesions, when it is but one with two maximum points. Again, if we assume that the murmur always follows the direction of the blood-current we may err ; the blood is not rippling in the air, as water over gravel ; the murmur we hear is due to vibrations of surrounding structures—chiefly the walls of the heart—set up by the vortices within them ; the heart is the fiddle, the blood is but the bow. We must rid our minds of these conceptions of blood running here and there in the chambers, as if from a water-cock into a pipkin, and realise that the walls are thrown into vibration by molecular collisions in a plenum. Not only so, but the fluid vein or veins generating the sound blend into the resultant of all the lines of motion, normal and abnormal, in the particular heart.

Of the alleged cases of aortic regurgitation without murmur, I can say only that in rare instances extremely soft murmurs are evanescent, but recurrent. Weismayer, in a paper which has been much quoted, accepts cases of regurgitation without murmur, a little uncritically, I think. Sir Hermann Weber's case, quoted on p. 469, is an example of the manner in which, in incipient cases, the murmur of aortic insufficiency may cease for a time, but probably with the insufficiency which engendered it ; again, like any other murmur, that of insufficiency may wane with the heart in which it is generated, or in a considerable acceleration of rate escape the ear, or by some other murmur may be concealed ; but that, with a comparatively vigorous heart and persistent insufficiency, a regurgitant murmur may be absent in the erect position of the patient, or may come and go, is contrary to experience, or at any rate to mine. Aortic competency does not depend as mitral competency depends, upon a co-operation of valve, ring, and muscle. At this orifice an intermittent murmur means more definitely that the static conditions are as yet unfixed. As proving exceptions I may refer to cases reported by Dr. Saundby, and by Musser. In Musser's curious case the corpora Arantii had been transformed into calcareous buttons (4 mm. by 2 mm.). During the formation of these excrescences regurgitation took place, and a murmur was generated ; but, as they wore down and the free surfaces became faceted, after the manner of gall-stones, the incompetent valve became again competent. In a case due to past rheumatism, which I saw in Cambridge more than once with Dr. Humphry, a regurgitant murmur absent on one occasion was present on another ; but we agreed that the regurgitation also was probably intermittent, the valvular defect being slight (no secondary changes—the murmur was found in an insurance case). In apparently healthy candidates for life insurance aortic insufficiency, if thus ill-marked, may easily be overlooked. In all these cases, however, as the lesion takes its permanent shape, the murmur will likewise become

permanent. In another case (syphilitic), which I saw repeatedly with Dr. S. W. MacIlwaine, while the patient was under specific treatment a regurgitant murmur kept coming and going for some months. Even when the regurgitant murmur was inaudible, the damping of the tones—so characteristic of these syphilitic cases—was always notable.

Whether regurgitation may occur in dilatation of the aorta with the valve unimpaired, and without aneurysm of a sinus, has been considered already (p. 437). At the mitral orifice relative insufficiency is well understood, and is by no means rare ; but here, as I may reiterate, it depends upon muscular tone, and other conditions of muscular and tendinous attachments of the limbs of the valve which the aortic valve does not possess.

If a patient with occasional regurgitation be examined at a time when the valvular disease is latent, or at any rate unrevealed by murmur, a grave error of diagnosis may be committed. The following case, resting on the authority of Sir Hermann Weber, is most instructive :—

A very active young man, aet. thirty-two, of weak muscular development, was examined by Sir H. Weber on arriving at a height of 8000 feet. The second aortic sound was replaced by a musical murmur at mid-sternum and a little to the right. The first sound was rather indistinct. The pulse was 105-112, feeble, but not characteristic of aortic regurgitation. On the following day the murmur had disappeared ; the heart-sounds were normal, and the pulse was 88. Two days later, at 9000 feet, the same murmur became audible ; and in like manner disappeared on the day following. Further climbing was forbidden, and he returned to work in good health. Seven years later, however, the patient died of " Herzschlag."

Among my own notes I find the following case :—

A man about middle-age complained of attacks of palpitation followed by great exhaustion. He saw many physicians, and had many prescriptions of diet and drugs. After some months a murmur of aortic regurgitation appeared, and at this moment he came to me, accompanied, fortunately, by his family physician, who confided to me his suspicions that the patient had had syphilis some years previously. Active specific treatment stopped both the functional attacks and the murmur.

Thayer describes a case of aortic regurgitant murmur, heard by himself and a colleague, which five months later had disappeared. This case was probably one of syphilis. In syphilitic cases by timely treatment acute aorti-valvulitis may be so abated that a regurgitant murmur may take its departure with a mitigation, perhaps with the virtual cure, of the local disease which gave rise to it. Relatively rigid as is the aortic ring, the vigour and soundness of the aortic muscular cushion have some considerable function, and we have seen (p. 438) that in atheroma, and perhaps in myocardial infections, this structure may be gravely impaired. Once more, it is alleged on some evidence that a murmur consistent with regurgitation, but really

due to an aneurysmal kind of eddy, may be set up in a largely dilated aorta without regurgitation. Possibly one of the little pouchings which occur about the Valsalval sinuses might give rise to an eddy audible during diastole. Barjon published two cases of Hodgson's disease (*vide* p. 633) with a persistent murmur of regurgitation; yet after death the absence of regurgitation was demonstrated, so far as the water test is to be trusted. The arch in both cases was very greatly distended. I have never heard the murmur in cases in which competency of the valve was verified. With normal elasticity the aperture on systole is not circular, but a narrow oval, or wide slit, which may be a better guarantee against regurgitation (Mai, Krehl, Hesse). Sir Oliver Lodge has explained to me that vortices which obstruct and throttle a pipe arise more readily through a round orifice than through an oval one; through an oval one they are much more unstable. So the ovalness secures that the stream shall issue in an "irrotational" manner—the freest exit. Yet as the systolic stream passes at higher velocity into a wider room, it forms vortices sufficient to close the cusps. The cusps float in the issuing stream and, never very taut, follow the blood-currents. Or suction, as well as aortic vortices, may have some effect in setting them close. With a more spacious orifice the cusps might lose some of this compulsion.

Again, we have seen that regurgitation may possibly occur at times of high blood-pressure—as for instance in exertion or in hyperpiesis, and wane or disappear, as, under treatment or otherwise, the aortic pressures fall. So perfect is the concert of the heart and vasomotor system that, as we have seen, in early aortic valvulitis the least regurgitation throws open the periphery, widely and at once. But to sum up, the clinical maxim, if not one without exception (p. 438), must be that in aortic regurgitation, even if intermittent, the valve itself is not intact. In cases of disease, especially of syphilitic disease of the aorta just above the valve, an intermittent regurgitant murmur may signify the moment of invasion of the valve itself, a moment when prompt and active specific treatment may still save it. Or, again, in the earlier stages of disease of one of its limbs, the valve, by mutual accommodation of its parts, or of vegetations, may suffice to keep the orifice closed until by excessive blood-pressure, or some other physical change, the regurgitation intervenes or becomes established.

Murmurs occurring during diastole may be heard in pericarditis and aneurysm; the former murmurs are not difficult to interpret. A venous hum may simulate aortic regurgitation; but with a due regard to the effect of respiration and other features this simulation ought not to give much trouble.

Dilatation of the aorta is said to be the rule in the aortic regurgitation of cardio-arterial disease, the exception (in any considerable degree) in primary aortic regurgitation. If so, the exceptions are many; I lectured recently in Cambridge on a juvenile case of regurgitant aortic disease of rheumatic origin, in which there was considerable dilatation of the aorta with "fireman's helmet" dulness; and, if deliberately sought

for, more of these cases would be recognised. Many are on record, and some few in which the aortic affection issued in aneurysm. A relaxation of the aorta in rheumatic and other infections may attain considerable dimensions, yet sometimes apparently atonic only ; so that a lifting arch in the episternal notch must not be put down at once as diseased. Such merely atonic expansions are often notable in Graves' disease, and similar states of arterial relaxation.

Simulation of a mitral stenotic murmur by aortic regurgitation may test our cleverness far more severely. Ordinarily the murmur of aortic insufficiency begins with diastole, is then loudest, and falls as the aortic pressure falls ; that of mitral stenosis ordinarily rising up to the systole. Aortic diastolic murmurs in the later part of the pause are very soft, because the pressures in aorta and ventricle are then nearing equality, or have attained it, the vibrations persisting for a sensible moment longer in the walls of the heart. If the murmur is heard at upper and mid-sternum, begins with the diastole of the heart and tapers off during the pause, it is an easy sign to interpret. But if the murmur, not as a rule so harsh or vibrating as that of mitral stenosis, be so soft that it may escape an unpractised or incautious ear ; if, instead of tapering off from the beginning of the pause, it occupy the middle, or even the latter part of it ; if, again, it be barely audible or inaudible in the sternal areas, or at any rate in the right upper sternum, and be loudest about the lower sternum, left fourth interspace, or axilla, the student of the "aortic cartilage" may be misled by whispers so stealthy and devious. He may attribute the murmur, if he hears it at all, to mitral stenosis ; or he may add the case to the list of vanishing aortic regurgitant murmurs ; or, again, he may add himself to the cloud of witnesses to pulmonary regurgitation.

Now, however distinct an aortic murmur may be in the left fourth space or axilla, it dies down as the apex is approached. We have seen already that murmurs occupying parts of the long pause, not necessarily the initial part, are consistent with, and in certain circumstances indeed significant, not of mitral but of aortic disease. A case shewn to me in a hospital a few years ago by two physicians as a murmur of mitral stenosis, was in my opinion such a case of broken aortic diastolic murmur, unconcerned with the mitral area ; in mitral stenosis in Dr. Mackenzie's opinion a murmur so placed would mean an advanced stage of this disease. But this is not all : without mitral stenosis a presystolic murmur is very commonly present, the aortic being the only lesion. A presystolic murmur, heard in the mitral area, and with the completer features of that of mitral stenosis, is often to be heard in uncomplicated aortic regurgitation ; so often that one wonders it was left to the late Dr. Austin Flint to describe it (in 1862). His lead was followed by many other observers, whose records have been well summed up by Dr. Lees. Rumbling presystolic and diastolic apex-murmurs, even with thrills, are now so commonly recognised in aortic regurgitation unaccompanied by

mitral lesion, that every senior student is alive to the ambiguity. The phantom murmur may fill the pause, or be mid-diastolic or take the ordinary presystolic form. Sansom, who recorded cases of this kind in 1881, carefully discussed the diagnosis; with Potain he leaned to the belief that this presystolic murmur might be due to impingement of the refluent aortic current on the anterior mitral curtain before it is made taut, whereby either vibrations are set up in the valve itself or, by bulging the valve, the orifice is obstructed. Dr. Fisher and others (Peacock, Fagge, Bristowe, Glynn) have published cases of this kind in which thickenings of the endocardium shewed the impingement of such eddies. The diagnosis is more difficult when the signs of aortic disease are indecisive. In Dr. Fisher's case : " The presystolic thrill and bruit were well marked and mitral stenosis was diagnosed ; but at the necropsy the mitral valve was found quite normal. The aortic valves were healthy also, it is interesting to add ; the aortic regurgitation was due to pouching of the sinuses of Valsalva with dilatation of the first part of the aorta." Dr. Fisher also adduces a case of Dr. Goodhart's to prove that this presystolic murmur may be heard in disease of the aortic valve without audible regurgitation. I think Debove has somewhere published a similar case. It has been suggested that a collision of the aortic and auricular currents may produce this murmur ; but if so, surely Flint's murmur should be the rule. Thayer found it, at some period or other, in about half of his cases of aortic insufficiency. It is said that positive aortic stenosis does not prevent it. Dr. Phear has reported on 46 cases of " presystolic murmur " without mitral stenosis. Dr. Gibbes and Dr. Fisher think it may occur in any dilated hypertrophy ; but in some such cases a long murmuring first sound may have given rise to misconception. Dr. Gibbes, indeed, seems thus to interpret the " presystolic murmur " in all cases. A further difficulty is that aortic disease and mitral disease may coexist, in cases of rheumatic origin not infrequently ; and again in some cases of aortic regurgitation the mitral valve may be involved later in a sclerotic process descending from the aortic. The general characters of the case, the quality of the first sound, usually booming and perhaps never sharpened up to that of mitral stenosis, the figure and site of impulse, the extension of dulness transversely or vertically are to be considered together. In aortic insufficiency, the right ventricle is for some considerable time undisturbed ; however, before the mitral valve is actually forced, pressures may begin to rise in the left auricle and pulmonary circuit, and the right ventricle begin to enlarge. A murmur of actual mitral stenosis hangs closely round the apex. A considerable damming back of the auricular blood would presumably modify the volume of the arterial output. (*Vide* also p. 362.)

Sphygmographic Signs.—The pulsus bisferiens is more frequent in aortic regurgitation than in stenosis (*vide* p. 452). It is often perceptible to the finger on the radial or carotid artery (in 13 of 20 cases, Lewis), better perchance on one arm than on the other ; by the finger it

may easily be mistaken for dicroty, but the second wave precedes the second sound and, on a tracing, the first wave is not higher than the second. D'Espine says it may be palpable at the apex. If it be palpable, Dr. Lewis computes the interval to be $\frac{1}{6}$ to $\frac{1}{8}$ second. A largely bisferient pulse (I am still following Dr. Lewis) signifies a greatly hypertrophied and dilated left ventricle, of whatever causation, with or without an incompetent aortic valve, and perhaps large aortic or general arterial dilatation ; but in any case it can give us as yet no standard of comparison for degrees of disease. It is independent of arteriosclerosis or other complication ; it seems, however, to be associated with a slackening of cardiac energy, as on occasion by pericardial effusion; and the first sound at the apex may be impaired. If, as Dr. Steell says, it may be produced by digitalis, this may be by reduction of conductivity. If these conditions obtain a consequential mitral regurgitation will not obliterate it. It is not generated in the auricle, the wave of which can be seen to precede it, for instance in the jugular vein ; yet as it is visible in the cardiogram, and sometimes palpable to the finger, it is certainly of central origin. Dr. Lewis supposes two types of pulsus bisferiens, with and without arteriosclerosis ; especially in respect of the height of the second wave. This form of pulse may come and go ; in one of Dr. Lewis's cases it appeared for two days only, during which period the heart flagged.

Concerning the dicrotic wave in aortic insufficiency our notions have undergone some modification in the light of a better knowledge of diastolic pressures in this lesion. We were wont to suppose that in regurgitation the diastolic pressures fell very low, so low that the excessive systolic pressures might thus in some measure be mitigated. The experiments of Hugh Stewart, Janeway, Lewis, and others have thrown new light on this aspect of the matter, and demonstrated that so far from diastolic pressures being below normal they run above this level, usually considerably so ; even in free regurgitation with an open periphery the mean pressures may be excessive. On the other hand, Strasburger, in plotting out "pulse pressures," seems to me not to give sufficient consideration to states of the periphery ; the diastolic pressure does not rise in proportion to the high systolic, and "pulse pressure" has far wider limits of variation. Dr. Lewis has clearly pointed this out to us, demonstrating at the same time that the earlier sphygmographic records of diastolic phases were imperfect. He anticipates that the relation of the diastolic level to the abscissa will prove of value in diagnosis. Of 20 cases of aortic regurgitation Dr. Lewis found dicroty in all ; in 9 normal, in 6 exaggerated, in 5 diminished. This wave bore no definite relation to the height of the blood-pressure, to the degree of arteriosclerosis or compensation, or to the length of systole. Dicroty may be well marked with large regurgitations, although we might have expected, if the dicrotic wave were due to recoil of the aorta, that when the bottom of this vessel is out the recoil would be prevented. Dr. Samways urges that the dicrotic wave is due not to a

longitudinal, but to a transverse recoil of the aorta; this might explain
the persistence of the wave in the circumstances we are considering; and
stenosis should promote it.   For the present, however, we must accept Otto
Frank's analysis of central and peripheral waves, and his demonstration,
with a manometer mirror of negligible inertia, that the dicrotic wave is
a recoil from the periphery.   Notwithstanding, then, the arguments, even
recent arguments, concerning the regular disappearance of dicroty in aortic
regurgitation, and those of Geigel and others, who have attributed its
appearance to mitral regurgitation, we must for the present regard them as
based upon fallacious methods.   I suspect that the dicrotic wave and its
place upon the descending limb have no important prognostic value, and
mean no more than degrees of expansion in the periphery.   For example,
I note that in four closely observed cases Janowski found that dicroty
became evident on an access of fever, on the concurrence of Graves'
disease, on mitral regurgitation, and on a suspicion of failing compensation,
respectively.   The coexistence of stenosis might be indicated by an
anacrotic wave in the tracing.   As to degrees of atheroma, it is difficult
to draw any precise inferences from the sphygmograph; the tendency in
such cases is, of course, to a broader-topped wave.

   Pain.—Pain is more common in disease of the aortic area than of
other parts of the heart.   The distress varies from slight stenocardial
oppression to full angina pectoris.   When insufficiency is of acute onset, as
in sudden valve-rupture, the oppression may be intense; but unless the
mischief be of extraordinary severity—bad, indeed, almost beyond hope
—the reserve capacity of the heart comes into play and, as the inflam-
mation in the area subsides, pressures are re-adjusted with wonderful
celerity; thenceforth, until the organ begins to fail, there may be less and
less discomfort.   If it be in elderly persons, the subjects of general cardio-
arterial disease, that angina pectoris, in its major or minor forms, is most
frequently met with, it is by no means confined to them.   Toxic aortitis
often occurs at earlier ages, especially in syphilis.   Dr. Simpson of
Cambridge will remember well the agonising and persistent angina of
an undergraduate, suffering from acute rheumatic inflammation, no
doubt suprasigmoid as well as valvular.   The case of the housemaid
(p. 427) is another example, out of many.   An uneasy substernal oppres-
sion may be the first hint of this complication, and usually it gets no
worse; but as the fires of the rheumatic fever are dying out, returns of
substernal oppression, or transient numbness of the left arm, with slight
rises of temperature, and often with a peculiar anxiety, may betray patches
still smouldering about the root of the aorta.   Of muscular movements
those of the arms seem to be the most efficient in producing anginous
pains; it has been stated that movements of the arms are the most instant
in their effects upon blood-pressure.   Another seat of pain in aortic regurgi-
tation, and this too rather in the later phases of it, is "gastralgia," or a
suffering so described.   This pain, which is of the nature of angina, is to
be discriminated from tabetic pains, and from aches of more superficial
origin.   With the gastralgia too often comes the persecuting flatulence

which besets all cardiac affections, even the functional.   To belch up wind is attended with relief, but it is another thing to say that the wind is the sole cause of the distress.   In some cases this suffering may be attributable to wind-swallowing, but this explanation is by no means generally true.   The atonic distension of the cardiac portion of the stomach, so frequent after debilitating illness, is particularly troublesome after rheumatic fever.

The Nervous System.—The coincidence between tabes and aortic disease is, of course, more than accidental.   Berger and Rosenbach were perhaps the first to point out its connexion with aneurysm, saccular and fusiform, and other forms of aortic disease.   They cited 17 cases from the Moabit Hospital.   Ruge and Hütter found aortic disease in 9 cases out of 138 of tabes (6·5 per cent).   In only one of the 9 was there no probability of syphilis ; in 5 this antecedent was definitely ascertained. Articular rheumatism counted for very little.   Schuster found the association in 18 per cent.   Sir W. Gowers accepts the association as causal, and Grasset and Rauzier are of the same opinion.   Grasset in his recent essay on tabes, in pointing out deep insensibilities (for example, on compression of the testis or the pit of the stomach, or on pinching tendons), suggests that this numbness may explain the remarkable latency of aortic disease in these patients, and the absence of angina pectoris. The reason is of course that both diseases belong to the syphilitic series, and may spring up together before the age of senile atheroma.   Gastralgic and other eccentric phenomena in aortic regurgitation own a tabetic origin more frequently than we always perceive.   How often one wishes a case back again for better investigation !   Once after I had completed my examination and discussion of a case of syphilitic thoracic aneurysm, my colleague in consultation was wicked enough to tell me I had not found out that, although his gait was scarcely affected, the patient was tabetic.   In all cases of aortic disease the pupils and knee-jerks must be tested.   It is alleged that a merely dilated aorta can produce inequality of the pupils.   In cases of doubtful causation Wassermann's test should be applied.   In one case of aortic disease with tabes (Schütze), this test was negative, although the specific infection (thirteen years before) was ascertained ; but in 11 other cases it was positive, and in 10 of these the infection was admitted.   Danielopolu in 14 cases of aortic disease —with and without tabes—obtained a positive reaction in 8.

There are certain other nervous disorders which are, I think, more prevalent in aortic regurgitation than in other forms of cardiac disease. Possibly some of these cases are tainted with syphilis.   In an article on Cardiac Delirium, published in 1885, I said that the sufferers from aortic disease are liable to derangements of mind and temper.   In the stealthier or even latent phases of aortic insufficiency we may note certain mental perturbations which, however, are not unknown in other heart-diseases. The patient may fail to realise his state, or the devotion of his friends and attendants.   Furthermore, we may note restlessness, fretfulness, change in temper, capricious aversions, waywardness even to violence ;

in some cases the restlessness goes so far as to urge the patient to spring from bed, to perambulate the house, or even to jump out of the window. The excitement in such cases of aortic regurgitation may simulate that of alcoholic delirium; and, as in these extremer degrees it occurs chiefly in men, a male attendant may be required. That it is not alcoholic is proved by its outbreak or persistence in patients of moderate habits, or who are, and for some time have been, under continuous observation. The restlessness is often due to a delirium of place: the patient is under the delusion that he is away from home or in a strange house; pacified for a few minutes, or for a few hours, the delusion possesses him again and again with an agitation which is fraught with ill consequences to the cardiac disease. Dr. N. Davies, Prof. Osler (83), and many others have verified these observations. In other cases, as in one of double aortic disease which I saw in 1901 in an atheromatous old man, aged seventy-eight, there is a wearisome frontal or vertical headache with some lethargy, or even stupor. In such cases, as it was in this one, there is no obvious concomitant variation of cardiac rhythm or pressures. This old gentleman, as I heard, became more alert, but the headache persisted. There was no renal disease. One sees many such cases in hospitals and infirmaries. Apart from mental disorder, headache in aortic insufficiency is frequent; and buzzings, dizzy sensations, momentary obscurations of consciousness, twitchings, or even convulsions, may indicate the perturbed conditions of the cerebral circulation; indeed this vascular instability may be perceptible to the patient whenever he stoops. Sleeplessness, not by any means always due directly to the cardiac perturbations, and often troublesome in other cardiac diseases, is especially noticeable in aortic insufficiency. The association of insanity with cardiac disease has been studied in its more general aspects by Mickle, Ball, Fauconneau, and others.

Nutrition.—Dilated as are the peripheral arteries, yet pallor and some falling off in flesh mark another distinction between aortic insufficiency and mitral disease in which the face is congested and emaciation, if present, is concealed under venous turgescence or oedema. So long as the left ventricle is fortified by hypertrophy, and the cardiomotive energy keeps up, there is practically no anasarca or ascites. Filling of the pleural cavities, swollen legs, albuminuria indicate a slackening ventricle and increasing residual blood; the heart is then entering upon those final phases of demolition which are described under Diseases of the Mitral Valve, and of the Myocardium.

Respiratory System.—While the mitral orifice and valve are efficient, the pulmonary circulation, if under some rising pressures, is protected fairly well. But as pressures rise in the left ventricle they thus rise in the auricle, and so in the lungs. With this the right ventricle, usually somewhat hypertrophied, copes for some while effectively, and indirectly may moderate the regurgitation. But a time comes when residual blood begins to burden the right ventricle also, and the equilibrium begins to

rock. Dicroty is no guide in these problems (p. 473). A mitral murmur, however, does not of itself categorically indicate an overwrought yielding ventricle. Before this stage is reached, the invasion of atheroma or fibrous condensation, propagated from the aortic area or due to the abnormal stresses, not rarely causes some mitral murmuring. The conjecture of Traube and Rosenbach that, with a competent orifice, fibrous degeneration of the papillary muscles may cause a murmur by vibration of the cusps at a lower tension, needs but an allusion; if susceptible of proof, it is unproved, and meanwhile has not much support in clinical experience. It is in the final stage of the atony of a jaded heart, or when the mitral valve is crippled, that the bases of the lungs begin to fill; and, indeed, as we see in other cases, in chronic renal hearts for instance, such changes due to rising venous pressures often appear under oppression of the chambers of the heart before a mitral murmur appears. The mitral valve may be forced by sudden aortic regurgitation. In two of my own cases of sudden rupture of the aortic valve by strain, in healthy men, acute dilatations of the left ventricle ensued with rapid irregular pulse, and a mitral regurgitant murmur with cyanosis and pulmonary oedema. In one of these men, who made a temporary recovery, the mitral murmur disappeared. Yet I may repeat that with a competent mitral valve, residual blood in the left ventricle will dam back the venous. Dr. J. Mackenzie warns us not to mistake a jolting of the liver by arterial jerks for expansile pulsation.

Dyspnoea, unless due to anaemia, is scarcely a prominent symptom till this stage is reached. The earlier dyspnoea is rather an inexplicable need or disquietude which the patient himself can hardly describe; if an exact person, he declines to call it shortness of breath; he speaks of it rather as an oppression which impels him to sit up. Otherwise recumbency, which irrigates the brain, is more comfortable than sitting up. If an exacerbation of the lesion occur in the supravalvular aorta angina may intervene, as "stenocardia," or in more explicit attacks. In later phases of the disease, the patient may be seized with what is improperly called cardiac "asthma," when the gasping and shortness of breath may be very distressing. Such cases are usually complicated with very high arterial pressures, of renal or other origin, and the dyspnoea is not the panting of mitral disease; its assaults are less dependent on effort and position, and are more abruptly and furiously paroxysmal. In high-pressure cases the sudden appearance of a streak of fine crepitation at one or other pulmonary base, or at both, is very ominous; rapid oedema may invade the lungs and suffocate the patient within a day or two, or even in a few hours. In these phases constant vigilance is imperative. Indeed, in aortic regurgitation the appearance of ordinary oedema of the pulmonary bases is always disquieting.

If respiration becomes audible a little distance away, and assumes that tubular quality which horsemen call "roaring" or "whistling," the trachea is getting constricted by a dilated aorta.

Cough may be an intolerable distress. When it does not spring from

incidental causes, it is generally spasmodic, and due to this pressure of a dilated aorta, either directly upon the trachea or upon the laryngeal nerves. When due to contingent catarrh expectoration will be increased. In cases of considerable dilatation of the aorta, even without aneurysm, the cough may be agonising. One patient of mine, when he felt an attack coming upon him, would throw himself on his hands and knees ; or the sufferer may anchor himself to bed or table to stay the racking of it.

The efficacy of the *renal function* is in all heart disease a matter of apprehension, and too often of anxiety. So long as the rise of venous pressures is postponed, this constituent of the prognosis is more easily appreciable ; still in certain cases of arteriosclerosis the renal conditions may be very obscure. This is scarcely the place to discuss the differentials of renal adequacy, renal disorder, and renal disease ; but a certain interesting argument of Bard deserves attention. Bard brings evidence to prove that by the jugular curve we may detect an early sign of rising venous pressures ; or again, the intervention of renal disease. In aortic regurgitation he finds the auricular wave to be accentuated, presumably by the effort of the left auricle, and therefore of both auricles, to drive the blood into the left ventricle against the aortic reflux. The wave *a* (of Mackenzie) is elevated, but is not prolonged ; the degree of the elevation offering some gauge of this encounter of blood-streams and of rising venous pressures. Now if chronic nephritis be present, it modifies the duration, and even may modify the time-incidence of the *a* wave ; it is longer in duration, and often a little later in starting (the ventricular impulse being protracted or even delayed also). So far as this comes about, the *a* wave encroaches upon the carotid wave, or may even override it. I can contribute no more to this problem than the caution that pressures may rise in the left auricle if with a fairly good myocardium aortic stenosis supervenes even upon a moderate regurgitation.

Diagnosis.—In cases of uncertain diastolic murmur the absence of thrill, or its distribution about the base, the absence, in the earlier stages, of the shortened first sound of mitral stenosis, of reduplicated second sound, of rise of pressure in the pulmonary circulation, and the constancy of the murmur on changes of position, will indicate that if there be a mitral presystolic murmur also, it is but a " Flint murmur." In the last stage, however, the failing systolic sound may become as short as in mitral stenosis, and the liver may swell. The jerking of the arteries, too, may then subside ; indeed the case becomes virtually mitral. To the possible simulation by venous hum I have alluded. Murmurs of aortic regurgitation may be exceedingly distant or faint, may appear erratically about the sphere of the left ventricle, and may even be inaudible at the aortic cartilage. While listening intently for a faint diastolic murmur the physician should tell the patient to hold his breath, as this suspension may not only remove a competing sound but may suspend a pulmonary whiff, or intensify a faint aortic murmur or a continuous hum. Pulmonary

regurgitation is too rare to bother us. Duroziez's sign may be useful in
an obscure case. I need not emphasise the importance of bringing all
the clinical facts to bear upon the interpretation of the physical signs.
Stethoscopic colours must be "mixed with brains."

In a patient, whom I saw but once, I had some hesitation at first in
deciding whether a chafing diastolic sound at the base was due to aortic
regurgitation or to the pericarditis of chronic renal disease. A study of
the whole case, however, left little doubt of the renal interpretation.
A like difficulty might arise in a rheumatic or a septic case.

Prognosis.—The course of aortic regurgitation is almost always
towards premature death, sudden or gradual. As in all heart diseases,
the main factors in prognosis are four : the age of the patient, his
calling, temperament, and habit of body, the specific kind of lesion, and
the degree of lesion. An accurate knowledge of the history of the
patient and of his symptoms, important as it may be, is not always to be
had. It is difficult to recall cases of aortic simple regurgitation in chil-
dren ; such cases, if rheumatic, would have no doubt a long average
survival, although a deformed valve segment is meet for chronic septic
or other deteriorations. A clean rent in a healthy valve segment should
be a less destructive process than a lesion of equal degree due to disease ;
it is said that a clean rent in an aortic cusp has been known to heal.
Syphilitic disease of the root of the aorta does well under specific treat-
ment, if caught early. The slightest stenocardia or evanescence of the
normal sounds in the carotids are warnings of evil. Mitral insufficiency
is not infrequently cured, aortic rarely. Potain's aphorism that aortic
insufficiency once established is irreparable is too rigorous. I have
quoted from my own notes two cases of cure (p. 468), and the occurrence
may often be prevented. Such successes are perhaps all in syphilitic
cases ; von Leyden, however, has recorded a case of recovery after a
traumatic lesion. Yet even if a murmur should cease the mischief may
be stealthily progressing. As age advances the prospects of life grow
less and less ; if the lesions may not be worse in kind, certainly adaptation
is less ready. In atheroma the manifestation of an aortic reflux signifies
not only progressive disintegration, but also an accelerating rate of it ;
and prognosis is far graver when regurgitation is added to mere obstruc-
tion. On the other hand, equivocal as the process may be, primary
adaptive dilatation must not be mistaken for that of enfeeblement or
myocardial degeneration. Death, probably by vagus inhibition, often
intervenes suddenly, even in a period of latency, primary or secondary.
As an old peasant, in his pathetic way, said of such a case, "Johnnie
went off sudden, like the bottom falling out of a pail o' water." When
the attention of the physician is drawn to the disease by complaints
about the heart, or of retrosternal oppression or anginal uneasiness,
a subacute phase has supervened upon the chronic effects of strain,
rheumatism, syphilis, or atheroma. In strain such sensations, severe
at first, will probably subside more or less as re-adaptations of cardio-
motive functions become established ; but if the patient's life is to be a

comparatively good one, as the reserve capacity of the heart comes steadily into play (secondary latency), they should pass off for some years. Such a sufferer often goes about his work again in ignorance of the fatal rift ; yet, when, sooner or later, he is brought up by some uneasiness about the heart, he is not likely to omit to tell the physician how that on a certain occasion of effort he felt that strange and distressing sensation in the chest which is rarely forgotten. This event in cases of strain may have been as much as five years before the recrudescence ; but usually it is not more than two or three, and in severe cases much less.

The duration of the latent period—primary or secondary—will probably depend more on the degree of insufficiency than on the sound-ness of the cardio-arterial system : for patients undermined by arterio-sclerosis are withdrawing from heavy work on account of virtual age ; and if thus the conditions of nutrition may be less favourable, those of labour are less exacting. If, however, rupture of a valve occur in a man whose arteries are degenerate, the latent period is very brief or absent. In such a case, under my own care, the immediate symptoms of injury never receded at all. When from rupture we turn to an insufficiency gradually established, we find, as I have already said, that too logical a view of the matter is taken by many writers, especially in the division of chronic aortic disease into the cardiac and the cardio-arterial. A long survival is not unusual in cases of general cardio-arterial disease in elderly persons, whilst on the other hand " young cases " often do poorly, and endure for a briefer span than we had reasonably hoped. That the duration of a heart maimed by aortic insufficiency may be at least as short in a young man, in whom an invasion of syphilis has been vir-tually unchecked, as in the old and atheromatous, will be granted ; but such apprehensions are not usually admitted of rheumatic disease, though a proliferative fibrosis, as opposed to mere "replacement fibrosis," may have very damaging cicatricial consequences, and, as Dr. Mackenzie and Dr. Byrom Bramwell (17*a*) have shewn, may invade the bundle of His. It is paradoxical to say that in some of these the outlook is worse than in cardio-arterial atheroma, but the part is not far from the whole truth. However, when the heart and arteries are other-wise healthy, such cases may remain long stationary ; even for thirty or forty years. I have in my note-books not a few perennial durations of (moderate) aortic regurgitation ; though in other apparently similar cases dilapidation was swift and inexorable. The capacity in elderly persons for a fairly sound hypertrophy of the left ventricle is usually much underrated. Even in the presence of dilatation of the aorta, of stiff vessels, or of a "fibroid" heart, one even with callus yet with a good deal of fair muscle in its walls, the crazy machine may last many a year, even, indeed, if the coronary arteries be very gradually obliterated ; though, of course, such accidents as embolism, mechanical or infective, or rupture of the cerebral arteries may intervene. Difficult as it is to apply general rules to individual cases, in atheroma, ten years is a fair duration for a case of aortic insufficiency ; although in temperate and tranquil elders,

we need not despair of a respite until they "be with ease gathered, not harshly plucked, for death mature." Every physician's experience must remind him that to be "harshly plucked" is not the fate of the older of these patients only ; of young men who die suddenly, no small tale die of aortic insufficiency ; for to die of syncope or inhibition, even with a fairly sound heart-muscle, happens to young and old. Death is frequently not by way of inherent incapacity, but by some functional trip, vasomotor or vagal, to the fatality of which a weak or clumsy muscle contributes. With seeming caprice the thread is cut, a quick step into a railway carriage, or a start up from bed ; or the bolt is drawn during sleep, so that the last years of such a life may be happy even in the ending of it. " Many times death passeth with less pain than the torture of a limb; for the most vital parts are not the quickest of sense." To say, then, that the disease in the cardio-arterial cases is " progressive," and in the rheumatic or strain cases not necessarily so, is an academic difference, and untrue even at that : the mechanical dislocation is always " progressive," even if the local lesion be not.

From the beginning the big ventricle, efficient as it is, racks the machine ; the aorta, being made chiefly of elastic tissue and not of muscle, suffers under the thrust, and the means of the heart's nutrition, instead of increasing as demand requires, are gradually sapped. Young patients then may die unexpectedly soon, old ones may live beyond expectation.

Angina pectoris in regurgitation is always menacing by vagus inhibition ; yet if it persists it may be kept at bay by the nitrites ; and, if the bundle of His be intact, the heart may be protected by atropine, perhaps for a year or two ; but the respite is a troublous life—a life of pain, of slavery to drugs, and of bitter adversity, physical and mental.

Other sinister signs are increasing pallor, vertigo, tinnitus, rising frequency of pulse and respiration rate, and arrhythmia even if transient. Or the watchful physician may note that muscular effort no longer raises, but even reduces, the blood-pressure—a bad sign indeed.

As to murmurs ; that a systolic murmur audible at the apex is often not yet mitral defect but a mere emergence of a direct aortic murmur we have seen. Loudness of murmur, other things being equal, speaks in favour of sustained cardiomotive energy ; and, although a murmur soft to the point of indistinctness may be consistent with slight or incipient injury, on the other hand it may be waning with the heart which generates it. A quickening pulse, if not due to temporary causes, is of ill omen, because it probably means a larger residuum at each contraction and ill-filled arteries. We are told that a fall of the specific gravity of the blood is likewise of ill augury. Increase of the area of cardiac dulness vertically may be a good sign ; its increase transversely is a bad one : speaking generally, changes in the chambers are of far more importance than changes in the murmurs ; there is an element of caprice in murmurs which may rise, fall, split, or perhaps vanish for a time, without definite prognostic meaning.

Of intercurrent diseases the infections are the most injurious in their effects upon the lame heart; and of these influenza and diphtheria are the worst.

If possible, "functional" perturbations of a transient kind must be distinguished from changes in the myocardium; but to estimate the value of the myocardium in fairly stable cases of heart disease is very difficult (p. 506). The results of treatment, especially the use of digitalis, may give us some hints of this kind. Arrhythmia, alteration of other sounds, diminution of urine, the appearance of albumin or casts, failure of remedies previously effective, are of sinister meaning. Neglect of treatment until late in the disease is against the patient's prospects; the command of skilled treatment, the means of carrying it out and a docile temper are in his favour.

To sum up: if no error be more gratuitous than prophecy, none is more perverse than false precision: in human affairs we cannot get beyond moral certainties, yet patients or their friends often importune us for a fallible prediction. Given a moderate lesion and good conditions within and without, I should say that, embolic or vagal accidents apart, in a patient under five-and-thirty years suffering from rheumatic, syphilitic, or traumatic aortic regurgitation, sufficient to evoke considerable hypertrophy, the prospect of life is about ten years; rarely more than twelve, save in very static cases, in which the lesion is slight in degree. To persons over fifty, in whom the arteries are atheromatous, and the aortic insufficiency is a leap forward in the work of decay, three or four years may be given; or if the aortic insufficiency be part of a slow development of atheroma about the base of the aorta, and the patient is in easy circumstances, death may be kept at bay for six or eight years. The previous rate of the change in the individual is, of course, an important element in our judgment. If it be syphilitic, instant careful and continuous management will mitigate the disease, and may compass a cure. In obstruction alone the expectation is much longer. If the contracting lesion be syphilitic, the mischief may be arrested and frequently cured; but fibrotic change may encroach upon the tract of His, or atheroma upon the coronary arteries.

Combined Lesions.—To enter into a discussion of combined lesions of the heart would lead to repetition of the work of other contributors; in every estimate of the duration of life in aortic insufficiency the values of the other component parts of the heart must of course be reckoned. It is of the first importance, however, to decide whether a coincident lesion elsewhere, valvular or muscular, is independent of or dependent on the aortic. It is contrary to my experience to assert, as many have done, that coexisting mitral regurgitation is helpful in any stage of aortic insufficiency, except perhaps as a relief to the aorta in the case of angina; that any degree of mitral contraction may be so is incredible. In rheumatic cases, aortic disease usually signifies extensive cardiac damage, and so far the prognosis is the worse.

Treatment.—Give your prognosis on the best suppositions, treat your

patient on the worst. If he be a man of easy circumstances and tranquil occupation, whether in a palace or in a workhouse, he has the better chance of survival. Care or worry, bustle or toil must be buffered off. There are men of such a temperament that they cannot form sedate habits : recklessly, as it seems to the physician, they skip up stairs two at a time ; they puff after trains ; they climb over five-barred gates ; they bounce up from deep sleep to pass water, and so forth : they do not mean to run risks, but such are their incorrigible ways. With such persons discipline must be attained by spending day after day in drill, in gaining self-control, in repressing volatility. In this precaution there is nothing false to a man's best self ; the purpose is to get the most work out of himself before he dies. Persons in toilsome callings must change them ; and spend the perhaps no less useful remnant of their days in some easier duties. A reasonable vigilance may be exercised without the encouragement of hypochondriasis : as some one well put it—find out what you can do, and do it ; find out what you cannot do, and never do it. For such reasons the conditions of survival are more favourable in women than in men ; women are generally more docile to treatment and rule.

The treatment of aortic disease cannot be divided into that of stenosis and of regurgitation respectively ; each disablement has its own consequences, yet the pathological changes are usually of the same nature. Indeed, for stenosis it is hard to see what can be done. Fortunately, as an accommodating left ventricle lies between the left auricle and the aorta, it is not often a perilous defect in itself ; a direct murmur is usually a signal of deteriorating tissues, and calls rather for general than for special remedies. In *diet* we have to look to three points : to the sympathy between the heart and the stomach, to good nutrition of the heart, and to moderation of its work. We must avert indigestion, and administer nutritious food without either raising the arterial resistance or increasing the bulk of blood to be lifted. Indistinctly we are aware that there are diets which increase arterial resistance, and, so far as our lights go, this danger must be eluded. Many elderly sufferers from aortic disease are gouty ; in such persons we shall avoid whatsoever may encourage this habit (Vol. III. p. 159). On the other hand, to reduce the diet below the needs, even of a person who can take little bodily exercise, may carry us into the peril of pining the diligent ventricle ; and to exclude nitrogenous food in order to avoid goutiness we may throw the patient upon a diet of carbohydrates, a diet both bulky and provocative of flatulence and gastric acidity. As indeed in gout itself, a moderated and carefully mixed diet will answer best ; and on two points we should insist—on restriction of liquids during meals, and on thorough mastication of the food, whether soft or hard. Great relief may follow fine chewing and restriction of liquid at meals. Even between meals it is not well to allow the patient to drink copiously ; the blood-pressure will not thus be raised, as for such a result illimitable quantities would be required ; but the output of the ventricle will be

increased, and therewith its work. It is scarcely needful to insist upon the choice of food which is at once easy to digest and worth digesting; yet some foods grateful to the eater are indirectly worth eating if they stimulate his gastric secretions.

Alcohol in all heart diseases is overdone. The immediate relief to the sufferer is often considerable, and as a fillip at a perilous moment indispensable. But as an ordinary article of the patient's consumption, its use is not without drawbacks; it disturbs blood-pressures, its effects accumulate more rapidly for harm in persons who cannot take much exercise, and the perpetual nips, in which too often they are led to indulge themselves, directly induce the venous stagnation and degeneration of myocardium which we are on our guard to avert. But on the other hand, if the patient is cheered by a little light wine or well-diluted spirit with his meals, we may do well to prescribe it; drams being strictly reserved for critical occasions. If on every access of palpitation or faintness the nurse is to run for the brandy bottle, the patient's state will grow worse rather than better. In his work "The Senile Heart," G. W. Balfour has given admirable directions for treatment of such heart diseases, and at greater length than is possible for me in this place.

In respect of management it is difficult to give precise directions. In no cases are tact and experience more valuable. The young practitioner must remember that if, on the one hand, there be a danger of injury from the effects of a careless life, on the other the harmful effects of "valetudinarianism" are no less; and the patient in gaining his life may lose it. We must trim our treatment according to the phases and peculiarities of the individual. Fräntzel well says that to know one has heart disease may be more mischievous than the disease itself. Let your patient understand he has an unstable heart, and that he must rigidly observe your rules of life, but not otherwise fash himself; then to some sensible and trustworthy friend of his tell the whole truth, even the risk of sudden death if such there be; that like other wise men the patient may not omit to put his affairs in order.

In the matter of *exercise* often lies the decision whether or not the patient be allowed to follow his calling. If the occupation be one of muscular labour he cannot but leave it; a working-man must seek some quieter means of subsistence, as a caretaker or the like. A sportsman must contract the field of his pastimes: the salmon rod must give way to the lighter wands of the trout-fisher; cricket to golf: the moors must be forsaken for the stubble and the covert, and the hunter exchanged for the nag. Cycling is by no means an unfit recreation for the subject of heart disease, in early stages, if he will ride circumspectly. Whatever pursuit be admitted, and much will depend on the degree of incapacity, this caution must be remembered on all occasions, namely, that although the sense of oppression which checks exertion, unless very severe, can be "worked off" by perseverance, it is a grievous error for the patient thus to practise upon himself. The heart may become blunted even to persistent strain. No doubt perpetual timidity may be worse than

occasional indiscretion, yet the cry of the burdened heart should not be disregarded. Yet above all do not let him mope, nor indeed become wholly possessed by the blind and ignoble desire of a mere prolongation of days; for under the eye of the physician the heart, a little embarrassed on its first essays, may be gently solicited and cautiously trained up to more efficiency. The physician who inspires moral health into his patient brings comfort also to his body. We, who too often have to minister to unprofitable uses of the world, can proudly point to the examples of high-minded men in our own profession, who have shewn us how to live most nobly when death was treading in their footsteps.

Technical exercises and mineral baths are not so considerable a part of the therapeutics of aortic as of other heart-diseases, but may be cautiously employed in some instances of early and remediable atony.

Drugs.—During the latent period of non-luetic aortic regurgitation those drugs only will be needed which are of common service; special remedies are rarely necessary. Even in cases of acute onset, such as rupture of an aortic cusp, the measures described under the heads of management and diet may be all that is required. Under the unwonted stress, while the heart is pulling itself together, the patient must be kept in bed until the heart has begun to realise its dynamic capacity in the statical condition of hypertrophy. As this is attained a gradual return to the ordinary habits of life may be practicable. Digitalis is not often wanted in this stage; on the contrary, gentle mercurials, gentle salines, a little potassium iodide, and means which moderate blood-pressure are more helpful. In this stage, too much care cannot be given to save the work of the heart in all directions, whether of muscular work, of the digestive and other organic functions, of cerebral and emotional activity. If there be intercurrent times of stress, due either to indiscretion or to some fluctuations of inner health, intervals of more or less seclusion will again be enjoined, and the above measures repeated.

The arguments which have been written upon the use of digitalis in aortic insufficiency are prodigious; it is best for the present that each observer should give the results of his own impressions as simply as possible. Against its use in aortic insufficiency we have the eminent authority of Corrigan; in favour of its use that of Balfour.

I may refer to what was said (p. 440) on tone in a hollow organ, the property of preserving its mean diameter. On increments of pressure we saw that the heart is not dilated passively and at once to its extreme, but by tone is still enabled to maintain its form and a mean if a larger diameter, to obey stimulus, and to perform its peculiar motions. Also that, within limits, increments of load, although they prevent the elevation of the lever to so high a point, are yet consistent with correspondingly stronger contraction; and dynamical reinforcement will take static form as hypertrophy. But if abnormal stresses accumulate too rapidly, or are more than the individual ventricle can cope with, tone is overborne and passive dilatation begins.

In the year 1868, when Dr. Milner Fothergill was the resident

medical officer to the Leeds Dispensary, I placed a large collection of cases of heart-strain under his superintendence, cases mostly of aortic diseases ; and in order to test our remedies for these patients, we carried out together a series of experiments on digitalis, with results which Fothergill afterwards published in his Jacksonian Essay. We demonstrated, on the hearts of frogs and small mammals, those effects of digitalis which are now too well known to need narration here. Suffice it to say that when a solution of digitalis is dropped on a frog's heart we see an increment not of contraction but of tone. The contraction volume is smaller and smaller, till for lack of output the animal is moribund. Now although in aortic insufficiency the regurgitant stream does not exactly "impinge upon the inner wall of the ventricle at a moment of relaxation," for it can hardly be said that the ventricle "relaxes"; yet the pressure is abnormally increased at a moment when the muscle is at the disadvantage of the greater cubic capacity, and when also the direction of muscular motions is coinciding with that of the regurgitant stream.

Tone is then the quality to be watched and supported, and in digitalis we have a means of intensifying tone, of moderating distensibility. But tone, like other qualities, may be in excess. That the residual blood should be reduced is good ; but if the shrinkage of volume goes too far, the output may fall as much below the needs of the system, as it may in excessive residuum. On the body the result is practically the same. Hence one reason why digitalis should be used with precaution is lest diminished capacity come to the same end as asystole ; a second reason is that digitalis may reduce conduction beyond the point of safety ; a third is that if the disease in its course should encroach upon Aschoff's tract, and conductivity be already impaired, digitalis may become positively injurious, and produce retardation, coupled beats, or intermittence of

Fig. 54.—Aortic incompetence with bigeminal pulse due to the action of digitalis. (Graham Steell.)

the heart without diuresis (Fig. 54). But so long as the cardiac muscle, although yielding, is structurally in fair condition, digitalis may be useful and even necessary to counteract distension, and to lessen the volume of residual blood ; if contingent dangers can be avoided this is too valuable an aid to neglect. Now we find, prolonged diastole or not, that in practice digitalis, used with discretion while the cardiac muscle seems sound, and so as to brace the heart at times when tone is slackening, is an indispensable weapon in our armoury ; or, in Balfour's phrase, is "imperatively needed." If, however, infrequency of the pulse, or protraction of the a.-c. interval, suggest that the fibres of conduction are deteriorating, by sclerosis perhaps or by disease of the right coronary artery, we shall regard digitalis with suspicion, or refuse it. Otherwise, the drug may be given experimentally, in single doses, say 10 minims of the tincture once

every second day; the cardiac rhythm and quantity of urine being carefully noted. As to the "prolongation of diastole," in so far as propulsion is better, refluence is less ; in so far as by tone the ventricular diameter is less, residual blood is less, and the auricle has more time to empty itself : moreover, the pause is not all full diastole ; during the first part the pressures in aorta and ventricle are approximating, and during the latter part are indifferent, or reversed. Acceleration of the rate is, generally speaking, an abbreviation of the diastolic period, yet it is rather the sign of the heart's undoing than of its relief. The moment when the tone of the ventricle is adapted to its abnormal conditions, is or may be a very unstable one ; the ventricle is but too prone to dilate beyond the primary and compensatory degree. By empirical rules we must note the kind of distension, and by the watchful use of digitalis restore the equilibrium, so that contraction-volume and output recover their due proportions.

What, then, are these rules ? No one would give digitalis when a big heart is thundering along its course, and the arteries are bounding under its beats. But if the left ventricle be yielding, put the patient to rest with his feet up, and diet him as already prescribed. Gentle deobstruents will probably be required also. Now if under these means the symptoms and signs of dilatation do not abate, and the one dose of digitalis proves harmless, administer another twenty-four hours later, noting the rate and rhythm of the pulse, and of course the volume of the urine ; thus a safe judgment may be made as to the further use of the drug. A pulse over 80, if it does not dictate, suggests the use of digitalis ; on the other hand, we should hesitate to use it if the pulse be below 70. It may be justifiable to try it in cases of arrhythmia if the pulses are quick and irregular. If the ventricular beat fails duly to follow the auricular (Mackenzie's a.-c. interval) digitalis must be distrusted ; a geminal pulse also would be dissuasive. But in later stages, when the right side of the heart is disturbed, digitalis is often helpful. We do not then look too curiously to murmurs or even to valves ; we watch the area of impulse, the areas of cardiac and hepatic dulness, and the volume of the urine.

Finally, as in digitalis continuously administered we see "not toleration but accumulation" (Mitchell Bruce), we shall not stop the drug suddenly, but reduce it after the few days to a much smaller dose. The grave allegation is made again and again that during its use in aortic regurgitation patients die suddenly. Aortic regurgitation is very prone to end suddenly, digitalis or no digitalis ; for this reason we ought, no doubt, to watch its effects even more vigilantly than in mitral disease ; and during the use of substantial doses to keep the patient at rest.

Since I wrote these paragraphs in the first edition, Dr. Mitchell Bruce has studied afresh the action of digitalis in aortic regurgitation, and, with the precautions I have mentioned and he has emphasised, encourages us to make use of it. In one exceptional case indeed he ventured, under these precautions, to push digitalis till the cardiac pulse was reduced to 48, and, as it proved, with happy effect.

In aortic stenosis digitalis is rarely needed; yet even in this malady, if the stenosis be constant or increasing, and the heart yielding, it might be necessary to introduce it occasionally, remembering, however, that, if the obstacle in front be very great, to spur on the ventricle is to ride for a fall.

The preparations of digitalis are so many, and the advantages and the drawbacks of this one and that are now so much better known, that for full discussion of these very practical points I may refer the reader to other pages. The drug, as met with in commerce, is subject to large variations in therapeutical value. Prof. Dixon has shewn in our laboratories that its preparations, as obtained from respectable druggists, vary from 90 to 0 per cent! Most of us get into the habit of using particular preparations; thus, I have some partiality for Nativelle's granules, which I adopted many years ago on the recommendation of George Balfour.

In respect of potassium iodide in stenosis or regurgitation, we have in its use in arterial disease, which is discussed elsewhere, a presumption of its value. In moderate doses over long periods of time it may be useful in "athero-sclerotic" cases. But of far more importance is its specific value in the syphilitic disease of which aortic lesions are so frequent an issue. As we have seen, syphilitic disease of the ascending aorta often precedes this kind of valvulitis; and it is imperative to check it at the outset by that assiduous and reiterated use of mercury and iodide which we prescribe in all forms of syphilis. Angina pectoris or "stenocardia" may betray its presence before the valve is injured; or, as the first sound begins to wane, by vigilant specific treatment we may save the valve, or restore it to competency; but, regurgitation once established, so good a result can rarely be achieved. One of my three successful cases was in a woman, aged forty, in whom miscarriages and anginous pains had preceded the establishment of a double aortic murmur. The left ventricle was enlarging. Under the vigorous use of mercury and iodide of potassium the condition improved, and in eleven months the murmurs had disappeared, the heart-sounds were otherwise normal, and the left ventricle had retired within its proper boundaries. Three months later a healthy child was born. Another was in the male patient of Mr. Wilkin mentioned on p. 456. In this case tabes appeared, but as specific treatment was thus successful as regards the heart, I can scarcely accept Prof. Osler's suggestion that in the tabetic cases the aortic disease also is parasyphilitic. Newton Pitt, Bouchard, and Dieulafoy have also published syphilitic cases in which an aortic murmur or murmurs disappeared. As then in obscure cerebral disease and in angina pectoris, so in other cardio-aortic disorders of uncertain origin, the sufferer should have the benefit of any suspicion or possibility of syphilis, and be treated accordingly.

Of strophanthus in aortic disease I have little experience; generally speaking, I should say that it is more useful in young than in old people; in some patients under thirty years of age I recall cases of heart disease,

though chiefly of mitral regurgitation, in which the drug acted with efficiency. But the preparations of strophanthus on the market are even less trustworthy than those of digitalis.

Arsenic and strychnine may be useful at times when drugs, which should be more directly potent, fail or are inadmissible. If strychnine be prescribed at a critical moment when rapid effects are desired, doses larger than those regularly given are needed. For an adult, fifteen drops of the liquor are not too much to prescribe thus as a single dose. If the patient complain of some slight rigidity the dose is not repeated, and no harm comes of the reaction. Arsenic is more adapted, of course, to chronic medication ; and, whether it act as a nervine or muscular tonic, it certainly has some efficacy. Broadbent regarded the virtue of phosphorus as superior to that of arsenic. Caffeine—the pure caffeine, not the citrate— is an old ally of mine ; it stimulates the heart when it flags, and it promotes diuresis. It is also useful in cardiac dyspnoea. From 1 to 3 grains may be given for a dose ; and in some persons it is better to push the drug early in the day, pretermitting it of an evening lest it disturb sleep. Caffeine is useful as a cardiac stimulant in cases of slow pulse when digitalis is out of the question. If no great precision of dosage be necessary, good and strong coffee, taken black, may be substituted for the caffeine. Theobromine may be no less efficacious. Diuretin is a valuable addition to cardiac remedies, and very often comes to our aid in states of arrhythmia and embarrassment when digitalis is inappropriate.

The nitrites are required when symptoms of an anginous kind arise ; and then as palliatives are invaluable. It is unnecessary in this place to dwell upon their actions and modes of administration. These agents seem to have some anodyne virtue besides the mechanical, for I have seen angina relieved by a nitrite while, by my finger at any rate, I was unable to detect any change in the blood-pressure. Moreover, angina is by no means always attended with high pressure. In extreme cases of aortic disease the assaults of angina may be so frequent that the life of incessant suffering and apprehension is almost more than can be borne ; in these cases the use of the nitrites may become almost a slavery. Apart from angina, the combination of vaso-dilators with digitalis is not to be recommended ; ordinarily in aortic regurgitation the periphery is widely expanded already. Of the use of atropine in angina to prevent fatal inhibition, I have already spoken.

I suppose that chloral is a dangerous remedy in heart diseases, especially in degenerate hearts. Broadbent proscribed it altogether ; Balfour, on the other hand, spoke of the drug with some favour. When chloral was first invented, being less troubled with modern speculations about blood-pressures and undisturbed by the reports of Drs. Gaskell and Shore on chloroform, I used chloral freely in the restlessness of heart diseases, not excluding those of old people. Indeed, to many old people with degenerate hearts I gave the drug for considerable periods, and certainly with great consolation. Distressing, battling nights are full of weariness and peril, and sedatives cannot be forbidden. I now use

chloralamide which, I am told, is safer than chloral; it acts nearly as well. Balfour also, while still clinging a little to chloral, latterly suggested chloralose or paraldehyde instead. Veronal may be useful in the milder cases of unrest. The bromides also are often of great service in the minor degrees of restlessness, but, if long continued, are depressing. All the salts of potassium are to be avoided, even the nitrate. Convallaria, spartcine, cactus, and the like, are known to me only in the blind uses of despair.

Venesection, which is efficacious in some conditions of embarrassed heart, is rarely or never required in uncomplicated aortic disease.

It is now forty years since, in the third volume of *The Practitioner*, I recommended the hypodermic injection of morphine in heart disease; and testimony of the best kind, such as that of Balfour, has been extended to it. Dr. Leonard Hill says "morphine is one of the best vaso-constrictors and cardiac tonics we possess." By the mouth the use of morphine is attended by well-known drawbacks; but hypodermically, in doses beginning at one-eighth of a grain and gradually ascending to a quarter if necessary, it is a precious means of relief. The physicians who still protest against its use are unfamiliar with the practice. There is no remedy which calls forth so warm a tribute from the patient himself, who, after nights of watching and agony, sleeps a peaceful and natural sleep, and awakes almost forgetful of his plight. For the drawbacks to the continuous use of morphine I may refer to the article on the subject (Vol. II. Part I. p. 946). Like any other potent remedy, it must be used seasonably and discreetly; but cyanosis and even some stationary pulmonary oedema are no absolute bar to its administration.

Dr. Morison, for the relief of very large labouring hearts, has suggested, and practised with some success, an excision of the overlying ribs; but the proposal is not yet ripe for discussion.

<div align="right">CLIFFORD ALLBUTT.</div>

REFERENCES

1. ALBRECHT. *Der Herzmuskel*, Berlin, 1903. — 2. ALLBUTT, Sir CLIFFORD. "The Hypodermic Use of Morphia in Heart Disease," *Practitioner*, 1869, iii. 342.—3. *Idem.* "Senile Plethora," *Trans. Hunt. Soc.* 1895.—3a. *Idem.* "Cardiac Delirium," *Prov. Med. Journ.*, July 1885.—4. AMBURGER. "Contusion der Brust, Aortitis (?)" *St. Pet. med. Wchnschr.*, No. 26, 1892.—4a. ANDERSON, H. B. "Aortic Ruptures by Strain," *Brit. Med. Journ.*, 1905, ii. 840.—4b. BABINSKI. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1901, xviii.—5. BALFOUR, GEORGE. *Senile Heart*, 1894.—6. *Idem. Diseases of the Heart and Aorta*, 1876.—7. *Idem.* "Disc. Card. Ther.," *Edin. Med.-Chir. Soc.*, Feb. 26, 1895.—8. BALL. "De la folie cardiaque," *Méd. mod.*, 10 Juillet 1890.—9. BARIÉ. *Arch. gén. de méd.*, March, June, July 1896.—10. *Idem.* "Recherches cliniques et exp. sur les ruptures valvulaires du cœur," *Rev. de méd.*, 1881, i. 132.—10a. BARD. *Semaine méd.*, Paris, June 3, 1908.—11. BARR, Sir J. "Causes and Mechanism of Cardiac Impulse," *Brit. Med. Journ.*, 1884, ii.—12. *Idem.* "Overwork of Heart and Aorta," *Edin. Med. Journ.*, Dec. 1876, and July 5, 1881.—13. BARJON. (Regurgitation without diseased valve.) *Lyon méd.*, 1903, c. 133.—14. BAYLISS, A., und STARLING. *Internat. Monatschr. f. Anat. u. Physiol.*, 1894, xi.—15. BERGER und ROSENBACH. *Berl. klin. Wchnschr.*, 1879, xvi. 402.—16. BERNSTEIN, B. *Vierteljahrsschr. f. gerichtl. Med.*, 1905.—17. BRAMWELL, BYROM. *Diseases of the Heart*, p. 502.—17a. *Idem. Brit. Med. Journ.*, 1909, i. 995.—17b. BROAD-

BENT, W. *Brit. Med. Journ.*, 1908, ii.—18. BROADBENT, Sir W. H., and BROADBENT, J. F. H. *Heart Disease*, London, 1900.—19. BRUCE, J. M. *Brit. Med. Journ.*, 1906, i. 8.—20. BRUNTON and TUNNICLIFFE. *Journ. Physiol.*, 1894, xvii.—21. *Idem.* "The Effect of Resistance Exercise upon the Circulation," *Brit. Med. Journ.*, 1897, ii. 1073.—22. CALWELL and MARK. "Rupture of Aortic Valve," *Ibid.*, 1901, ii. 1736. —23. CAUTLEY, E. "Aortic Regurgitation due to Bicycling," *Ibid.*, 1893, ii. 115.— 24. CHAPMAN, P. M. *Physics of the Circulation*, Goulst. Lect., 1894.—24a. COLE and CECIL. "Axillary Diastolic Murmur in Aortic Insufficiency," *Johns Hopkins Hosp. Bull.*, Balt., 1908, xix. 353.—25. CORRIGAN. "Patency of Aortic Valves," *Lond. Med. Gaz.*, 129, iii. 433, and *Edin. Med. Journ.*, 1832.—25a. CURSCHMANN (and others). Arbeiten (*Path. d. Kreislauf*), 1893.—26. DA COSTA, J. M. "Treatment of Valvular Diseases of the Heart," *Trans. Assoc. Amer. Phys.*, 1888, iii. 216.—27. DANIELOPOLU. "Séro-réaction des affections syphilitiques de l'aorte et des artères," *Compt. rend. Soc. biol.*, Paris, 1908, lxiv. 971.—28. DAVIES, N.W. "Cardial Delirium," *Prov. Med. Journ.*, Nov. 1885.—28a. DEGANELLO. Quoted in *Arch. mal. du cœur*, Paris, 1908, i. 710.—28b. DEHIO. "Myofibrosis cordis," *Deutsch. Arch. f. klin. Med.*, lxii.— 28c. *Idem. Kongr. f. inn. Med.*, 1895.—29. DICKINSON, W. H. "On the Occurrence of Musical Mitral Murmurs in connection with Aortic Stenosis," *Med.-Chir. Trans.*, London, 1897, lxxx. 409.—29a. DMITRENKO. *Ztschr. f. klin. Med.*, 1907.—29b. DUFOUR, C. "Des insuffisances aortiques d'origine traumatique," *Thèse de Paris*, 1896.—30. DUROZIEZ. "Du double souffle artériel comme signe de l'insuffisance aortique," *Arch. gén. de méd.*, Paris, 1861, and *Gaz. hebd.*, 1865-67.—30a. *Idem.* (Trauma of Aortic Valve), *Union méd.*, 1880.—31. EDWARDS. *Med. News*, 1895, lxvi. 548.—31a. D'ESPINE. *Rev. de méd.*, Paris, 1905, xxv. 23.—32. FAGGE, HILTON. "Diseases of the Valves of the Heart," Reynolds's *System of Medicine*, iv.—33. FAUCONNEAU. *De la folie cardiaque et troubles psychiques d. mal. du cœur*, Paris, 1890. —34. FERRANNINI. *Ztschr. f. Heilk.*, 1903, xxiv. 273.—35. FERRIO. "Pericarditis and Aortitis," *Gazz. d. osp.*, Sept. 24, 1905.—36. FISHER, T. "The Presystolic Apex Murmur of Aortic Disease," *Lancet*, 1895, i. 609.—36a. *Idem.* "Rupture of Aortic Valve," *Brit. Med. Journ.*, 1903, i. 421.—37. FLINT, A. *Diseases of the Heart*, 1859. —38. *Idem. Amer. Journ. Med. Sc.*, 1862, xliv. 29.—39. FOXWELL, A. "Case of Aortic Stenosis," *Birm. Med. Rev.*, 1896, ii. 362.—40. FRAENTZEL. *Vorlesungen über d. Krankheiten des Herzens*, Berlin, 1889-92.— 41. FRANCK, FRANÇOIS. *Trav. du Laborat. de Marey*, 1878.—42. *Idem. Soc. de biol.*, Paris, March 28, 1894.—43. FRIEDREICH. "Mal. du cœur," *French Trans.*, 1873.—44. GAEL und LUDOWITZ. *Ztschr. f. klin. Med.*, xx.—45. GERHARDT. "Mechanik d. Klappenfehler," *Verhl. d. Kongress f. inn. Med.*, 1905.—46. GIBBES. "Presystolic Murmurs," *Lancet*, 1901, i. 1601.— 47. GLYNN. "Specimens of Heart Disease," *Liverpool Med.-Chir. Journ.*, 1885.—47a. GOSSAGE. "The Tone of the Cardiac Muscle," *Proc. Roy. Soc. Med.*, London, 1908, i. (Med. Sect.) 146.—48. GOWERS, Sir W. *Diseases of the Nervous System*, ed. 1892.— 49. GRASSET et RAUZIER. *Mal. du syst. nerveux*, 1894.—49a. GRASSET. *Le Tabes*, Montpellier, 1909.—50. GROOM, W. "Rupture of the Heart by External Injury," *Lancet*, 1897, i. 1202.—50a. HABERSHON. Quoted by P. H.-S. Hartley, *Clin. Journ.*, 1902, xix. 254.—51. HAMILTON, D. J. *Textbook of Pathology*, 1889.—52. HAMILTON and BYERS. *Amer. Journ. Med. Sc.*, 1903, cxxvi. 671.—53. HARTLEY, P. H.-S. "Statistics of Six Cases of Ruptured Valve," *Clin. Journ.*, London, 1902, xix. 254. —54. HEIDENHAIN, L. *Deutsche Ztschr. f. Chir.*, xli. 286.—55. HELLER. *Deutsch. Arch. f. klin. Med.*, 1903.—56. HERRINGHAM and WILLS. "On the Elasticity of the Aorta," *Med.-Chir. Trans.*, London, 1904, lxxxvii. 489.— 57. HILL and BARNARD. "Sphygmometer for Clinical Use," *Brit. Med. Journ.*, 1897, ii. 904.—58. HÜRTHLE. *Arch. f. de ges. Physiol.*, 1891, xlix. 61.—59. JANEWAY. "Clinical Blood-Pressure," 1904.—59a. JANOWSKI. *Ztschr. f. klin. Med.*, 1907.—60. KEYT. *Cincinnati Lancet*, March 29, 1879, 224.—61. *Idem. Sphygmography and Cardiography*, New York, 1887. —62. KING, TH. "Extreme Aortic Stenosis," *Trans. Path. Soc.*, London, 1873, xxiv. 40.—63. KING, WILKINSON. *Guy's Hosp. Rep.*, 1837, ii. 122.—64. KREHL. (In the Systems of Nothnagel and of von Mering).—65. LANGWILL, H. C. *Scot. Med. and Surg. Journ.*, 1896, 723.—66. LEARED. "Aortic Valve Diseases apparently caused by Syphilis," *Trans. Path. Soc.*, 1868.—67. LEES, D. B. "Presystolic Apex Murmur due to Aortic Regurgitation," *Amer. Journ. Med. Sc.*, 1890, c. 460.—68. LESPERANCE (of Montreal). "Souffle présystolique dans l'insuffisance aortique," *Thèse inaugurale*, 1891.—69. LEWIS, T. *Practitioner*, 1907, lxxxviii. 207 ; *Journ. Physiol.*, 1906 ; *Lancet*, London, 1906, ii.—70. LEYDEN, VON. *Soc. med. int. Berlin*,

Ap. 4, 1892.—71. LITTEN. *Deutsche med. Wchnschr.*, 1896, xxii. 325, 435.—72. LITTLE, JAS. *Chronic Diseases of the Heart*, 1894.—73. MAHOMED. *Med. Times and Gaz.*, Aug. 10, 1872.—74. MAI. *Ztschr. f. klin. Med.*, 1906, lviii. 393.—74a. MARKOFF, N. "Traumat. Entstehung d. Herzklappinfehler," Diss. Zürich, 1902.—75. MARSHALL, A. L. "Sudden Aortic Regurgitation," *Lancet*, 1908, i.—76. MACKENZIE, JAMES. "Diseases of the Heart," 1908.—77. McWEENEY. *Brit. Med. Journ.*, 1903, i. 251. —78. MICKLE, J. *Heart Disease and Insanity*, Goulst. Lect., 1888.—78a. MOORE, NORMAN. *Lancet*, London, 1909, i.—79. MORISON, A. "Treatment of Aortic Valvular Disease," *Brit. Med. Journ.*, 1896, i. 650.—80. *Idem.* "Thoracostomy in Heart Disease," *Lancet*, 1908, ii. 7.—81. MUSSER, J. H. "Disappearance of Endocardial Murmurs of Organic Origin," *Brit. Med. Journ.*, 1897, ii. 1055.—82. OLIVER, G. *Pulse Gauging*, 1895.—83. OSLER. *Principles and Prac. of Med.*, 6th ed. 1905. —84. *Idem. Angina Pectoris*, 1897, 64.—85. PEACOCK. *Valvular Disease of Heart*, 1865.—86. PETER, M. "L'insuffisance aortique," *Clin. méd.*, 1871.—87. PETIT, A. Art. "Maladies du cœur," *Traité de méd.* (Charcot, Bouchard), v. 93.—88. PHEAR. *Lancet*, London, 1895, ii. 716.—89. PITT, NEWTON. "Aortic Incompetence due to Dilatation of the Orifice and not to Disease of the Valves," *Trans. Path. Soc.*, London, 1898, xlix. 46.—90. POTAIN. "Des traumatismes cardiaques," *Clinique de la Charité*, 1894. — 91. *Idem.* "Souffle présystolique dans l'insuffisance aortique," *Gaz. des hôp.*, Mar. 14, 1893.—92. PYE-SMITH, P. H. "Syphilitic Arteritis of the Ascending Aorta," *Brit. Med. Journ.*, 1896, i. 213, and *Trans. Path. Soc.*, London, 1896, xlvii. 26.—93. RANKIN, G. "Aortic Disease," *Brit. Med. Journ.*, 1907, i. 608. —93a. ROBERTS, LAWTON. M.B. Thesis, Camb., June 7, 1906.—94. ROLLESTON, GEORGE. *Harveian Oration*, 1873. — 95. ROSENBACH. *Arch. f. exp. Path. u. Pharmakol.*, 1878, ix. 1.—96. ROSENSTEIN. "Diseases of the Heart," *Ziemssen's Cyclopaedia.*—97. ROY, C. S. "Elastic Properties of the Arterial Wall," *Journ. Physiol.*, 1880-82, iii. 125.—98. RUGE und HÜTTNER. (Tabes and Aortic Disease), *Berl. klin. Wchnschr.*, 1897, xxxiv. 760.—99. SALLAVARDAN. *Lyon méd.*, 1907.—100. SAMWAYS. Thesis for M.D. Cambridge, 1896.—101. SANSOM, E. *Diagnosis of Diseases of the Heart*, Lond., 1892.—102. SAUNDBY, R. *Edin. Med. Journ.*, 1887.—103. SAVILL, T. D. "Idiopathic Arterial Hypermyotrophy," *Brit. Med. Journ.*, 1897, i. 188.—104. SCHUSTER. *Deutsche med. Wehnschr.*, 1893, No. 41.—105. SCHÜTZE. (Aortic Disease and Syphilis), *Deut. Ztschr. f. Chir.*, 1908.—106. SEWALL. "Steth. Pressure in Physical Examin. of the Heart," *N.Y. Med. Journ.*, 1897, lxvi. 758.—107. SHAW, L. E. "Rupture of a healthy Aortic Valve," *Trans. Clin. Soc.*, London, 1901, xxxiv. 220.—108. SIGNORELLI, B. *Policlin.*, 1904, xi. sez. med. 174.—109. SINNHUBER, C. *Deutsche med. Wchnschr.*, 1904, xxx. 1161. — 110. SMART. "Double Stenosis of Aortic Orifice," *Lancet*, London, 1904, ii. — 111. SMITH, EUSTACE. *Diseases of Children*, 1884.—112. SMITH, F. *Vet. Physiol.*, 3rd ed., 1907.—113. SMITH, S. C. "Digitalis in Aortic Regurgitation," *Brit. Med. Journ.*, 1892, i. 51.—114. STANLEY, DOUGLAS. *Ibid.*, 1896, ii. 1572. — 115. STARLING, E. H. "Pathology of Heart Disease," *Lancet*, Feb. 27, March 6 and 13, 1897.—116. STEELL, GRAHAM. "Pulse in Aortic Stenosis," *Lancet*, 1894, ii. 1206.—117. STEWART, HUGH. *Arch. Int. Med.*, Chicago, 1908, i. 102.—118. THAYER, W. S. "Observations on the Frequency and Diagnosis of the Flint Murmur in Aortic Insufficiency," *Trans. Assoc. Amer. Physicians*, 1901, xvi. 393.—119. THÉRÈSE. "Des aortites aigües et de leur rôle dans les lésions chroniques de l'aorte," *Gaz. des hôp. de Paris*, 1892, lxv. 1237.—120. TIGERSTEDT, R. *Lehrbuch der Physiologie*, 1897, Bd. i., Leipzig. — 121. *Idem. Ergebn. d. Physiol.*, *erster Jahrgang*, *Abth.* 2.—122. TREADWELL. "150 Cases of Valvular Lesion due to excessive fatigue in the War of Secession," *Boston Med. and Surg. Journ.*, Sept. 1872.—122a. TRIPIER. Eichhorst's *Handbuch.*—123. VIERORDT. *Diagn. d. inneren Krankheiten* (4th ed.), 1894.—124. VULPIAN. *Clin. méd.*, 1879, 150.—125. WALLER, A. D. *Human Physiology*, 3rd ed. 1896.—126. WALSHE. *Disease of Heart*, 4th ed. 1873.—127. WEBER, F. PARKES. "Syphilis and Atheroma," *Amer. Journ. Med. Sc.*, 1896, cxi. 531.—128. *Idem.* "Aortic Stenosis with Bradycardia," *Trans. Clin. Soc.*, London, 1897, xxx. 224.—129. WEISMAYER. *Ztschr. f. klin. Med.*, 1897, xxxii. Suppl. 29.—130. WEISS und JOACHIM. *Arch. f. Physiol.*, Bonn (Pflüger's), 1908, cxxiii. 341.— 131. WEST, S. Discussion at Clinical Society, *Brit. Med. Journ.*, 1897, i. 589.—132. WEYRAUCH. *Deutsche Arch. f. klin. Med.*, 1907, xci.—133. WHITBY, C. J. *Lancet*, 1896, ii.—134. WILKS, Sir S. *Trans. Path. Soc.*, London, 1865, xvi. 77.

C. A.

FUNCTIONAL DISORDERS OF THE HEART

By Sir Clifford Allbutt, K.C.B., M.D., F.R.S.

To the purist the vulgar distinction between functional and structural disease is fallacious. We are assured that in every change of function a change of structure is implied ; indeed, that structure and function are one, and to regard them severally is but to see the same thing in different aspects. It is not so much that the materialist and the idealist have lain down together, as that the idealist has swallowed the materialist. This we admit, but we know also that molecular constitution permits of a certain degree of deflection, from which, when released, it springs back to the normal ; and as, therefore, in our studies we conveniently detach physiology more or less from anatomy, so it is with nosology when we analyse symptoms apart from morbid anatomy. At the same time we shall not forget that knowledge thus severally obtained must continually be reintegrated.

Furthermore, we shall not be discouraged from using the term functional disease in a still narrower and more artificial sense,—in the sense of perturbations of a contingent kind, sufficient to disturb but not to alter the moving equilibrium (33). Every beat of the normal heart is a disturbance of equilibrium, and cessation of all disturbance is the peace of death ; on the other hand, disturbance beyond the stability of the equilibrium is death or disease. In health the disturbances are rhythmic, harmonious, controlled ; in functional disease they are arrhythmic, uncontrolled. In functional disease the going system halts or staggers, but not beyond recovery ; the humming-top swerves under a puff of air, or reels over a grain of mustard-seed ; but the deflection is counteracted, and resolved. Such temporary eccentricities are common to the heart with other organs, but in the heart are more conspicuous because its workings are nearer to our consciousness, and lie, moreover, in the track of emotional gusts and typhoons. Is there a man so stoutly knit, whose inhibitory nerves are so powerful and alert, that in passion or "'twixt doubtful fear and feeble hope" he has never felt his heart climb into his throat ? Thus it is that functional disorders of the heart are familiar to us all ; and occupy our thoughts the more, as the heart tells us where the centre of life is, and where we cannot afford to have things go wrong. It may be truly objected, that these are but matters of degree—that persistent functional disease ends in structural disease. But surely this is not necessarily the case. While we need the warning not to overlook sinister "functional" disorders which may be the first signs of the stealthy invasion of structural disease—such as a notable delay of conduction, for example,—we shall not put in the same reckoning contingent,

functional disorders, such, for example, as an attack of dyspeptic palpitation. Whether a disorder originally purely functional by damnable iteration can hammer disease into a harassed organ, *non vi sed saepe cadendo*, it is hard to say, or to say more than that in many cases a lifetime of functional disorder of no little persistency has proved not long enough to bring this event about. On the other hand, it seems no less certain that perennial depressing causes, exile or bondage in an invisible Babylon, may induce degenerative changes in the heart and blood-vessels, or in the kidneys, as I alleged in 1877, and physicians have since generally admitted. Paroxysmal tachycardia, when severe, may wear out the heart; yet such derangements are rather of the nature of dilapidation than of mere disorder: notwithstanding, in particular cases it may be hard to distinguish between a perturbation, such as a variation in rate, which is an indication of intimate heart-failure, and one of central or eccentric nervous, or of toxic origin; yet on the distinction successful forecast and treatment will depend. Anxiety long continued seems to pervert nutrition at its sources; perhaps to prevent healthy metabolism, and to favour auto-intoxication, with damaging effects on certain organs. Such influences, however, belong rather to the remoter causes of diseases of the myocardium than to functional disease of the heart; whilst, on the other hand, some of the conditions of functional heart-disorders must be deferred to the article on Neurasthenia. For our present purpose functional disease may be taken to include temporary irregularities of rate, rhythm, tone, excitability, force, and volume. Such variations may be important features in many grave maladies, as for instance in diphtheria or meningitis, or they may themselves be the leading morbid features, and appear to the patient, and to his medical adviser likewise, to stand almost alone.

A more difficult problem of nosology is to decide where we are to place the quick pulse, say, of larval Graves' disease; if both goitre and exophthalmos be absent, as often they are, can we describe the case as an obstinate functional disorder of the heart? I think that to speak thus would be an abuse of terms; if on due analysis the pulse belongs to the process or series which we call Graves' disease, we shall place it in that category, and not in a diffuse assemblage of mere accelerations. And so on for other frequent and infrequent pulses, we shall distribute them according to the several series to which they belong; we shall no longer allow ourselves to call quick pulses of all kinds "tachycardia," nor all sorts of slow ones "bradycardia."

Again, we cannot consider the heart apart from its nervous connexions; like a well-handled team, its good going depends upon the man on the box. Although the myogenic and neurogenic controversy seems to me to be a mere logical exercise, yet it is true that if the organ be severed from its exterior nervous governance, it falls back into more intrinsic rhythms, whether muscular or neuro-muscular in nature. Inherent rhythm may suffice for less complex organisations, but it will not do for a mammal: for instance, the contraction of the left ventricle,

although always a maximum effort, does not at every beat supply the whole arterial tree. That at a very low resistance, all the arteries being expanded, it might do so is conceivable ; some of the strange perturbations of women attended with heat and flushing may thus come about; yet even in them the distribution must be more or less partial. In health the output is turned now here, now there, as—if I may be permitted so unsavoury a simile—in a sewage farm the fertilising streams are diverted by locks to this way or that. The lock-keepers belong to the nervous centres of the cardiac machinery, and the volume of the arterial pulse in a district varies, not as the output of the heart, but as the temporary diameters of the channels. In study the active brain, after a meal the stomach, demand their alternative streams ; by means of the nervous system anaemic areas call for more blood, satiated areas for less ; and by means of the vagus nerves the heart itself is protected from too great importunity. If then in an anaemic girl the heart beat too fast, we shall not formally call that a "disease" which is an attempt on the part of the heart to respond to the calls from anaemic areas all over her body.

Again, as men vary in other features, so they seem to vary in heart-values. As the heart is in proportion to the skeletal muscles, men have on the whole stronger hearts than women. Many persons seem to be deficient in muscle, both skeletal and cardiac ; they are pursy, climb hills badly, and after much exertion become weary and listless. After the frame of bone and muscle is complete in the adult, though something may be done for cardiac and skeletal muscle by appropriate exercises, no considerable reform seems possible.

Thus, as we extend our explanations we diminish our unwieldy group of Functional Diseases of the Heart. For as we proceed to consider the effect of certain poisons on the heart, as, for example, digitalis, aconite, coffee, tea, tobacco,—we shall scarcely call their ill effects on the heart functional disease of this organ ; we shall turn rather to the articles on these poisons, and regard the cardiac perturbations subordinately, as features of the symptomatic series introduced by the particular agent (Vol. II. Part I. p. 985). The heart is often set on edge by obscure causes which seem to us to be of the nature of poisons ; of poisons, generated perhaps in the body or bowel and circulating in the blood, which irritate or depress the heart directly ; or indirectly perhaps by some obscure interference with the blood-pressure, as in the malady or maladies popularly known as "suppressed gout." But when we come to know what is meant by "suppressed gout," wherein it consists, we shall remove these cardiac phenomena from the section on functional diseases of the heart, and put them in their own place as subordinate phenomena of the peculiar series. How various and baneful the effect of the poisons of certain infectious diseases upon the cardiac mechanism may be is familiar to us all. In diphtheria the heart's action may be reduced "almost to extinction" (Powell) ; and the effects of influenza in the same direction were described by Sansom (35). Syphilis, again, is said to cause irregular heart, as a functional disorder apart from any focus of disease in the heart,

vessels, or kidneys. Such considerations as these, indeed, threaten the very existence of an article on Functional Diseases of the Heart, save in Merklen's general sense of a survey of the behaviour of this organ under all sorts of diseases, including its own. Meanwhile we have to deal with the cardiac disorders as yet unrelated in a somewhat miscellaneous way ; besides, certain of them do seem to have an individuality of their own.

On commencing this article I formulated a pathological scheme, of large brackets and small brackets, of big heads and little heads, which, however, as I advanced, proved too artificial ; the framework of logical categories belied the variety of nature. For really the peculiar features lie less in the kinds of the maladies than in the kind of the person who has them. I have hazarded, therefore, the alternative errors of a desultory conversation. Yet, to begin with, I may consider briefly some of Dr. Gaskell's functional components—tone, rhythm, conduction, contraction, excitation, the full explanation of which will be given elsewhere ; of some of them, as we have yet little definite knowledge, we can treat only allusively. For the application of Dr. Gaskell's theory to clinical practice, we are deeply indebted to Dr. James Mackenzie, and to Wenckebach, Engelmann, Erlanger, and others.

Tone.—The old-fashioned word "tone," in respect of the pulse, has fallen into disuse ; the more is the pity. When I was a student we were asked how a pulse might be for tone ; now if a student be asked such a question he talks about "tension," although he does not clearly know what he means. To measure, or even to estimate roughly, the degrees of tautness of the coats of an artery is a very complex and usually an insoluble problem ; yet to the coats only can the word "tension" apply. The "blood" cannot be tense in any but an abstruse mathematical sense, which no student of this subject has in his mind. Tension is the stress which tends to split the artery longitudinally or transversely ; and such stress is at more advantage when the vessel is relaxed. Tension and tone then stand more or less inversely the one to the other, as we can see more readily in the ventricles of the heart, where it is not till tone slackens that tension begins to tell. We may say, indeed, that one of the chief functions of tone is to counteract the tension which provokes it. A living vessel is not merely an elastic tube or chamber, it is also a tube endowed, both in its own tissue and in its innervation, with vital contractile capacities. How tension acts upon an artery is well seen in aortic regurgitation, in which malady the effects of tension are only too manifest ; in order to facilitate the work of the heart by diminution of peripheral resistance, the arterial tone is frequently almost abolished. In an advanced case of this kind we have but to look at any long artery to see what tension really is; the artery is not actually split, but it is considerably disintegrated. If, in all circumstances, whether tight or slack, there is more or less tension of the coats of the radial or other artery, it would be most difficult to ascertain the degree of it, even roughly.

Difficult, then, as it is to estimate the hydrodynamical factors in the

circulation, tone and blood-pressure are easier to estimate approximately than tension. With some approach to accuracy even the finger can tell whether the pressure be low, moderate, or excessive; though it is only by the sphygmometer that relative degrees of it can be recorded. Tone in a vessel is that which preserves its mean diameter, the due proportion between the extremes of dilatation and recoil, and has furthermore the somewhat different virtue of keeping the vessel well home upon its contents; a character which the finger appreciates with sufficient accuracy for current clinical purposes, if not for research. Therefore when we speak of a pulse of good or ill tone we are not talking altogether of what we do not understand; by ordinary tone we mean that the difference of pressures between the base of the pulse-wave and the apex is somewhere about 35 mm. Hg. Again, when we speak of maximum blood-pressure we are talking of that which we can estimate with fair accuracy; namely, a full systolic pressure of about 110-120 mm. Hg in a young male adult,—conditions which the skilled finger is able to guess at. But when we speak of tensile effects on the walls of a vessel we are talking in the dark; other things being equal, the higher the blood-pressure the more the tensile stress; but until we have allowed for tone the net tensile stress, however considerable it may be, is incalculable. Now, in functional disorders of the heart and arteries tone often fluctuates excessively. Even in the large arteries, such as the aorta, which are not rich in the muscular constituent, tone may be conspicuously in abeyance. The aorta, structurally healthy, largely but temporarily expanded, may throb diffusely in the episternal notch and below in the epigastrium, as observed by Morgagni; even the wall of the chest may thrill as the hand is laid over it, the sounds of the heart may be carried far along the vibrating walls of the larger vessels, and the abdominal aorta may leap like an aneurysm; so that the patient may himself complain of the bounding of slack arteries all over his body. In some such cases, without aortic regurgitation, a capillary pulse may appear. To the finger the radial or other accessible artery may be filled well or ill as the cardiac output may be; but the sphygmographic curve will shew that the due proportions between expansion and recoil are no longer preserved; the lever falls to the abscissa before the dicrotic wave is formed. Yet all this vascular exorbitancy may subside in an hour or two.

Acute transient dilatation in cases of "nervous prostration" is recorded by physicians of repute (Stark, Kress). The volume of the heart's chambers must be continually fluctuating, as repletion varies. After an extra-systolic pause the ventricles must expand considerably. Concerning transient cardio-vasal dilatations during bodily efforts, the reader is referred to the article on "Over-stress of the Heart" (p. 193).

Tone, which in cardiac fibre must be a summation of intimate contractile vibrations, has a fundamental affinity to contractility. Porter and Gossage regard it as a mode of contractility and cardiac reserve. It is the persistence of tone which prevents dilatation; and this endowment, Dr. Gaskell tells us, is innate in muscle, but may be raised or

reduced by the nerves. It may vary under nervous governance, if it persists beyond all nerves. Presumably in the higher animals tone consists in a perennial breeze from the nervous centres prevailing, now here now there, under the functional tides. Thus, in states of general exhaustion it will die down. Some of Dr. Waller's experiments suggest that nerves, like muscle, may have their refractory periods; and the same character has been indicated by other observers; for example, by Richet at the Toronto meeting of the British Association in 1891; but this can scarcely be true of the stubborn vasomotor system?

Rate.—How widely the rate of the heart-beats may vary between extremes is too familiar to need description. In one bed may lie a patient with a pulse of 30, in the next one whose pulse is 170; and these are by no means the utmost extremes. Under Bradycardia and Tachycardia these phenomena will be discussed more intimately in connexion with disorders of the intracardiac motor centres. So long as " sinus-rhythm " is stable, careful observers agree with Trautweiler that the rate does not exceed 170-172 (p. 529), and, except for intercurrent extra-systoles, keeps regular. The most general factor in acceleration of the heart is loss of vagus control, for the vagus may be regarded as the escapement of the arterial train. Loss of vagus control may be relative or positive; the accelerator nerves may be abnormally stimulated, and thus overbear even a normal vagus function; or the vagus may itself be more or less in abeyance, as after a dose of atropine, or in the later stage of meningitis after super-stimulation in the earlier. Again, agents acting directly on the heart itself may either stimulate the vagus, and so slow the pulse, or may overbear its control, so that the pulse-rate may rise; variations in blood-pressure have these effects, an increase of pressure tending, other things being equal, to the retardation of the heart, and a fall to acceleration of it. In functional heart-disorders we are frequently met by problems of this kind, problems sometimes very difficult to analyse; we may remember, however, that controls, being a later development than the functions below them, tire sooner. Vaso-constrictor action seems to be inexhaustible so long as the nutrition of these nerves is preserved; the accelerators are susceptible of fatigue, but the vagi tire before them; thus the accelerating nerves may seem to exhaust the vagi, and to run away with the heart. A summation of vagus palsy and accelerator irritation may presumably occur, when a higher speed would be attained than either of these causes alone could explain; and this may be the explanation of rapid pulse in certain poisonings, infections, and the like; but in fever blood-pressure often falls also by peripheral expansion, or by a reduction of the viscosity of the fluid. In a convalescent from acute disease, yet under no toxic influence, the pulse rate, between standing and lying, may vary at the rate of forty beats per minute. Again, quasi-normal catabolic products may act directly on the heart-muscle, as fatigue or putrescent products seem to do. That states of the cardiac muscle itself are often directly concerned in its rate seems also probable from the clinical phenomena of "irritable heart" (p. 235), a phase which can scarcely be due to fatigue products only.

Conversely fatty degeneration of the heart is often betrayed by retardation of the pulse, but probably only when the atrio-ventricular bridge is affected. One sees occasionally in elderly persons a rather sudden attack of high rate—perhaps at the outset of 130 or 140—with a sense of oppression and much flatulence; the attack subsides, but the heart continues to be too frequent, say 115, 100, 90, for many weeks, though on rest and treatment, especially dietetic, it usually returns to the normal. This being so, I suppose the disorder must be regarded as "functional," and probably due to some eccentric cause which the senile heart is less able to withstand. It is not quite true that "senile acceleration of pulse is always pathological."

Once more : if the heart be severed from all nervous connexions the rate may scarcely alter; but no demand for work can be met (Funke, Hering). In work we have to deal not with the nerves only but also with the cardiac centre in the bulb; a nervous factor which may conveniently be considered apart, as through its efferent fibres it is chiefly concerned in regulating response to the demands of the system. If the bulb be destroyed the heart may go on, even for an hour or two, but death ensues by fall of blood-pressure and loss of the heart's own oxygen. Thus, not in the case of circulating poisons only, but also under the fluctuations of ordinary blood-changes, the cardiac centre is constantly active. Thus in haemorrhage or chlorosis, the extensive anaemic areas throughout the body,—that is, in this case, the afflux of impoverished blood to the cardiac centre,—excite the centre to quicken the heart; a rise of arterial blood-pressure on the other hand stimulates the vagus roots in the bulb, and *caeteris paribus* the pulse is slowed. The name tachycardia, if used for mere rate with no other connotation, is a pedantic word. The name "embryocardia," likewise, is pedantic if it means merely a very rapid heart; misleading if it means that the heart has undergone some reversion to a fetal state, or even that the organ is itself retrograding. Every heart goes "tic-tac" when its rate reaches a certain degree; rapidity, for instance, is not in itself a sign of the heart's undoing but of extreme reflex excitation of the accelerators, due to a diminution of the total volume of the blood, to accumulations of it in the venous reservoirs, to alterations of its viscosity, or to peripheral expansion; besides, the effects of morbid or catabolic poisons upon the medulla no doubt often aggravate the disturbance. A rapid rate does not necessarily mean an increase of total work done; on the contrary, in mere increase of rate without valvular disease, hypertrophy is far from being the rule, although dilatation is the common result of atony. Indeed, acceleration tends rather to diminution of output per unit of time, and to the increase of "residual blood."

Finally, in an obscure case of altered cardiac rate all possible causes of local interference with nerve strand or nucleus, or with Tawara's fibres, as by neuritis or fibrosis, by pressure of a gland, gumma, aneurysm, or neoplasm, by tabes, and so forth must be discussed; and laryngoscopic and other means of detection applied. To ascertain how far effort, and

the upright attitude, or a slight muscular exertion quicken the rate is a ready test of vascular resistance; for Dr. Waller's electrotonic work enforces the axiom that increased capacity is associated with diminished susceptibility to contingent impressions. The acceleration on rising from the chair normally continues at about 5. Continuous rises of 15 and 20 are abnormal.

Abnormal rates of the heart depend then on many factors, and the variation of any one of these will modify the action of the organ under observation.

Rhythm is not synonymous with rate, as is too often assumed by hasty writers. Rhythm is not the rate but the proportions of motion, insidious alterations of which may escape the unaided finger. As Dr. J. Mackenzie says, we cannot estimate it without the records of the sphygmograph. On the other hand even this instrument is incapable of recording very rapid or irregular pulse-waves, as too many published tracings bear silent witness. A true rhythm, scarcely attained before the comparative perfection of the human system, depends upon the harmony of a large number of component rhythms; of five at least in the heart itself, besides those blending in from the respiratory, nervous, and other systems. The wonder then is, not the liability to occasional discord, but the common concord. A normal cardiac rhythm should consist not only in virtually the same number of beats in each unit of time, but also in identical values; thus, as in the case of rate, we learn that the rhythm of a given period is due to a composition of causes which are not always easy to analyse. Arrhythmias of similar or even identical form may thus come about in different modes; so that, admirable as his work is, I find it difficult to attribute much practical value to Wenckebach's division into essential and incidental discords. Physiology and clinical experience tell us that intermittence of the heart may be due directly to the cardiac muscle itself, or may be a compound effect of influence on the heart and vagus together. Now vagus influence may be eliminated by atropine; and it is often of the greatest importance in diagnosis thus to ascertain the proportions of these factors; for instance, a notable *pulsus alternans* may signify changes far graver than a crowd of extra-systoles : these being generally "functional," those generally "organic." Perhaps no form of arrhythmia is definitely indicative of organic disease, not even a short upstroke following an extra-systolic pause and the effort of a reinforced beat (Mackenzie). I think we must admit that even such irregularities are in many cases by no means easy to interpret. In the three varieties of extra-systole the abbreviated wave begins before the curve falls to the common ordinate, in pulsus alternans after it has reached it. The moment before an extra-systole may be too brief for the ventricle to fill sufficiently to send a wave to the wrist; it may fail even to raise the semilunar valves, in which case there is no second sound, yet an extra-systole is usually or always audible, often sharp. So in the common functional pulsus geminus, as definitely distinguished from the graver pulsus alternans, the younger twin may not reach the wrist. In some

cases these extra-systolic interpolations, of different values, may so tumble into and over each other as to make an analysis very difficult, even with the sphygmograph ; yet upon this analysis the decision between functional and organic disease often depends. After an extra-systole the pause should be prolonged by summation ; if it is not, degeneration may be present. Notwithstanding, that pulsus alternans may occur in healthy persons I feel sure. Digitalis may produce it. In my own pulse at times of stress, such as mountain climbing, I have often felt short beats apparently independent of any extra-systolic phase ; and this before my myocardium could be suspected of senility. An overcharged auricle might be the cause. Conversely, although extra-systole is usually due to transient and comparatively harmless interferences, it is a common, if not a crucial, feature in the senile, degenerate, or fatigued heart. In old hearts the liability to arrhythmias accumulates. Again, the rate may be retarded by passing causes. We have seen that vascular, nervous, and cardiac rhythms combine in the pulsation. Our periodical sensations of hunger, for instance, are probably due to diurnal rhythms of splanchnic dilatation ; these in sensitive persons cause " sinking feelings " which may be represented in the pulse. Coffee or tobacco by vaso-constriction raise the arterial pressure and remove the sensation.

Intermittence at the wrist due not to abortive extra-systoles but to uneven heart-beating is no doubt of grave significance. This irregularity is not so much an alteration of the rate as of volume and energy ; the ventricle delivers variable quantities of blood with variable impulse ; the output is unequal. It seems probable that this kind of irregularity, as in mitral disease, depends upon fatigue or degeneration of the auricle, which lets its contractile initiative fall to the auriculo-ventricular node (Mackenzie). There may be, as in cerebral disease, for instance, an irregularity of time only, the volume and energy remaining constant : but such a condition is momentary ; if equal quantities of blood are not delivered from the several chambers in equal times, inequalities in distribution must accumulate. The size of the pulse depends on the two variables, output and tone of artery. Strictly speaking, no pulse is equable, as a time-line at the foot of a sphygmographic tracing will prove. Janowski's recent laborious measurements shew that in health the irregularities of wave are not inconsiderable. The respiration perceptibly disturbs the order, as does muscular effort or even attention ; especially in nervous or otherwise unstable systems, in which the respiratory tides are more evident in the sphygmogram ; indeed, on deeper respiration these may become palpable at the wrist. The "pulsus paradoxus" is the extreme of such interference. There are also obscurer fluctuations, detected by Hering and others, which probably depend upon vasomotor tides. Clinically, however, by pulsus alternans we mean a shortcoming which the finger can perceive. In some persons the pulse is habitually irregular, in the clinical sense. Sir Thomas Watson mentions such a case in a brother of his own ; whether the brother was a tobacco-smoker or not his distinguished kinsman did not record. In my own experience

I have often met with a palpably unequal pulse in smokers,—never, I think, in the normal state. In acute disease irregularity, as it generally means irregularity of output, if not reducible to extra-systoles, warns us of evil; probably of distension of one or both ventricles; on the other hand, an arrhythmic pulse, even with occasional symptoms of exhaustion, may be consistent with long life, especially if by auscultation and graphic methods the disorder can be analysed into extra-systoles. In some elderly men in whom there is no appearance of gout or other such cause the rhythm is much and permanently disturbed by extra-systolic groups or caprices. The cause is unknown, and the significance little understood. Sometimes they indicate that the blood-pressure is high, when they are probably of ventricular origin. In the case of an old friend under my occasional observation for the last five or six years I have not yet made up my mind whether his attacks of arrhythmia and languor are significant of myocardial deterioration or not. In his better intervals he can hunt quietly twice or thrice a week, and year by year is getting better rather than worse. On the whole, musk has done best for him. It is incorrect to say that if ordinary intermittences arise in advanced life they necessarily signify incipient cardiac degeneration; equivocal they are, yet even in cases when the symptom has endured for two or three years in persons of sixty years and upwards, careful attention to the diet, and a vigilant supervision of the use of coffee, tobacco, and the like, may remove them; on the other hand, even in young persons, intermittence may accompany vascular deterioration, cardiac strain, or valvular disease.

In acute disease, however, intermittence, even of the ordinary extra-systolic kind, is often of grave augury, especially in cerebral disease, in infections, such as diphtheria, and in the pulmonary attacks of the elderly or weakly. On the other hand, in dyspepsia, in obscure nervous irritations, in gout, in smokers, and even in persons in whom no flaw is to be found, it is almost trivial. I once found this kind of intermittence in two brothers who came together to me for life insurance; both of them were very angry with me for refusing them, or rather for stating the facts which led to their refusal. In neither case was there abuse of tobacco, alcohol, tea, or coffee. They were vigorous young men, their digestions good and their teeth sound. The intermissions were occasional, on an average about one in twenty or thirty. Perhaps no one passes through life without occasional cardiac intermissions, usually perceived on the rebound of a ventricle reinforced by the prolonged pause. In aortic regurgitation it has something more sinister about it (p. 481). Another curious but common sensation is a *flutter*, felt rather in the epigastrium than about the heart. This elusive kind of flutter seems not always to be cardiac; there may be some alternative machinery for its production, perhaps by the diaphragm: sometimes certainly it is due to a brief series of rapid and irregular beats, but when the disturbance is more persistent, its nature is more easy to determine. In my own person, if it be cardiac, I have never been in time to catch it at the wrist; when

the finger gets there the pulse is going demurely enough. It is well perhaps to remark that such irregularities intervene occasionally in the healthiest and steadiest systems. They are apt to attack women at or about the menopause. Like the intermittence often associated with it, flutter is ordinarily of dyspeptic origin ; and the best remedy for these discomforts, for they are little more, is to insist on slow mastication ; they are very apt to arise in persons who bolt their food. If a patient complains of his heart, examine his stomach.

An intermittence is frequently rhythmic ; it will recur in twins, triplets, quadruplets, and so on for a while ; such a rhythmic intermittency is often found in persons under the use of digitalis. Before discussing any arrhythmia, it must be asked if the patient is taking, or recently has taken, digitalis. It is said that an intermitting action which does not reach the consciousness of the patient is of worse omen than that which attracts his attention, a partially true saying founded no doubt on the presence or absence of the reinforced beat, a bounce which does not occur in that graver radial lapse which is not an interference but a backsliding ; an intermittent slip which should be distinguished by another name. But the bounce, or beat reinforced by the longer pause is often unperceived, though by some over-sensitive persons it is felt so acutely as even to leave behind it a sense of exhaustion. In the intermittences of acute disease, as of senile bronchopneumonia, the missing beat, even if a well-marked extra-systole, is not usually perceived by the patient.

Concerning the nature of reduplication of first sound or second sound all that can be said here is that either is consistent with mere functional cardiac disorder. A few months ago, in a case of tea excess with prompt recovery, I found reduplication of the second very distinctly, with great rhythmical disorder probably reducible to extra-systolic forms ; and it is not rare in Graves' disease.

Nervous Shock.—By the earlier medical writers, not by the poets only, it is said that intense emotion is perilous to the heart. On a scientific consideration of this belief we must divide the question : we shall first consider injury due to interference more or less direct with the circulation itself, as, for example, by such efforts of the inspiration as to force and arrest the intrathoracic negative pressures to an extreme degree ; and, secondly, indirect interference, as by inhibition, through the nervous system. Of the first kind of case I remember a strange example in the West Riding Asylum at Wakefield. A woman, afflicted with violent mania, one day in a fury held her breath preparatory to an outburst ; she became livid, fell to the ground, and died. At the necropsy it appeared that death was due to extreme fulness and dilatation of the right heart and venae cavae ; though it is possible, of course, that it was due to a fulminating shock arresting the auricles by way of the vagus. A case is reported by Feilchenfeld, verified by Senator, of a girl, thirteen years old, in whom after an hour's violent laughter, dilatation of the heart set in with dyspnoea, cyanosis, and other characteristic symptoms. Recovery

was tedious, but complete.   Montaigne's tradition (Book I. Chapter II.) that "Diodorus the logician, being surprised with an extreme passion or apprehension of shame, fell down starke dead . . . because he had not been able to resolve an argument," would not, I fear be acceptable as evidence.   Of death through the heart, clearly dependent upon mental shock alone, we have little experience.   All that we know, as yet, respecting such nervous influence on the mechanics of the heart, is that vagus irritation, by diminishing auricular contractions, slows the heart and retards its output.   For the heart this is a conservative function, but even in a healthy adult it may be carried too far, even to death.   The depressor effect of dilatation of the splanchnic veins could scarcely menace the heart itself, whatever the syncopic consequences.   In so far as the accelerator nerves are stimulated by emotion, ·the rate of the heart would be quickened ; but if, as we may presume, the output is proportionally less, and the resistance less rather than more (if the depressor be influenced also), no excessive mechanical stress would fall on the pump. Augmentor action is too little understood to allow us to say much about it.   Intense emotion attended, as in fright for instance, not with inhibition but with a universal or very widespread constriction of the peripheral vessels, might perhaps force up arterial pressure to a dangerous height ; emotional constrictions, however, are probably very transient ; generally speaking, in emotion the tide turns to relaxation of these vessels, and of the sphincters, and the blood-pressure falls.   However this may be, we see no perilous cardiac distress during a severe rigor, as in an attack of ague. It is no unreasonable surmise that deterioration of the nerves or their centres, due to prolonged mental distress, may be followed by degeneration of the cardiac muscle ; and it is not improbable that such decay would be attributable to perverted metabolism and impurity or defect of the blood.

Submammary pain, if not logically is practically a part of this chapter, as rightly or wrongly it besets the mind with apprehensions of cardiac distress.   Whatever its origin, it is often associated with depression of spirits.   It may be a referred pain of cardiac origin, it is true ; usually, however, it has no such significance, and in any case has little or no diagnostic value.   If, unfortunately, there be with this pain some indifferent defect of the heart, the patient may dress up a bogey which forbids him to listen to any encouragements.   In one sad case the "hypochondriacal" pain associated with a relatively harmless mitral murmur of sclerotic nature, led more than one physician to give a diagnosis of angina pectoris !   In spite of all protests, the patient, a healthy man enough of some sixty years of age, threw up a good position and fell into chronic invalidism.   The pain of angina is substernal, very rarely submammary, perhaps never exclusively so.   In women, in whom this pain is very common, an injudicious suggestion of "weak heart" may be very unhappy.   If the pain be, indeed, a referred pain of heart disease, we may depend upon it there will be other and far more certain grounds for the diagnosis.   On the other hand, so frequent is this pain

as a companion of functional cardiac disorder, one may say paradoxically that palpitation, sighing or panting, and submammary ache, are rather a presumption against heart disease than for it.

Of such attacks the following is a good example :—W. J. W., aged thirty-two, farmer. Past health good. No worries. Comfortably married. Temperate in all habits. For indefinite time subject to left inframammary pain. In June 1908, and on four occasions since, he found in the morning the left arm growing peculiarly weary and slack. To use it caused unpleasant sensations. This passed to right arm also. Worse as day gets on. In evening, sudden ague-like seizure ; is cold, and jaws tremble. The inframammary pain becomes very severe, and also an abdominal sinking sensation, which ascends to his throat and suffocates him, to the dread of death. Heart beats violently, and he is generally prostrate and ill all night. Recovers in a day or two, and returns to full work till it recurs. No substernal pain ; no radiation to arms. Aspect normal. Physical examination wholly negative. No arteriosclerosis nor high pressure.

A very intelligent patient once made the interesting remark to me that during attacks of this pain, to which he was subject, he was never able to perceive the beating of the heart by the touch, which otherwise he was always able to detect. He had no palpitations.

Hysterical dyspnoea is again a subject for allusion in this place, lest it be regarded as of cardiac origin ; an error by no means always avoided even by physicians of repute. This dyspnoea may, indeed, be startling to an observer not familiar with it ; but to a close observer its capricious incidence, the inconsistencies in detail, the absence of cyanosis, of pulmonary oedema, of venous stasis and so forth will betray its true nature. In these cases, as also in emphysema, in early stages of arterial hyperpiesis, and so on where the malady is not cardiac, or so only in a secondary sense, we must not be afraid of pronouncing for an acquittal of the heart, if a careful examination reveals no definite evidence against it.

Intrinsic Heart Value.—Speaking empirically, and with due regard to the broader features of each, we have presumed that in the majority of these chronic cases there is little difficulty in segregating the functional and the organic diseases respectively ; those, on the one hand, with statical change in which *restitutio ad integrum* is out of the question, and those wherein the disorder, however stubborn, is not one of static change, and, if uncomplicated, *restitutio ad integrum* is possible, and indeed probable. Yet a few cases will remain in which the heart's sphere of reserve, the intrinsic value of the myocardium, may be appreciable with difficulty, if at all ; for example, case, p. 502. For instance, we are told that even in attacks of acute rheumatic carditis so slight and transient as to do no apparent harm, strands of fibrosis may creep into the walls of the heart, and intermingle with the proper muscular fibres. Some of these, as Dr. Mackenzie notes, may insinuate into the auriculo-ventricular isthmus (bundle of Kent and His) and, by retraction, or by the furtive growth characteristic of the tissue, interfere more or less seriously with that

vital fillet. As, therefore, we edge off from the more definite classes of functional disorder, we are drawn almost imperceptibly over the border into classes of inchoate or insidiously extending organic disease. It is manifest, then, in borderland cases, especially in obese or elderly persons or in those who have suffered from some infection apt to poison the heart, that before dyspnoea or other tell-tale symptom reveals a forward stage of disease, a timely test of intrinsic myocardial integrity would be invaluable to us.

To take two recent instances out of many from my notes :—Male, aged forty-five, hard-worked professional man. A few years ago had influenza, when his "heart was poisoned a bit." Is getting fat about omentum, etc. At end of the day's work, or on exertion, is fagged and short of breath ; but he is out of training and smokes rather heavily. First heart-sound at apex rather ill expressed, especially in muscular part of it ; second sound a little loud at apex, which is a trifle outside mid-clavicular line. Arteries normal. No arrhythmia. Recovery, on regimen, etc., satisfactory ; but could not be confidently anticipated.

Female, aged twenty-eight, seen at Baldock. No cardiac reserve. Dyspnoea on effort. Pants on the gentlest ascent of stairs. Not anaemic nor neurotic. Four months ago diphtheria, mild but genuine. Cardiac disability since. Spare person in whom physical appreciations are not difficult. Heart unquestionably dilated, chiefly to left. No murmur. No albumin. This case closely resembles those skiagraphed by Dietlen.

Unfortunately no trustworthy gauge is as yet within the resources of clinical practice ; but we may conveniently glance at the tests at present under investigation : (*a*) physical signs ; (*b*) skiastic methods ; (*c*) manometric methods ; (*d*) jugular tracings ; (*e*) the electro-cardiogram. Besides these there are Vaquez' salt test, Sahli's instrument, of which I have been unable to obtain full information,[1] Katzenstein's test, and Gräupner's test. The salt method consists in a measure of the rate of the excretion by the urine of a known quantity of salt (10 grams) administered to a recumbent patient ; an interval said to be in inverse proportion to the heart's capacity. I have no experience of this method, which would seem to be related to the salt deprivation treatment of dropsy ; even if wholly extricable from retained salt in the body, it seems rather a test of blood and lymph distribution than of essential cardiac virtue. Katzenstein's test consists in the response of the heart to bilateral compression of the femoral arteries. This process is so unpleasant that the effect is marred by the psychical reaction. Oertel's test by percussion-area after effort is of course full of fallacies, if used for more than the roughest appreciations. Gräupner's test depends upon the degree of the phase of secondary rise of arterial pressure on prolonged cessation of exertion. I cannot invite the reader to rely upon sphygmometry of such refinement. Estimates based on the fluctuations of the pulse, in various positions of the body, depend

---

[1] I have tried to obtain the instrument, but have not succeeded. It seems to be after the fashion of the Riva-Rocci manometer, but with a peculiar lever on the cuff, which is said to give records of absolute heart-values.

on too many variables to be trusted for the record of any one of them; such records may be useful in school tests and the like, but depend much on tone (*vide* art. "Over-stress of Heart," p. 216). Haldane and Lorrain Smith's well-known respiratory method is said to throw indirect lights upon cardiac capacity, but the process is, as yet, too elaborate for ordinary medical practice. The a.-c. interval is not a constant; it may vary with toxic causes, or with the prosphygmic duration; and at most is a test only of a part of the myocardium. Crepitation at the bases of the lungs belongs to the more obvious degrees of failing heart when diagnosis is no longer doubtful.

From manometric methods, unless Sahli's machine prove more precise than at first sight seems probable, we cannot get the information we want until we know more of the physics of the subject. As yet we can rely upon records of systolic pressure only, which, taken alone, tell us nothing of absolute or ultimate values. A rough method of my own is to play upon the brachial artery with one hand while the other is on the radial pulse; when by pressure on the brachial the pulse at the wrist is nearly suppressed, the constancy of the pulse can be tested by slowly raising and lowering the arm, and by noting if all waves come through of fairly equal magnitudes. Yet a pulse variable in this respect is consistent with a sound heart. Skiastic methods, orthodiagraphy especially, certainly ought to give us more definite evidence of the dimensions of the chambers, and so inferentially of tone and of relative intracardiac pressures; but even in these pictures more than one opening for fallacy is admitted; in any case these dimensions do not give us what we want, namely, intrinsic myocardial values. The electro-cardiogram (Einthoven, Kraus and Nicolai, Strubell) is a record of which we may have high expectations; but as yet it is impracticable for the consulting room. If it be true that each person has his own cardiogram, it may not be easy to plot out a standard tracing. For such purposes as this its data should be placed side by side with those of Otto Weiss's method of recording cardiac tones and murmurs. The auriculo-ventricular interval may be prolonged by temporary causes, such as drugs, toxins, etc., so that protractions have no negative value. More importance attaches to the measure by the sphygmograph of the second and third beats after an extra-systole (Mackenzie), which by shortcoming may signify, as ordinary pulsus alternans may do, degrees of impaired contractility. Hoffmann and Wenckebach have made like observations. The rough rule that, if by exercise an irregular or grouped rhythm is dispelled, we may attribute the arrhythmia to contingent causes, is a good one for ordinary purposes. And the converse is no less useful: that an arrhythmia provoked by exertion forebodes misfortune.

This is not the place to discuss the large subject of physical signs. It is difficult to conceive how any physician versed in the multitude of conditions which meet us in the consulting room could credit the physical signs with the rather fantastic values attached to them by the Nauheim school. We all know too well the embarrassments due to the variable levels

of stomach and diaphragm, to the form of the thoracic cage, to the volumes of the lungs, to the adiposity or ossification of the chest-walls, to the length of a diseased aorta, to the swinging hearts of neurasthenia, and so forth. One is often told that an altered timbre of the first sound indicated a "thin-walled ventricle," when its clang depended only upon a ballooned stomach lying just below it; and still more often, when an emphysematous lung overlaid the heart, that the first sound was "weak." "Weak action" is assumed on the slenderest grounds. Again, what physician has not felt himself nonplussed when consulted as to cardiac value in a fat and pursy patient, especially a deep-bosomed woman, and admitted, at least to himself, that for finer discrimination physical signs too often leave us dissatisfied? A repetition an hour or two later of the most careful appreciations of this kind will often prove to us how transient is their value. A boy was brought to me two years ago because his home physician had reported the heart's apex outside the nipple-line, and, inferring strain with hypertrophy, forbade the boy his games. On stripping him, a slight kypho-skoliosis was apparent, and by the cyrto-meter a definite lateral flattening of the left chest. The heart was not hypertrophied but harmlessly displaced; the youth was restored to the football field, and, as I have lately heard, with impunity. None of us can be so foolish as to undervalue the great services which physical signs render to us daily; but, even if they do reveal organic disease, I urge my pupils even then to ask themselves, "What about function? Where is the blood?" Conversely, a diseased heart often eludes all our inferences of dimension, sound, graphic curves, skiagraphy, and the rest; and, what is worse, these cases are often the most dangerous in kind with which we have to deal.

In respect of hysteria or of tobacco-poisoning, the visual fields will be mapped out. A point of scotoma for green or red, or for both, between the blind spot and the macula will be sought for. Tea or coffee is often the cause of tumultuous extra-systole. But after all many cases will have to be watched for a long time, and the treatment cautious and tentative in respect of baths, exercises, and the like, until by accumulation of signs and symptoms we may be enabled to infer the facts and so to act more decisively.

Cor Mobile.—It has been ascertained by many careful observers that the normal heart in the normal body shifts a little on change of bodily position; but this normal shift is small, it should not exceed an inch or an inch and a third (2-3 cm.). In thin persons the shift is a little more apparent; but if the mobility run to 3 inches or 4, it is aberrant, and even morbid; if not due to static disease, such as elonga-tion of a diseased aorta, it may signify atony of the parts due to some debility or nervous disorder, such as neurasthenia. There is danger lest such a deviation, especially if associated with an irritable beat, should impose itself upon an unwary observer as hypertrophy; a deception which might be strengthened by a high position of the diaphragm, another variable which is now receiving fuller consideration. We

see by the x-ray-screen that the diaphragm fluctuates widely, both with the respiration and the volume and position of the abdominal organs. In organic as well as in functional disorders of the heart the position of the diaphragm is too often overlooked; if much carbohydrate food be allowed, and digestion be imperfect so that carbonic acid gas is disengaged in considerable quantities, the diaphragm may be so tilted upwards as to embarrass the heart seriously. Attention to this point, whether in heart disease or in temporary perturbations of the organ, as Broadbent was wont to impress upon us, is amply rewarded. But the heart itself may drop considerably. Aug. Hoffmann, de la Camp, and Rummo were among the first to describe cases of "cardioptosis," and Hoffmann and de la Camp noted definitely the part played by the diaphragm in many of them. Wenckebach has shewn, by skiagrams and otherwise, that in extreme lapse of tone, with flaccid abdominal walls, the heart may dangle from its own vessels (cor pendulum), and may even tug at the trachea on each systole. In these cases the cervical veins may swell on deep inspiration while the radial artery diminishes. As we shall see presently (pp. 514 and 518), in atony of the splanchnic vascular area the radial pulse may, on raising the patient upright, lapse even to nothing.

Palpitation.—This disorder, which is to be distinguished from disorders of "nodal origin" (*vide* Paroxysmal Tachycardia), is even more common than intermittence; in greater or less degree it lies within the experience of every one during the more emotional stages of life. In palpitation of auricular rhythm the rate does not exceed 150-170 at most (p. 529), and irregularity is usually due only to extra-systole in some form or another. It is more common in women than in men; and in women is often a very distressing and persisting torment. In the later article on Neurasthenia some of these cardiac disorders will be discussed in that relation; but there are many "nervous hearts" without neurasthenia. Under the alarm of a severe attack of palpitation, with its no less painful sense of choking, even long and trying experience is scarcely enough to steel the patient against the dread of its return (*vide* case W. J. W., p. 505). Indeed, as the gale in which the heart is caught often arises from the quarter of the nervous system, the apprehensions are disordered as soon as the heart itself, or before it. A sensitive woman, physically courageous perhaps, yet one who starts at every sudden sound, may well be appalled by the horror of heart wreck or sudden death. For attacks of palpitation, like those of tachycardia, often pounce upon the sufferer in a moment—even in a quiet moment, and without apparent cause. It is no unusual thing for an attack to set in during sleep, perhaps with nightmare. Such sufferers are often unable to lie on the left side. Either thus or more gradually the heart begins to throb tumultuously; and it may become irregular, intermittent, variable in force, volume and rate, though always frequent, until the squall blows over, or is dispelled by a reflex stimulant, such as smelling-salts, a cordial, or a conflicting impression, which may recall the control of the vagus, or cut a vicious circle at some other point. If the ventricle be contracting on insufficient

blood, the glass of cold water or draught of air may refill the arteries by splanchnic contraction ; or on the contrary a tense and so more excitable ventricle, always near "full cock," may go off in a volley of premature explosions. The attack may subside gradually, or it may cease suddenly with a shock, as if rending the patient before quitting her body. Such a finish is usually seen likewise in tachycardia. During palpitation the patient instinctively presses her hand upon the region of the heart ; the kindly support seems to soothe the tumult. Under the hand the heart's beating, like the arterial pulse, is vibrating, diffused, turbulent, and disorderly ; now striving and violent, now tremulous and faint. The impulse is not heaving but thumping or rapping, usually out of all proportion to the work registered at the radial pulse. The attack is followed by a calm, as it were of exhaustion. Beat for beat, the cardiac contraction is of course always maximal ; that is, it is not proportional to the degree of stimulus ; the disproportion probably lies in the cardio-vascular tones. In chlorosis and like conditions retraction of lung may throw up the heart, falsely suggesting dilatation. The history and circumstances of such seizures are generally enough to serve us for interpretation ; indeed, such vascular storms are unusual in organic cardiac disease (cf. "Vaso-vagal" attacks, p. 515). Still, during these discordant and confused dynamics the static conditions of the heart are not often to be appraised ; and, in the case of a new patient at any rate, we shall postpone a final diagnosis till the vessel is in still waters. Speaking generally, embarrassments due to toxic causes, such as infections, tea, tobacco, or to dyspepsia, lactation, leucorrhoea, etc., clear up, if the cause be removed, before "the neurotic attacks" ; and more completely. But both kinds of causes may coexist ; some nervous persons are intolerant of alkaloids, and indeed of all drugs.

Murmurs at apex or base are often present in the palpitation of functional disease. They are always systolic, and not infrequently at the apex. The basic murmur varying with the respiration, not carried into the arteries of the neck, and probably due to pressure relations of the pulmonary artery and the chest-wall, ought to be well known ; but too often it is misinterpreted as evidence of disease. The causation of other transient murmurs, especially of those about the apex, is unknown ; some may be "anaemic" ; some may depend upon a temporary atony about one of the orifices ; some may be due to inordinate action of the papillary muscles ; some again may be "pulmonary." Until the patient is tranquil, and the physician at liberty to map out the heart and to listen to its sounds without embarrassment, no final opinion should be attempted. In a functional case the murmur may then have ceased, and dilatation, if any, may be reduced ; and resonance of the second sound at the apex and the sharp knocking quality of the accelerated systole of an ill-filled heart and pulse may mark the case as "neurotic." Heart hurry with empty pulse (short output) is a less usual condition (except in late stages), and is not difficult to discriminate ; for example, by comparing the pulses of the standing and recumbent positions.

Of the apical murmurs, Sansom says that a systolic murmur, arising

independently of structural disease, seldom attains its maximum audibility at the exact apex, but slightly to the right and left of it. A functional murmur is variable, usually soft, and does not supplant the sound. It does not follow the usual lines of conduction; it may be diffused, yet inaudible behind. Again, he says, it does not occupy the whole, but the middle of the systole ("it is meso-systolic"). Although not of pulmonary origin, it is much influenced by respiration; it is intensified both during expiration and inspiration (especially the latter), and it may almost vanish at the end of an expiration. These points of diagnosis by murmur are more fully discussed in the several articles on Heart Disease, but we cannot be too careful in these discriminations. In one of Dr. Michell's cases (alluded to in the article on Over-stress) a variable soft, "come and go" murmur regarded as "functional" proved two or three years later to be "organic."

In the "irritable heart" of young people (p. 519) "functional murmurs" are, as Broadbent has said, not uncommon. (For other papers see Foxwell, Rudolf.)

The immediate *prognosis* in palpitation can rarely need much discussion. If generally it must be tentative, yet a serious *diagnosis* in such cases must not be suggested; if a woman is told that she has got a "weak heart," that confidence in herself which is essential to her cure is shattered. Even if in long weariness or sorrow the heart dilates a little, with some arrhythmia and an apex murmur, on amelioration of the general health and circumstances it may recover itself completely. The palpitation of chlorosis may be the result of the combination of poverty of the blood in oxygen-value with persistent or increased mass of it, which increases the demand upon the heart in respect of output (*vide* art. "Chlorosis," Vol. V. p. 705). By anaemia Dr. Mackenzie says the a.-c. interval may be nearly doubled, and yet return to the normal. The *treatment* during the attack consists in recumbency, warmth to the legs and feet, and such stimulants as ether, ammonia, valerian, smelling-salts, and hot or cold applications to the cardiac region; but alcohol, which relaxes the vasomotor system, must be forbidden. Belladonna is useful in the case of vagus irritation. Digitalis, if an occasional aid, is not to be used indiscriminately; for instance, in the atonic dilatation mentioned above it might, by restoring the mean diameter of the ventricles and the arterioles, increase contraction-efficiency, and so be appropriate; but in retarded conduction (a.-c. interval) it would seem inappropriate. In acute attacks these measures will suffice; but in some cases the palpitation does not take the form of occasional seizure, but, though less violent, is chronically recurrent, or almost persistent. As palpitation, if consisting partly in defect of central, and possibly cortical, control, is usually determined by eccentric causes, rules for general management are of chief importance. Of such are regulation of the bowels and other secretions, attention to piles or uterine disorders, mitigation of worry, moderation of work; temperance in food and avoidance of tea, coffee, tobacco, and alcohol; regulated exercise, hydrotherapeutical methods, sufficient sleep. Occasionally

such drugs as aconite, the bromide of sodium, ammonia, or camphor are often very helpful; or an ice-bag over the heart may be needed. Aconite, cautious as its use must be, in some of my cases has stilled such palpitations notably.   Convallaria and cactus also have their advocates.   The expulsion of a worm has sometimes proved to be the cure of a troublesome palpitation.   In a case I saw recently, a lady who had suffered long and severely from such attacks, had also vertiginous and syncopic seizures, and a persistently tumultuous pulse.   She had been under prolonged and very expensive methods of "specialist" treatment, but, by cutting off her tea-bibbing, she was cured in a week or two. Satisfied that the heart was fundamentally sound, and feeling sure that some such toxic cause was at work, by the method of exclusion we happily soon lighted upon it.   Palpitation coming on for the first time in later life is a matter for anxiety; still it may be gouty, or due to the influences, toxic or dyspeptic, of bad teeth.   In respect of uterine disorders, I need scarcely add that vascular instability is eminent during the female climacteric, and at the change of life may arouse some fears of static disease of the heart, or of an insidious rise of arterial pressures.

False Palpitation.—In my Goulstonian Lectures of 1884 I stated that it is not uncommon for patients, especially for highly nervous patients, to complain of palpitation, although on examination little or nothing of it is perceptible to the stethoscope, or at most the heart may be accelerated by some five or ten beats; yet to judge by the bearing of the patient the distress is acute.   Such patients will probably complain of other hyperaesthesias, and of pains in other regions, such as the head and back; and it would seem as if there were a hyperaesthesia somewhere about the heart itself or its attachments, so that its function surges into consciousness.   It is perhaps not fanciful to compare this with the hyperaesthesia of the stomach in such or similar patients. There would seem to be a like hyperaesthesia in the vessels also, as rushings in the arteries, whizzings in the head, and other "determinations of blood" are complained of; sensations apparently due to slackening of the arterial walls.   There is generally definite tenderness of the skin, and flinching about the apex of the heart.   Medical students' and other "psychical hearts" are often of this kind.   In the cardiac region the patient complains of tightness and oppression, of urgent heaving, or bursting of the heart, or of cramp in the part, with panic fears.   Or the pains may be boring or cutting : the husband of such a sufferer, in writing to me, tore from Bradshaw the advertisement of a corset-maker, and drawing a dagger with its point entering the left submammary region, enclosed the picture as a graphic representation of his wife's agonies. Many of these patients suffer from air-hunger, and from anomalous pains . in the chest and arms, when the case may merge into the fantastic class of "pseudangina pectoris."   The attacks may recur many times a day, and, in the broad sense, are not difficult to appraise as neurotic : the story of the case rarely leaves much doubt of this interpretation.   The blood-pressure often rises during the attacks, the vasomotor vexation being on

the constrictive side, but rapidly falls as it passes off. The vasomotor phenomena are usually secondary to the intercostal neuralgia, or other inhibiting distress. When we remember that in the bulb, the cardio-inhibitory, the vasomotor, the respiratory, and the gastric centres abut upon each other, we shall feel no surprise that the functions related to all these centres are often reciprocal, or are influenced together (*vide* p. 515). As auscultation and other means of investigation do not reveal any change, or next to none, the intimate mode of these phenomena is not easy to ascertain. In ordinary palpitation, as the pulse rises, perhaps to 120, the pressure falls and the face flushes; or the patient turns pale and the pressure rises; but neither of these events is seen in the false palpitation. Until a better hypothesis is suggested, we may suppose that there is some morbid susceptibility to the impact of the ordinary stroke of the heart.

Weak heart is used in two senses; statically of a heart failing in fibre, dynamically of a heart subject to a transient laxity. Of the former kind we have not here to speak, or only to premise that one heart is not as good as another. It is a common experience that, for one reason or another, the range of cardiac capacity differs considerably in normal persons. The second kind may appear as follows:—The patient, usually but not always a neurotic woman, tells us the heart ceases to beat; in the severer cases the patient is so convinced of this as to fear that an attack may be fatal. Sometimes as the attack comes on the face turns grey and the lips blench; in other cases the face may flush, or illness is betrayed rather by an expression of apprehension and distress than by vascular signs. Air-hunger may be intense, but cyanosis, I think, never occurs in mere functional disorder. The hand is pressed to the region of the heart, where in many cases a very acute and agitating pain has arisen; in other cases it is not so much a pain or throb as a sinking, a sinking not at the heart only but a general "lipothymia." She may also complain of pins and needles, of yawnings, shivers or chills, and present other evidences of irregular blood-distribution. After a time of passionate agitation, and almost violent restlessness, the distress passes off, and the patient recovers with that sense of utter exhaustion which is notable in many merely functional affections of the heart. Such seizures, with plentiful lack of clinical sagacity, have been separated into a sub-class of angina pectoris. The pulse during the attack is not con-stant in its character; it may seem weaker by shrinking in volume; sometimes it slows a little, usually it increases in rate—say 100 to 110, yet it is not the pulse of syncope; sometimes, however, the patient feels swimming or misty, things fade into the distance and the pupils dilate; then she will gasp, and return to life with a sigh or two of relief. Speaking generally, there is no peril in the attacks except that which lies in the habit of taking drams to cure or prevent them. Mrs. Gamp's prescription, for the "spasms," of two drops of brandy on a lump of sugar is too well known to these patients and their friends; the medicine is at hand and is assiduously administered, with the rubbings of the extremities,

hot bottles, and the like, which are grateful to these patients. And for the moment the alcohol is helpful; probably by a reflex influence it pulls the heart together, or imparts something or other which may be Dutch courage, or more mechanical relief; moreover, it may counteract vaso-constriction by dilating the arteries of the surface. For these seizures are vasomotor storms, set up by some incidental cause, very often by severe intercostal neuralgia. In a frail and very "nervous" man in whom well-marked attacks of the kind were set up by paroxysms of intercostal neuralgia, the stomach was widely distended. This I hoped might be the cause of the attacks, but it proved to be one of their minor consequences. By exile to a dry, warm, equable climate the neuralgia and this troop of consequences were cured repeatedly, to return again in cold damp weather at home. The case had been confused with angina pectoris, a disease to which it bore no intimate resemblance.

There is another not uncommon kind of functional cardiac instability, as follows :—A gentleman, aged thirty-three, apparently healthy in all other respects, but of nervous temperament, complained of breathlessness on ascents. There was no anginal or other pain. At nights he would awake with a sense of faintness or impending death. His pulse, while standing, was 130, on sitting down a little less; but as he lay down flat the radial pulse instantly oscillated widely for two or three seconds, and then fell to a steady rate of 80. He had some reason to suspect that his nocturnal discomforts were due to an abnormal fall in the rate of the circulation. His aspect was healthy; no cyanosis. He did not smoke, nor drink much tea or coffee. The heart on examination proved to be free from any abnormal sign, unless it were that the apex-beat was obscure, and the impulse rather diffused. He had had attacks of the kind before, if not quite so severe ; and had always been cured by going to sea. An abdominal pad, or a weight on the abdomen, to compress the splanchnic veins has been often suggested in such cases ; but this mechanical device is disappointing, even when it is not annoying. In one lady who suffered much from this heart-sinking, to raise the arms was almost a certain means of producing an attack or a threatening of it : she assured me that she dared not raise her arm to knock at a door.

Cardiac Asthenia.—This ailment is similar to those Da Costa described as "irritable" or "weak heart." He says that in these sufferers the action of the heart is feeble for long periods ; a feebleness to be distinguished from the weakness due to organic causes, and again from the more transient upsets of lithaemia, gout, tobacco, and the like. The affection generally manifests itself in persons whose nervous system has been strained by worry or overwork; though the full brunt of the disease may be sudden. The patient is prostrate in bed; all attempts to sit up cause swooning and vanishing pulse. The heart's action is enfeebled; the pulse is not only small, but soft; it is generally increased in frequency. Although without pain, there is a sense of uneasiness in the cardiac region. The bodily temperature, as well as the warmth of the extremities, is lowered. The breathing

is undisturbed. "I am out of heart rather than out of breath," was the reply of one of Da Costa's patients. Insomnia is not infrequent. The patient rallies but slowly; two months in bed may be his portion, and months more of ailment before he recovers; for the issue is as tedious as the onset may be brusque. In some few cases the rhythm of the heart is irregular. The disorder may occur in either sex, and at any time of life between childhood and old age. There is no increase of percussion dulness; the impulse is feeble; the first sound is short, lacking in volume; the second sound not accentuated. The flusters of some cases described in a previous paragraph are conspicuously absent; in "irritable heart" the patient can still get about, and the heart's action is more obviously and noisily disordered (*vide* p. 519).

As regards the *pathology* of these anomalous cases, hazily apprehended as "neurotic," "hysterical," and so forth, Sir William Gowers, in his Lectures on Vagal and Vaso-vagal attacks, in gathering them together, and in interpreting their affinities, has done good service. Their features, as Sir William shews, occur in series definite enough to be formulated; for, as concerns the heart and vascular system, the symptoms are mainly referable to disturbance of some of the functions of the pneumogastric and the allied vasomotor nervous system, whether by some toxin, pain, or visceral irritation. Similar attacks have been classed by Pierre Bonnier as "syndromes medullaires." The vagal and vasomotor symptoms vary in relative proportion, so as to pass under various disguises; the pulse may be much retarded. As they are more frequent in women, and start from centres which are readily influenced by emotion, they have been treated as merely whimsical or gusty. These fairly consistent series, however, often occur in equable, sensible persons, and need not be in origin psychical. The sensations referred to the stomach, the respiratory system and the heart, and the sense of indescribable oppression beginning in the epigastrium and often ascending to the chest, with a respiratory distress sometimes so intense as to amount to orthopnoea, are attributed to the vagus. With them direct cardiac symptoms may be combined; discomfort, or in some cases acute pain, and the sensation of stoppage of the heart followed by rapid action. In some rarer cases, with the dyspnoea, or the cardiac sensation, or both, may come the sense of impending death, causing alarm or even intense dread; this, as I have repeatedly stated in my studies on angina pectoris, is of vagus origin. The vasomotor spasm, in some cases, shews itself in symmetrical dead fingers, small pulse, and other vascular fluctuations. For the mental perturbations and apprehensions which often accompany these attacks, Sir W. Gowers suggests the name of "Psycho-vagal" symptoms. Among other remedies this author finds advantage in the persistent use of nitroglycerin, which "seems to exert a permanent steadying effect on the vasomotor centre." Women about the menopause are liable to disturbances of this nature, either severe, or on the other hand so slight as to pass without notice, if the observer does not note their signification. In a lady sent

to me a few years ago, a very notable phenomenon appeared. I saw her a day or two after a severe attack, when I found the left lobe of the thyroid distinctly enlarged. The pulse at this tranquil time was only 70, and quite normal. Without any very leading questions she told me her neck always enlarged about the time of the attacks, and diminished to normal in the following few days. When I saw her it was gradually diminishing.

The *treatment* is to steady the vascular oscillations by the wet sheet, the shower, the douche, massage, regular exercise, and the like. Unfortunately in this country we have not had the advantage of scientific hydrotherapy, such as that of the school of Winternitz and Determann; and English people do not readily fall in with German food and customs. Of the value of electrical methods, blended as they are with various suggestions, no opinion can yet be given. The data are insufficient. At first rest in bed may be needed. Then graduated baths, Swedish exercises, gentle riding on horseback can be arranged by degrees. Nutritious feeding is of course essential. Aerated waters should be avoided. In Da Costa's opinion for the cases of his mode a generous allowance of alcoholic stimulants is necessary; in my experience temperance in alcohol, even to the point of total abstinence, is paramount in all the functional cases, for the sake of both body and mind. Some cordial these patients will have, perhaps ought to have; they are frightened out of their wits, and a stimulant seems their only help. But warmth, warm or cold applications, ether, valerian, ammonia, camphor, or peppermint will serve for the moment; and as the immediate anxiety passes away, attention to the general therapeutic needs of the case will, in a broader and more wholesome sense, remove the need for such artifices. Of drugs Da Costa regards strychnine as pre-eminent in "cardiac asthenia." The dose need not exceed $\frac{1}{30}$ gr., but it must be given continuously. Arsenic is the next best; iron is not usually indicated, and the need of digitalis, if any, is but occasional. Nitrites have not been so useful in my experience as Sir W. Gowers has found them to be, and they are often ill tolerated. Bromides, valerian, or even opium may be required in special circumstances. It is impossible here to treat of the eccentric causes always to be sought for, not infrequently to be detected; such as latent haemorrhages, menstrual or other uterine disorders, and so forth. The personal ascendency of a confident, gentle, patient, cheerful physician is everything to these sufferers. While always resourceful in assuaging their various instant distresses, he must never fail to be consistent in his opinions and methods, and to keep their attention fixed upon convalescence. His methods must be seconded by a nurse with similar qualities. Patients of the kind we are discussing are often inconstant and freakish; not a few are compounded of unstable mind and frail body; yet, feeling they need a firm rule, are inwardly grateful for it.

Syncope.—Whether in syncope the heart stops for a brief interval altogether, or only falters, is unknown; it may beat with a beat so feeble

as not only to escape the finger, but to be inaudible at the heart. "No one dies of a faint," one may say; or another may say with equal truth that sooner or later almost every one does. Yet the syncope which cuts the vital thread is evidently something so different in degree and signification from the ordinary faint of the ladies who are carried out into the vestry, that we may now fix our attention exclusively upon the functional disorder. For the church faint is primarily not a cardiac failure, but an expansion of cutaneous and splanchnic vessels with a sharp fall of arterial pressure. Dr. Dukes says accordingly that school-boys who faint in chapel have albuminuria.

Yet of this curious disorder no full explanation is forthcoming. It used to be a very common malady; and even now most women have had a little experience of its premonitory symptoms. But to faint is not woman's exclusive privilege; every physician has seen strong men drop like oxen—for instance, in the gallery of an operating theatre. A sturdy man once fell suddenly to the floor in my consulting-room, where a moment before he had been complaining to me of some temporary disorder, partly dyspepsia partly fag. I have known him for some quarter of a century since that day, and, so far as I am aware, he has never fainted since. On another occasion, to our dismay, a young physician of robust habit of body, but tired with work by day and night, rolled to the floor at the bedside of a sick lady where we had met in consultation. Again, an old friend of mine, then a young man of some five-and-thirty years, and then and since hardy and sound, on springing suddenly from bed in the night to empty his bladder, fell backwards, drenching himself with the contents of the chamber-pot. His wife told me that he lay "unconscious for a minute or two." The anxiety in such a case is whether the attack were syncopic or epileptic: the circumstances of this attack, chiefly the rapid recovery on the prostration, pointed rather to syncope, an opinion which time has ratified. For a fuller discussion of the differentiation between syncope and epilepsy the reader is referred to the papers of Sir W. Gowers. On the other hand, syncope is not usually an isolated event in the life of the patient. People who faint are, as a rule, "given to fainting"; such persons dread hot rooms and congregations where the distribution of the arterial blood may oscillate widely. Or, again, they dread certain strong sense-impressions—such as the sight of blood or strong odours; Italian women are said to be peculiarly liable to faint on the smell of flowers. On one occasion when dining with a charming hostess who had decked her table with exotic roses, one of her guests, apologising for his weakness, said that he should faint if he sat with his back to the fire, and at some sacrifice of harmony he was conveyed to another seat; no sooner had he been dealt with than another guest thought he had better add that he was subject to faint in the midst of a strong scent of flowers. There was nothing for it but to clear the table of the spoils of the Riviera; after which twofold commotion things fell a little flat. In such hypersensitive persons, of either sex, the pulse varies too widely on quickly rising, sitting, or lying down, or even

on elevation of the arm, often more than twenty beats. In fagged, prostrated, or neurasthenic persons this enervation of the compensatory mechanism is often remarkable. The pulse of the weak but otherwise normal, may rise to 120 on assuming the erect position. Now, as in the patient described on p. 514, let this person lie quickly down flat; the pulse will run on for a few seconds as before, and then—usually with a few irresolute waves—drop suddenly to the normal rate. This means rest and such restorative method as a sea-voyage. Or the condition may be more perilous. During the summer of 1907 I saw with Mr. Wingate, an undergraduate of Magdalene who had bicycled from Ely to Cambridge under a burning sun. As he had only a cap on his head he felt the heat at the back of his head intensely. Besides he had been at this time a little tired with rowing. During the evening he felt very fretful, then being faint went to bed. Next morning, on every attempt to rise, his head swam, and Mr. Wingate found that on raising no more than his shoulders from the pillow, the radial pulse vanished. I saw him an hour or two later, and ventured, against Mr. Wingate's advice, to raise him very gently. He had hardly been lifted a foot from the pillow when he blanched and the radial pulse snuffed out. I dropped him instantly, and the pulse flickered back. After a languid week or two he was able to go home, where he made a slow but good recovery. Here also presumably the vasomotor centre was in abeyance.

Even without organic disease syncope may be fatal; such cases are not extremely rare; they are common enough to give caution to the prognosis and vigilance to the treatment. The faints due to agonising pain are more likely to be fatal than those arising from sudden displacements of blood-pressure. The inhibitory effect of intense pain may through the vagus, as in gall-stone or in angina pectoris, arrest even a healthy heart, and to a diseased heart it is very perilous.

The premonitory symptoms of fainting are known to every one. He is a fortunate man who, under fatigue, or in the weakness of some malady, such as influenza or the like, has not been aware of the swimmings and exhaustions which may usher in a full attack. If many of us have never fainted, we have all of us felt faint. When the attack is fully established unconsciousness is complete; the respiration is to be detected only by the use of a feather or a mirror, or not even thus; and the radial pulse, or even the heart's contractions, may be imperceptible. But if urine or faeces are voided, it may be said with some certainty that the attack was something worse than a faint.

Whatever the remoter causes, such as general anaemia, vasomotor lability, fatigue, and the rest, the immediate link in the faint is encephalic anaemia. The same is true, of course, in organic diseases, such as those of the heart. It is the first duty of the physician, as it is the care of nature herself, to place the patient in a position to favour the return of blood to the brain; the head must be dropped even lower than the trunk of the body. As Junot's boot will produce syncope, so, conversely, to elevate the legs will aid in its dissipation. The blood-pressure

must also be raised by causing contraction of the superficial blood-vessels ; cool air, and the admission of it to the skin by unfastening the dress, is one means of attaining this end ; and any bands which may be hampering the " respiratory pump " should be loosened. The respiration may be called upon by reflex stimulants also, such as smelling-salts, dashes of cold water, and so forth. In cases of anaemia compression of the abdominal veins may be useful ; or the application of an Esmarch's bandage to one leg or both legs ; in extreme cases artificial respiration, or even transfusion of saline or blood, might be needed ; but in the functional cases which are now under discussion, such difficult means are rarely, if ever, necessary. It is desirable, perhaps, to add that after the restoration of consciousness the physician should not leave the patient without a strict caution against sitting up or standing until all tendency to a recurrence is averted. For a fuller discussion of events of this kind the reader is referred to other articles in this work.

Irritable Heart.—Since the publication of Da Costa's and Myers' well-known papers this derangement has been too exclusively attributed to muscular over-exertion. It seems, however, that we must divide the subject of irritable heart into two classes : the irritable heart of young persons now to be described, a curable disease ; and the " Soldier's Heart," already described under " Over-stress of the Heart," p. 235, which is a very incurable one. The irritable heart of adolescents is a product of many conditions ; the irritable heart of maturer adults—the irregular fretful heart which sometimes goes on to dilatation and static disease—is more definitely the result of over-exertion. In the irritable heart of adolescents the upstroke in the sphygmogram is brisk and high, with large dicrotic wave ; in that of dilating heart it is low and less brisk, and the rhythm is often irregular. The irritable heart of the former kind is much as follows :—A young man, more often than a woman, says that he is bothered by his heart ; he has pains about it, always tiresome, often sharp ; the organ throbs and jumps ; it never lies outside his consciousness. If he exert himself it beats violently ; if he lie still in bed it also makes itself a nuisance, echoing in his ears when he ought to be asleep. When he is stripped he is generally a lanky, long-chested fellow with wide intercostal spaces ; and the apex of the heart kicks rather than heaves in the interspace. The heart may seem a little out of place, displaced somewhat outwards and downwards ; but in these cases of flat chest and ill-developed lungs the dimensions of the heart cannot easily be measured. It may be rather dilated, possibly even a little hypertrophied ; but probably no more than too palpable and visible. How the strong, even vehement beat yet effects so little blood-delivery is an old marvel, which has recently been discussed again by Krehl, Albrecht, E. Hering, and others. Their conjecture is that the action is excessive only in the apical part of the muscle (*vide* Hering). I may say in passing that in such subjects tuberculosis rather than heart-disease may be the fear, especially if the pulse-rate be accelerated. A few years later such a man consolidates, his lungs expand, the heart

relatively recedes.   Now in many of these men there is no doubt a story of
considerable, or indeed of excessive exertion ; but often there is not, and
in these cases warnings to refrain from active exercise may be too rigidly
enjoined.   That in the intervals of rest the mean arterial blood-pressure
is nearly always moderate is against persistent hypertrophy : when
pressure falls, the heart cannot long remain above its strength.   Some
dilatation there may be — a dilatation not of high intraventricular
pressures but of atony.   The peripheral arteries are lax, the rhythm is
often a little or sometimes very uneven, and the second sound at the
apex too loud.   The first sound is not muffled, as in unquestionable
hypertrophy ; it is a shorter rap.   Sometimes there is a murmur, more
often there is an "impurity" of the first sound, as if dimmed by some
distant murmur overheard.   This murmur is often "pulmonary" in
origin, as a halt in the respiration will prove.   In very fussy hearts
no opinion about murmur may be possible without delay.

To account fully for this state of things in the circulation of such
patients is to know all the ins and outs of the ways of youth, a know-
ledge which comes to us best by reflection on our own young days.
The manly absorption of grown-up and more than grown-up doses of
tobacco ; the reckless meals and drinks ; the hot arguments on the
framework of the universe and the destiny of man, protracted till two
o'clock in the morning ; the spasmodic bouts of study ; the examina-
tion bogey ; the conflict with untamed and rebellious passions, some
generous, some unwholesome ; the violent games and the bear-fights ;
the ardent hopes and the bitter griefs — what elder is there who remem-
bers all these things, and does not long to dash pell-mell into it again,
and accept irritable heart into the bargain ?   It is a narrow step between
the wise young man and the prig ; still it is no priggish advice which
would cut out of this gay, ardent and careless life some of its idler and
less lovely follies, and complete the cure of irritable heart by better-
regulated exercises ; not violent stress one day, and idleness not unmixed
with dissipation on the next, but an education of mind and body, to
promote a uniform development not only of lungs and heart but also of
the whole man.   Muscular exertion, then, is in most cases the cause
of this kind of irritable heart only when pursued in an irrational and
unsystematic manner by a more or less nervous and dyspeptic young
person whose heart, lungs, and general development are not yet big enough
for prolonged efforts, and whose ethical and intellectual life likewise is as
ardent as it is immature.   Irritable heart due directly to strain is
described in its proper article ; but many of the cases are difficult to
apportion between the two classes.   The irritable heart of coal-miners,
described by Snell and Dr. G. A. Gibson, was very familiar to me in the
West Riding.   It is attributed by these authors to the continuous
labour with the pick in a cramped posture on the side.

The treatment of the common forms of irritable heart becomes
apparent from its causes.   Muscular exercise must be systematic ;
perhaps prohibited for a while, or restricted.   The exercises should be

designed to open the chest and strengthen the back. Half an hour's rest on a couch before luncheon and dinner, and at least as much after these meals, may be enjoined. The food must be well masticated, and the fluid at meals reduced in quantity. Sleep with open windows, and in the morning the cold sheet, douche, or sponge bath are also very helpful means. It is better to avoid specific drugs, unless the symptoms be unusually vexatious, when small doses of digitalis with a little bromide may be economically used. For more continuous use the tincture of *Prunus virginiana,* or cactus, is helpful. Above all things there must be no "atmosphere" of invalidism. Many of these folk find their way to Nauheim, and fill up the lists of its successes. They do still better under judicious management in this country. These patients are often a little shy and sombre in spirit; change of scene, pleasant society of both sexes, and frank and kindly advice on sexual matters, are a part of the services which a sympathetic physician may render to young men ; and while we may have a kindly smile for their heroics, we must remember that they are often acutely miserable. Some excellent remarks on the management of such subjects are to be found in Sir William Broadbent's works.

In **obesity** the function of the heart needs very careful appreciation. Even among the laity acute disease is well known to be perilous to fat persons. The fibre of the heart may be in decay, or the action of the organ impeded by the accumulation or the insinuation of adipose tissue in excess ; but there are antecedent factors. Approximately speaking, the cardiac and skeletal muscles are duly proportioned to each other ; and, as obese persons are often deficient in muscular development, the heart too may be deficient in proportion to the bulk of the body. Again, the fat are usually a thirsty folk, and excessive imbibition, whether of strong waters or of weak, is apt to lead to excessive volume of blood, and to dilatation of a muscular chamber of which the muscle may be already somewhat inadequate. Here our partition between functional and organic heart-diseases may become very thin. Except in cases of hyperpiesis, which belong to a different category, the arterial pressure is prone to fall, and the venous to rise ; in many stout people the ankles are liable to oedema. The nice and responsible task of reducing obesity is considered elsewhere (Vol. IV. Part I. p. 501).

The Neurotic Element in Organic Disease of the Heart.—We are too ready to suppose that death from organic disease of the heart is a direct result of demolition ; that the crippled organ stumbles along until it can do no more, and then sinks to its rest by sheer mechanical inability. This may sometimes be the case ; in many instances, however, this is not the course of events, not even within the confines of the organ itself. Much functional disarray may be lifted off cases of organic cardiac disease. The harmony between the different segments, or the several qualities, of the heart may, and frequently does, become deranged; and it is not surprising that irregularity should result. For instance, the arrhythmia of mitral disease in Dr. J. Mackenzie's opinion is not a mere result

of inefficiency, but may be a cause of it, and due to the transference of excitation from one tract to another—say from the sinus venosus to the auriculo-ventricular bundle. The synthetic function of the several parts of the heart and its allied vessels is but too easily broken in upon or perverted at one point or another. Again, the stress of organic disease elsewhere, or of derangements of the stomach or bowels, torpor of the liver, pulmonary spasm, cerebral or bulbar interference, the absorption of toxic products such as choline, and so forth, are potent to depress or to disturb the heart's action beyond its mere mechanical disadvantage. Thus it is that in most cases of heart-disease there are many phases : in one month the patient is pretty well, in another he is at death's door ; yet again he comes round, and this not necessarily as a result of treatment, or if of treatment, of such a remedy as an injection of morphine, which may re-adjust, or permit the re-adjustment of, internal cardiac stimuli ; or may block some reflex arc. Again, even in a few minutes, the vomit of a little sour mucus, or the discharge of an offensive stool, may set matters straight. On the discrimination between a toxic and an asthenic state of the heart I may refer to a paper by Dr. J. D. Rolleston. It was with this conception in my mind that in 1869 I recommended the subcutaneous injection of morphine in heart-disease ; not only, in appropriate cases, to cut the vicious circle of a paroxysm of dyspnoea or restlessness, but to prevent the heart from being "tripped up by the intrusion of a neurosis," as the late Dr. Solomon Smith used to put it. In a fatal seizure of angina pectoris, death is directly due to such an inhibition. The importance of these considerations in respect of treatment is obvious. It has been well said that our choice of remedies lies no longer only among cardiac stimulants or depressants, constrictors or dilators ; a whole range of remedies is opened to us which, although without direct action on the heart, relieve heart trouble by blocking the departures of nerve derangements, or by smoothing away eccentric irritations. I may add that not only are new remedial means thus opened out, but in these words we have the explanation of the value of many remedies which, in a more or less empirical fashion, have long been familiar to us. On the peril of the converse error of being blind to an organic heart-disease covered by a cloud of nervous disorders I scarcely need insist ; yet the dilemma is often very puzzling. During the last few years I have had a growing conviction that some few cases of cardiac disorder, especially in women, regarded as "functional," are cases of latent mitral stenosis. In some instances I have thought this diagnosis morally certain. Although no murmur can be evoked, a sharp first sound, perhaps an occasional reduplication of the second sound, a habitual short cough, and above all a consistent, as contrasted with a capricious, embarrassment in certain efforts, may point to this organic defect. A slight thrill at the apex, though not pathognomonic, would fortify suspicion.

PAROXYSMAL TACHYCARDIA.—The names Tachycardia and Brady-cardia are often used merely to signify frequent and infrequent rates of the heart respectively ; but such uses are very slovenly. If, whenever we talk of "tachycardia," the mind is to range over an indefinite scattering of cases, from peritonitis to Graves' disease, in which the pulse ranges, say, over 120, we shall waste a great deal of time in discussion and a great deal of space in books.   In the preparation of this article I have been obliged to discard some essays because of this ambiguity.   For
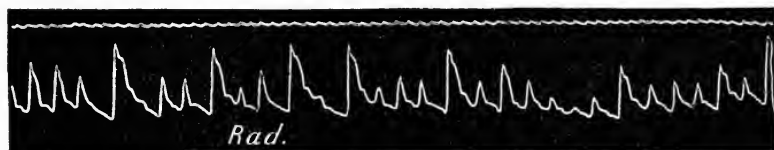
FIG. 55.—Tracing of the radial pulse, shewing the irregularity characteristic of the sudden inception of the rhythm of the heart, as in paroxysmal tachycardia, probably due to the irritation of the auriculo-ventricular node (Mackenzie).
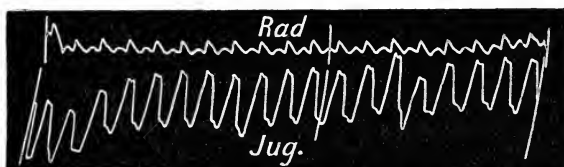
FIG. 56.—Simultaneous tracings of the radial and jugular pulses twenty-four hours after the onset of an attack of paroxysmal tachycardia. The jugular pulse is of the ventricular form. At the end of the attack, when the heart slowed down, the jugular pulse changed to the auricular form (Mackenzie). (Eichhorst thinks the auricular form may persist throughout.—C. A.)
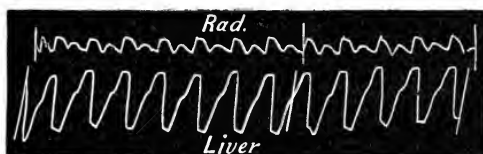
FIG. 57. — Simultaneous tracings of the radial and liver pulses during an attack of paroxysmal tachycardia towards the end of life. The liver pulse is of the ventricular form (Mackenzie).

instance, in contemporary papers I find some authors including under "tachycardia" not only heart-diseases and toxaemias, but all kinds of palpitation, tea and tobacco cases, dyspeptic gusts, and so forth.   One author will include cases of larval Graves' disease, another a case of the stormy onset of an infection ; whilst many a one, without due discrimina-tion, sweeps in a medley of cases of valvular disease in which the heart was accelerated.   Even Schmoll, in his excellent essay, apparently dis-heartened by his failure to catch a "clinical entity," confuses our ideas by thrusting back tachycardia into the wilderness of simulations and contingent or transitional forms.   By the use of so fine a word, we

convey to the student's mind, and even engender in our own, some notion, some association of ideas, some other symptoms, beyond mere rate. Indeed, if we do not, the word is an incubus. Tachycardia, then, or Paroxysmal Tachycardia, ought to be used, as apparently its inventor, Gerhardt, in 1882, intended it to be used, for a definite concept; though Bouveret seems to have been the first to attach it definitely to the paroxysmal disease. To reduce the word, then, to its prairie value is to throw our clinical labels into confusion. In this contention, urged in the first edition, Sir R. D. Powell and Dr. Samuel West (83) support me; of tachycardia, Powell says this name should be strictly limited to the paroxysmal heart-hurry; West says "it would seem to imply some peculiar clinical condition," yet is "used so indiscriminately as to have no meaning." The use of the word "tachycardia" to signify rates above 170, although not desirable, for in the disease before us the rates are often much lower than this, yet might be defended; for a transgression of this limit does signify a change in kind. However, so far as I know, no one has suggested this limitation.

To what concept then, or series, is it convenient to apply the name Tachycardia; or Paroxysmal Tachycardia, as this longer name is now customary? Not to any case of rapid heart, but to an enormous acceleration (Herzjagen) of the pulses of a heart not necessarily the seat of static disease; an acceleration which attacks the patient suddenly, which does not persist indefinitely as does, for example, the rate of Graves' disease, but for a variable period rounded off by an equally sudden reversion to the normal state, less certain phenomena of exhaustion, and which is now presumed to consist in a dislocation of rhythm-inception from the sinus venosus to the bundle of Kent and His ("nodal rhythm"). Heart-disease, in the static sense, may coincide with Tachycardia, it is true; mitral stenosis may coexist with chorea, nay, perhaps it may favour the occurrence or intensify the peculiar symptoms of chorea; but that does not deprive us of the name chorea, nor justify us in using it loosely.

Careful clinical observation and no less careful verification after death (so far as this has gone) indicate, at present, that periods of very rapid pulsation coming suddenly, departing suddenly, and attended with certain other symptoms, objective and subjective, are consistent, if not always coincident, with a heart practically sound; that coarse heart-lesion is not an invariable antecedent of this malady. Hypertrophy is not implied in rapidity of pulse, of any origin, as such; for increase of rate generally means not only diminution of output per beat, but perhaps per minute also, so that, if exhaustion and atony set in, residual blood begins to accumulate heavily; especially, as it would seem, on the right side. If output per beat and per minute is increased, a rise in rate cannot be considerable. The records of necropsies in pure tachycardia, before the stages of dilapidation, are few; but the sudden subsidence of these phases, and the complete recovery in not a few cases of the malady, without any signs of disease of the heart, support the indications

of our scanty pathological material that it does not consist in any gross lesion.

The first description of the disease we owe to Drs. Payne and Cotton. I shall describe it from reflections on characteristic cases of my own. A woman, passing through the menopause without any peculiar derangement, since her adolescence has been liable to seizures of tachycardia. She is a healthy-looking, well-nourished person, and is now getting stout. Her rather anxious and fidgety temperament may indicate a neurotic bent, or may be the consequence of her malady ; but her family history is without apparent importance. Her own life, though more than once broken into by calamity, has been, on the whole, one of prosperity ; moreover, her ailment dates from a time of life long before any trials had afflicted her. She is happily married, but has had no children. In the attacks, which have preserved the same characters throughout, she turns a little shivery and pale, at times even ashy ; a peculiar lassitude and restlessness possess her ; the extremities are cold, and these and other parts are "numb." She soon becomes aware of a tightness, tremor, and oppression rather than of a beating about the heart ; the tightness may amount to actual pain, and may dart here or there. The pulse is now, however, beating, or rather vibrating, at the rate of 160 to 200 a minute (the reckonings of the pulse have not been systematic ; and often the record is that the pulse could be counted, if at all, only by the stethoscope). The respiration is sighing and uneven rather than a dyspnoea. If the attack continues for a day or two the area of cardiac dulness extends towards mid-sternum, or even beyond it ; the veins of the neck swell up, and residual blood in the ventricles increases ; the sounds are tic-tac and rather tinkling, but no added sound or arrhythmia is to be detected. As it proceeds she becomes very fretful and wretched ; but the oppression and tightness pass off. The urine in some cases is scanty ; but in her, and in another of my patients, polyuria attends the attacks throughout. In severe attacks she is more or less aphasic, with the aphasia of exhaustion—a phenomenon not uncommon in migraine, and in persons spent by fatigue. Such an interval of aphasia was described by Tyndall in his own person after a dangerous and exhausting scramble among the rocks above the Grimsel. Moreover, as we may find in some other cases of nervous exhaustion, she may have a particular sense of weakness or heaviness in the left arm— "functional hemiplegia."

The duration of attacks varies within wide limits. In some patients an attack ceases after a few hours or even a few minutes ; in one of my cases it lasts for about fifteen minutes only ; in others, for three, four, five days or more. It is said to have lasted in some cases as long as ten or eleven days ; or indeed for weeks at a time, but on the consideration of such records suspicions of a wrong diagnosis present themselves. In one of Bouveret's cases, however, genuine attacks endured for as much as thirteen days. In one of my patients the attacks would often recur in groups, giving thus an impression of a paroxysm longer

than was strictly the case. He would have such a series of four or five
attacks, and then, perhaps, none for a year or more.   During the nights
of an attack the patient may be almost sleepless, but during sleep the
tachycardia pursues the same course.   Sometimes during these nights the
female patient first mentioned is a little delirious.   The rhythm of the
heart is, generally speaking, regular, until the ventricle becomes dilated,
when every pulse may not reach the wrist ; and herein tachycardia may
be contrasted with similar accelerations in ordinary heart-diseases, when
the pulse is usually irregular.   In one case of paroxysmal tachycardia,
however, which I saw with Dr. Lloyd Jones, the pulse was irregular ;
although the heart was not failing.   At these high rates residual blood
must accumulate, and every beat may not reach the wrist.   The only
means of an approximate count is at the heart, and during fractions of the
seconds circle.   The form cannot be delineated ; no sphygmograph can
follow it.   The arterial pressure keeps up wonderfully until failure
sets in.   The rate will not infrequently seem to range somewhere
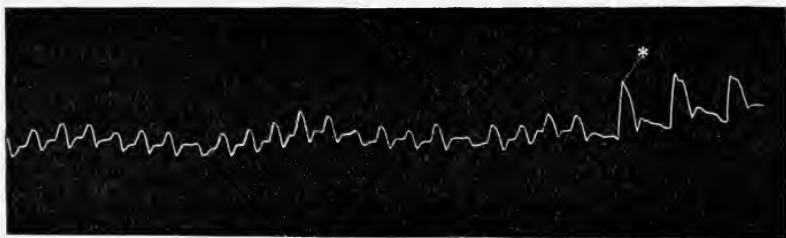
FIG. 58.—Paroxysmal tachycardia in a man, aged 36 (Dudgeon's sphygmograph).   Tracing at
conclusion of attack.   (Eichhorst.   By kind permission.)

between 200 and 250 ; yet the blood-pressures, although influenced by
the psychical state, are not much below normal (p. 530).   In a case which
I should hesitate to quote were it recorded on any authority less than
that of Bristowe, the pulse number is said to have been counted up to
308 a minute.   Dr. Mackenzie drily remarks that no tracing of a rate
above 200 has been published.   In many instances, of course, the rate is
not very extravagant ; sometimes the range may be only from 120 or
even sometimes 110 to 180.   The rate may differ widely in the several
attacks, even in the same patient ; though usually in each case a certain
consistency is preserved.   In a simple case, between the attacks, the heart
is free from any sign of defect.

The cessation of the attack is always brusque, generally sudden ; it
may end with a brief arrhythmia, with a few slow hard beats, or with
one violent rebound, followed, as more than one of my patients has said,
by " a sort of swim " ; others speak of a breeze of air wafted through the
system.   Three big beats form a common conclusion (Fig. 58), and when in
a certain patient of mine an attack would seem to be winding up with two
bounces only, she was dissatisfied ; for she said a double bounce was no
real arrest, but was always followed by a renewal of the run ; whereas

after a treble bounce she was safe, till next time. The urine in my own cases never contained sugar, albumin, nor any substantial excess of urates or phosphates. Attacks cannot be traced as a rule to any incidental cause nor to any season ; they may come on at a moment of rest ; often indeed they begin, or end, during sleep. One of my patients was an epileptic tailor, with a good family history. In him over-exertion might bring on an attack ; but he could always arrest an attack thus produced by holding his breath in inspiration and then stooping tightly down with his belly between his legs. The spontaneous attacks could not be thus cut short, though once (on the third attempt) he stopped one in my presence, but for a few seconds only ; the pulse fell suddenly from 166 to 80, and as he rose up the rate as sharply returned.

Such is the ordinary course of tachycardia, though cases of greater and of less severity occur. In some the initial symptoms are peculiar, or extremely severe. For instance, a seizure may be ushered in by indications of cerebral anaemia ; such as dimness of vision, buzzing, vertigo, or even syncope. Twice I have seen a characteristic and single attack arise suddenly in the course of ordinary acute pneumonia, about the fourth day : one of these cases was fatal, but after the tachycardia had ceased ; the other recovered without peril, and by normal crisis. In neither case did the patient seem much the worse for the incident, and in both it passed off as suddenly as it appeared. I was consulted in each case only on account of the alarm naturally excited by this event, and I saw neither patient again. In most of my own patients there was no audible abnormality of the heart, save the equal spacing and short sharp systole. In stages of cardiac exhaustion dilatation of the heart supervenes, gradually or suddenly, and may be irremediable. In one extreme case Dr. Mackenzie was able to watch an increase in transverse diameter of two inches in three hours ; the face became livid, and the veins of the neck rose up and pulsated in nodal rhythm. In 24 hours the legs were oedematous and the liver swollen. Notwithstanding, on cessation of the attack, and of the nodal rhythm, every vestige of heart-failure vanished. By orthodiascopy this dilatation appears wholly or mainly to affect the right ventricle, the arterial pressures falling coincidentally. Tricuspid regurgitation during attacks has been recorded by many observers. Although in all cases there is a subordinate disturbance of respiration, often an acceleration of it with sighing, yet it is to this late stage of distress that definite cardiac dyspnoea belongs. Oedema of the lungs is prone to occur in any seizure which lasts more than five days (Herringham), and may accentuate the other signs of dilatation ; such as the oedematous feet, the swollen liver, and albumin in a scantier urine. Repetitions of such incidents render the heart less and less able to recover its normal tone, and the ordinary symptoms of dilapidation may set in. Death may be by gradual failure or by syncope ; the attack itself is rarely fatal.

In the lady first mentioned, the initial attack cut short a prolonged and severe skipping effort, when she had reached a high tale of skips.

Ever since she has been subject to the attacks, but of late years they are not so severe. They seem to come on capriciously ; but she thinks more in spring and autumn than at other seasons. Sometimes they have been determined by a shock, physical or emotional, as once when she made a false step in the street and "jarred" her foot ; and once again when a drunken man accosted her. Dyspepsia may seem to call forth an attack, but by far the majority "come on of themselves." Her pulse generally runs at least to 200 ; the highest guess I made was 280. With her the attacks go off somewhat variously : either "hardly"—that is, more gradually with a peculiar sense of agony, when she used to think she would die—or suddenly with a thump or two. The attacks may last for a few seconds, a few minutes, or a few days ; some attacks have lasted as much as ten days, but this duration is unusual. After the first few minutes she is conscious enough of the beating ; it is like a rapid tapping or vibration : when she was younger it would shake the bed, and even light articles in the room. Some patients feel no more than an anxious fluttering or agitation within. In later years she has borne them better, probably they are milder ; she can even read during the attack : formerly she was prostrate throughout the course of them, and long after them ; but they are all exhausting, and leave behind them for some days weariness, and often some instability of rhythm. Her family history is very good ; her parents were hale octogenarians. No notable nervous disease has been heard of in the family ; there are no permanent signs of cardiac failure. In the epileptic case, the heart attack came on at 14, the fits at 42. The two maladies have seemed to move quite independently.

The urine, except for a common polyuria, is in the typical case normal ; the azoturia, albuminuria, or glycosuria, etc., found in complicated cases are inessential.

Pathology.—In the few careful necropsies on record, evidences of secondary cardiac failure apart, no constant changes have been found. Examination of the vagi, of the sympathetic nerves, and of the intra-cardiac ganglia have been negative, except for secondary changes such as a degeneration of muscle and ganglia in common. As then the evidence of the stethoscope, in respect of organic disease, is also negative, and as for many years at any rate the patients recover their ordinary health between the attacks, and may ultimately recover completely, we must regard tachycardia for the present as a functional disease. In cases which, after the lapse of years, have proved fatal, the necropsy may reveal, as in a case of Fräntzel's, fibroid degeneration of the walls of the heart and dilatation of its cavities in all directions. In another case total synechia was revealed, and in many valvular lesions more or less contingent or consecutive have been observed. Dr. E. E. Prest, in a graduation exercise at Cambridge, reported a case from the London Hospital (1902) in which 40 oz. of fluid were found in the pericardium, and the pericardium was much thickened ; but microscopically no change was found in the valves, in the myocardium, in the vagus nerves, or in the thyroid gland. The liver was not congested. There was obsolete

tubercle at both apices. The patient died at length of the pericardial effusion. Ultimately, then, the disease may wear out the heart ; but where or how it is engendered we know not.

Until recently, indeed, we had not even a hypothesis of tachycardia ; only conjectures of the kind which Martius calls *Scheinphysiologismus.* The phrase cardiac tetanus, so often used in this kind of connexion, is misleading and inadmissible. The heart never loses its refractory phase, however brief it may be ; and with every contraction work is done. Whatever the explanation, it must cover that of all accelerations over 172 ; for Trautweiler seems to have been correct in stating that this number is the extreme limit of ordinary cardiac accelerations (de la Camp, Stähelin). Unless some new and specific alternation intervene, no stresses, no diseases drive the heart above 170-172. I have collected some pulse-figures from such violent disturbances as rupture of a valve (aortic or mitral), and thrombosis of the pulmonary artery, but they did not exceed such numbers as 160, 145, 150, and the like. Other current conjectures worth consideration are as follows : (i.) That the vagi are spent, or thrown out of gear. The suddenness of the attacks, both in onset and issue, seems against the opinion that these nerves are spent ; thrown out of gear they may be. We know of many cases in which the vagi are thrown out of gear ; as for example in bulbar disease, or under the pressure of growths or glands (cf. Proebsting's well-known case), or in experiments upon animals ; but in tachycardia the rate of the heart seems too rapid for such a cause, if taken alone. It does not seem probable that abeyance of the vagi in man gives the heart play beyond 120 beats in the minute, or thereabouts. In Müller's case of tachycardia in the course of disseminate sclerosis the vagus nucleus was involved in one of the patches ; and in Reinhold's case of syphilitic arteritis with tachycardia the arterial disease had gravely deteriorated the bulb. No tracings of the movements of the vessels are given. Hoffmann (of Düsseldorf) seems disposed to assume a bulbar, or at any rate an intracranial origin ; but the protracted liability, and the evidence of not a few necropsies, in which the bulb and its vessels proved to be perfectly normal, seem to dispose of this opinion. Riegel observed that during the attacks the diaphragm sinks ; and his observation seems to have been verified by other observers. He attributes the disease accordingly to an affection of the phrenic nerve. (ii.) That the vagi may be in abeyance, and at the same time the accelerators excited or the vasomotor centre affected. This suggestion sins against the economy of causes ; and, as we must assume in each a close synchronism of disorder, we should be thrown back upon some cause behind them both. The features of the series are too uniform to make us content with Proebsting's dilemma, viz. that it depends sometimes on the one, sometimes on the other antecedent. (iii.) That the accelerators may be so stimulated as to overbear the normal vagi. Whatever the mechanical conditions, the onset and issue of the attacks seem in favour of some such supposition. In no experiment, however, has the rate been increased by more than 70

per cent by accelerator stimulation. Moreover, the accelerators exercise not a constant influence, but an auxiliary assistance ; and reflex irritations of eccentric origin do not push the heart beyond 150, if so far. Nevertheless, it is true that we need more experiments on this subject. The pupils often oscillate, and are described variously as normal, contracted, dilated, fluctuating, or even unequal. Here also more precise information is required. (iv.) That the cardiac ganglia are the seat of the disorder. But on necropsy we find either no changes in them, or none which is inconsistent with a secondary origin. (v.) Martius, in an able essay on the subject, argues that dilatation is the primary event, an opinion which Sir J. Barr had published a year before. Martius is prone to use technical terms in somewhat equivocal senses : but if he really means an acute myocardial insufficiency I must disagree with him. It is difficult to prove moderate dilatation by the evidence of physical signs, and Dietlen failed to find it by orthodiagraphy in 3 cases. Indeed, both he and Hoffmann allege on orthodiagraphic evidence that the volume of the heart is diminished. And Pawlow states that fatigue induced by irritating the accelerator fibres does reduce the heart's capacity. (See also " Over-stress of the Heart," p. 200.) The face is pale rather than cyanotic in the attacks, until the heart ultimately breaks down. What dilatation, clinically known to us, produces such a kind and degree of acceleration, with a regular rhythm and sudden onset and departure ? (vi.) Some sudden change in general blood-pressures ; but arterial blood-pressures are always rising and falling, and more persistent changes of pressure are soon compensated in the ordinary way ; furthermore, whilst no ordinary tides of blood-pressure, as we may test them by Mosso's or Dr. Leonard Hill's experiments (Junot's boot, etc.), seem competent to bring about extreme changes of rate, the cases present no evidence of extensive areas of anaemia or of vaso-dilatation. Recumbency or compression of the abdomen is of no avail. A fall of arterial pressure is very unlikely, and in such circumstances would be very perilous. Sir James Barr took the pressures during the tachycardial state in three cases; in none was there any very notable alteration. In one of them there was a slight increase. With such guesses as "excessive irritability" we cannot occupy ourselves ; it is the business of all thoroughbred neuro-muscular structures to be of high mettle; if they get out of hand, the fault is in the control. (vii.) Stoppage of a coronary artery is a *vera causa*, but we have no evidence of its occurrence in this disease. (viii.) One serious hypothesis remains, namely, a dislocation of the seat of rhythm. This hypothesis is, of course, incomplete ; it offers a very interesting step in advance, but why this dislocation occurs, and why it engenders tachycardia, and if it is invariable, are questions still unanswered. Perhaps indeed this change of seat may not always imply acceleration, although this usually comes about; possibly indeed the paradox is true that we may have a tachycardia *andante*.

Dr. James Mackenzie's hypothesis is based upon an application of Dr. Gaskell's physiological doctrines to clinical medicine. Mackenzie's records of the fluctuations of relative pressures in the chambers of the

heart by the polygraphic method, records which are a monument of thorough and systematic industry by no means confined to the subject immediately before us, have largely contributed to our understanding and classification of cardiac disorders. It is not too much to say that his researches have made all previous clinical work on the heart seem a little antiquated. And there can be no withstanding an observer who, in support of a particular proposition, is ready to produce from his stores five or six hundred exact and pertinent tracings. If ultimately some redistribution of the contents of the type may be found necessary, yet from his records we are now provided with a test of tachycardia proper ; and so far the clinical physician may be fortified in his experience that the symptomatic series named Tachycardia is sufficiently uniform to keep its place among the "Diseases." Moreover, we may have herein a clue to explain how at its margins paroxysmal tachycardia, like all other diseases, shades off into other symptomatic series ; for diseases are not close precincts of symptoms, but areas of their maximum density. Thus, if Mackenzie be right, paroxysmal tachycardia may glide in one direction into the accelerated and irregular pulses of mitral disease ; in another into states of ventricular and other extra-systole ; or otherwise again, paradoxical as it is, even into "Bradycardia."

From Dr. Mackenzie's jugular curves, it would appear then that paroxysmal tachycardia consists in a dislocation of the rhythmical centre ; in its displacement, that is, from the main or sinus centre to the nodal metacentre. Hering was disposed to regard tachycardia as a "tetanised" rhythm originating in the auricle ; yet, on comparing the sets of tracings, the precision of Mackenzie's jugular curves seems to make them almost indisputable. Moreover, from Hering's own laboratory, Rihl's polygraphic curves rather support the interpretations of Mackenzie. (See also Hering (17a).) Now equilibrium about a metacentre must be less stable—more "labile"—than about the main centre ; so that, dislocation having once occurred, we shall not be surprised if it occurs again ; or if, for instance, it skips still farther from the a.-v. bridge to a ventricular metacentre.

Recent observers, Schmoll for example, have stated that the numerous period of paroxysmal tachycardia consists, or is made up in part, of extra-systoles. It seems to me that in respect of normal paroxysmal tachycardia the term "extra-systole" is better avoided. In a normal tachycardia—in the concept, that is, which we still agree to use as a type —it is suggested that the inception of the beats has leapt from auricular or sinus centre to the nodal or a.-v. subcentre ; auricle and ventricle now contract simultaneously and equably, so that it is not easy to regard any one beat of the period more than another as an extra. If, however, the excitation skips back again for a moment to the main centre, so that an independent auricular beat intrudes ; or, again, if the excitation skips inconstantly forward from node to ventricle, we may call such intercalations extra-systoles.

We have got thus far then, that in paroxysmal tachycardia the spring of the rhythm seems to be dislocated from the normal centre to the meta-

centre in the a.-v. bridge ; but we have not got so far as to discover how this is brought about. On Mackenzie's curves auricle and ventricle contract simultaneously ; but as to the acceleration, whether it depends upon the comparative insecurity of the transitory balance, or upon some release from the vagus, which has more power upon the auricle, or upon some peculiarity of the node itself or of its innervation, or again upon some poison abating or abolishing the sinus centre, are mere guesses. Our data are still insufficient. Dr. Cowan's two cases, in which the auriculo-ventricular rhythm seemed to preserve the normal order, are not crucial ; the second case was complicated, and the first did not happen to exceed the 172 limit. As clinical observers, we note that the character of regularity, or comparative regularity, is so general a feature of these cases that for the present it must form a component of the concept or type of paroxysmal tachycardia ; we have observed, notwithstanding, that, by the caprices of change from centre to centre, irregularities of rhythm, which in themselves may or may not be of grave or crucial significance, may be introduced. But there are other kinds of irregularity which are only too grave. For instance, Dr. Mackenzie's records seem to reveal that in other series, in mitral disease, for instance, a leap of rhythm-inception from sinus centre to nodal centre often occurs. If so, there is a flank movement upon our explanation of paroxysmal tachycardia, seeing that in mitral disease the typical rhythm is as irregular, as in paroxysmal tachycardia, typically speaking, it is regular. In the one malady regularity seems normal to the nodal rhythm, in the other irregularity. As yet we can do no more than point out the apparent inconsistency, one which Dr. Mackenzie himself is not yet in a position to explain. Suffice it at this point to suggest that if cardiac failure or a more general exhaustion be concerned in the matter, this may be the element which perverts a regular into an irregular nodal rhythm ; or again, much may depend upon the variable blood-distribution and intraventricular tensions rather than upon labile balance or failing reserve. On Fig. 8, p. 18, it will be seen that, in this case of paroxysmal tachycardia, before the nodal rhythm became irregular, a sign of "exhaustion," namely, pulsus alternans, became perceptible.

Provisionally, then, we may suppose that normal paroxysmal tachycardia is an intrinsically cardiac fault, and consists in a sudden leap of rhythm from the sinus centre to a metacentre, and in a very rapid but regular succession of beats ; yet, as nodal rhythm is an unstable balance, if the heart is at a valvular disadvantage, is fatigued, is subject to uneven blood-supplies, or if its a.-v. bundle is encroached upon or degenerates, co-ordination of function, already in jeopardy, may be upset. If this be so, in paroxysmal tachycardia irregular rhythm, or pulsus alternans, is ominous of another phase—of the phase of dilapidation.

Of the lower range of numbers in the tachycardial pulse we have no definite information, but paroxysmal accelerations of moderate degree, if dislocated in rhythm, are to be accepted as paroxysmal tachycardia. In one of my cases the pulse rarely exceeded 160-170 ; but,

as 180 was reached occasionally, we inferred that it belonged, nevertheless, to the group of tachycardia, an inference supported by the other features of the case. At lower speeds, of course, the sounds are not so tic-tac. But we have to go much farther than this ; we have seen to our surprise that, in rare cases, paroxysmal tachycardia, departing from the type, may turn toward " Bradycardia," or may even be merged into it; or conversely a phase of tachycardia may intrude upon a case of bradycardia. The explanation of this contingency, if not as yet decisive, is perhaps not very far out of sight. In one of Dr. Walter James' cases, intercurrent attacks of paroxysmal tachycardia, at a rate of 204-250, thus intruded upon a typical Stokes-Adams series with a prevailing pulse-rate of 24-36 or less. Conversely, Dr. Clarke has published a case in which bradycardial phases, with Stokes-Adams phenomena, appeared in the course of paroxysmal tachycardia ; phases which he attributed to " exhaustion," but may have been jolts to a ventricular metacentre. More recently still, Dr. Gibson has produced a case of " heart-block " (brachial pulse-rate 36), in which atropine shot up the ventricular rate to 270 ; a phenomenon by no means easy to interpret. We know that upon the a.-v. bundle the conduction of the rhythm depends ; and that in any case a degradation of this bundle, transient or permanent, implies a halt in the rhythm. We may presume then that if the inception of the rhythm be suddenly thrown upon a bridge for some cause already unsafe, the unwonted burden may aggravate, or at any rate betray, its unsoundness ; and by retarded conduction the tachycardial phase may approach or be changed into a phase of bradycardia.

Finally, regularity of time and evenness of function may depend upon the precise seat in the bridge of the new rhythmical departure. It would seem that the capacity of propagating the rhythm is not confined to the middle of the bridge, but may shift a little toward its abutments, toward the auricle upwards, or downwards towards the ventricle ; whereby other variations may be instituted. Now the number of the rhythm, unless otherwise balanced, may be in some proportion to the volume of the part whence it springs. If pulsations start in the little auriculo-ventricular strip they are rapid ; if in the larger auricle, less numerous ; if in the ventricle, the number drops to 35, or less. Furthermore, much may depend on the site of its departure, even within the isthmus itself ; whether at the centre of the strip, or at the auricular or ventricular abutment of it. That the morbid rate is a double or other multiple of the normal (Hoffmann), is not my experience ; but finger counting at high rates is very fallible (p. 526).

Causes.—Why in paroxysmal tachycardia the starting-point of the rhythm should be thus jolted from sinus to a.-v. bundle we have no notion. We cannot do more than guess whether in this disease the causes of the dislocation lie in the cerebral cortex, in the mesencephalon, in the bulb, in the vagi, in the accelerators, in the cardiac ganglia or muscle, in areas of peripheral dilatation, or in eccentric irritation, such as floating kidney (Balfour, p. 84). Neuritis has been alleged as a cause ;

but there is no evidence of its presence, nor would it be consistent with the long intervals of health. But as the phenomena are remarkably uniform, the causation is probably not a complex one.

Of 126 cases collected by Hoffmann, 23 were attributed to "fright"; 3 to blows on the head; 2 to organic disease of the brain; 19 to debilitating causes, infections, etc.; 23 to abdominal visceral reflexes; 21 to physical strain; 24 to disease of the heart itself; in 19 no cause was apparent. In many cases the epigastrium is tumid, the tongue is foul, wind is belched up, and vomiting may occur.

Of other causes, over-exertion, dyspepsia, bodily or mental shock or emotion, gastroptosis (Reynaud, Weinstein), uterine disorders, auto-intoxication, loaded bowels, gout, sudden shocks, sudden straining, muscular efforts, heredity (Kirkland), have been accused with more or less hesitation. We may suspect that in many of these cases the name tachycardia was loosely used, and the condition was no more than a sympathetic acceleration. The victims of this disease are often, but not always, of the neurotic habit, and thus a few cases have seemed to be hereditary (Williams). One of my cases occurred in an epileptic, and Deganello, and I think others, have reported a similar concurrence. It has been alleged, indeed, that tachycardia presents some homologies with epilepsy, but the affinity, if any, is probably remote, or indirect. The same comment may be made upon the occasional alternations in the same person of tachycardia and migraine. The nervous centres, ganglia, and fibres have been examined and found negative, or at any rate without characteristic or consistent alteration. Ott's researches were very complete, and have been verified repeatedly. The pupils may dilate in any kind of faint.

Sex.—The records indicate that this factor has little or no influence in the causation of tachycardia; the disease falls almost impartially on the two sexes. Cases are recorded also from many countries and races.

Age.—In 40 cases of Dr. Herringham's collection the age was recorded. In 7 the malady dated from childhood; five of these were women. In 12 the first attack appeared between the ages of twenty and thirty; of these, 6 were men and 6 were women. In 13 cases the onset fell between the ages of forty and fifty; in 3 the patients were over fifty on the first attack. Dr. Watson Williams reports a case in a man of eighty, in whose attacks the pulse would leap suddenly from 60 to 130. H. C. Wood reports a case as still recurrent in a physician of eighty-seven years of age; the attacks began in his thirty-seventh year; the onset is abrupt, and the pulse rises quickly to 200.

Diagnosis.—Tachycardia as an intermittent disease is thus readily distinguishable from more abiding accelerations. The larval form of Graves' disease—that by no means uncommon form in which the thyroid is not enlarged nor the eyes prominent—may thus be distinguished from tachycardia; and so likewise post-typhoid or post-influenzal frequency. Fine tremor may be seen in many cardio-neurotic cases, as may exalted reflexes also. Tachycardia is not a mere incident of

nervous debility. Alcoholic acceleration—I do not refer, of course, to the temporary excitement of the debauch—may shew some paroxysmal behaviour, but more usually it is continuous ; and other evidence, such as morning retching, or before-breakfast diarrhoea, will be obtainable. The pressure of a tumour on the vagus may be attended with a persistent rapidity of pulse. In cases of idiosyncrasy, cases in which the pulse runs in the individual at accelerated rates, the persistence of the peculiarity again will decide the judgment against tachycardia ; and it may be added that in these cases, and in others of more or less persistently quick pulse, the patient suffers little or no distress. Cases are recorded on good authority in which the pulse of a person presumably healthy habitually ran at 150 a minute : Binswanger has recorded such a case in a woman ; in her the peculiarity had endured all her life. Balfour also refers to the case of a lady, then over seventy, who had had a large family and enjoyed good health, though of nervous temperament ; her pulse had never been under 150. I remember one day, when I was driving with a medical friend, a man passed us on horseback—a fine-looking country squire in whom was no apparent flaw ; my friend told me to note him as he passed, because his pulse ran habitually at 120. The owner of the pulse, patient I cannot call him, enjoyed fair health, but in his doctor's opinion he would be a "bad subject" for acute disease ; this opinion he founded not only on a mistrust of the pulse, but also on a certain lack in him of resistance to fatigue and trivial ailments. The tobacco pulse, if rapid (at first it is not quickened), is very irregular. Of heart-diseases the two most similar are simple dilatation and mitral stenosis. Although not dependent upon heart-disease, tachycardia may, of course, be associated with it ; but at such rates of speed, even if we presume that fluid veins could be effectively generated, no murmur could be made out. By one paroxysmal period only (as, *e.g.*, p. 499) tachycardia cannot be definitely diagnosed.

Some years ago I saw a woman, circ. sixty, with degenerated arteries, who was seized very suddenly with cardiac acceleration (for the first time). The rate was about 180. She died in two or three days substantially unrelieved. The heart's sounds were of course tic-tac ; no adventitious sounds. No necropsy. No notable pain nor dyspnoea ; but great distress. No physical sign other than the heart-hurry and the arterial disease. We attributed the seizure to an occlusion of one coronary artery or large branch. And, again, in an old gentleman whose heart was dilating, a tachycardial rate over 180 appeared. A definite murmur of mitral regurgitation had appeared before the attack, and reappeared after it. There were obvious signs of general cardio-arterial disease. The attack itself passed over, and I heard no more of the case.

Old men who continue in sexual indulgence may present pulses of acceleration or retardation ; but a heart-hurry which seizes upon a man for the first time in his old age, is not very likely to be tachycardia proper. On the other hand, as it often appears in early years, and for other negative reasons, it is probably not gouty. In the stormy onset

of some infection, or the prostrating stages of fevers, diarrhoea, influenza, and other toxic or exhausting conditions the pulse-rate may be enormous, but such instances are not likely to be misinterpreted.   In bulbar palsy the pulse is persistently altered ; if accelerated it is usually irregular and intermittent ; in tachycardia the rhythm is normally even : moreover, bulbar disease has its own features.   Besides, in none of these kinds is the disease paroxysmal.

Prognosis.—If the ultimate prognosis be doubtful, if in a certain number of cases death is premature, the immediate prognosis, in earlier years at any rate, if grave, is not without hope.   Life is often extended to full term.   I have mentioned Dr. Watson Williams' well-marked case in a patient aged—at the time of his writing—eighty years. Dr. Herringham thinks that after thirty years of age no patient of tachycardia is safe, and that few pass fifty.   This is a darker forecast than I should be disposed to make.   Much depends, as he says, on the duration of the particular attacks and on the frequency of their return ; if these last longer than five days the stress on the dilating heart accumulates, especially in elderly patients.   In one of Bouveret's cases, however, an attack of thirteen days' duration ended in recovery.   Four patients of my own are well past their climacteric ; and at least three others have recovered permanently.   One of these was a lady of highly-strung temperament past the menopause, who had suffered severely and frequently for many years.   After the trial of all kinds of remedy, and some temptations to seek a partial relief in morphine, she was cured decisively by Nativelle's granules, and "recovered her youthful activity and spirits." The second case was in a man of business, about sixty years of age, of keen temperament and oppressed by some anxieties.   In the first few of his visits to me, as I was not able to detect any cardiac disease, I had rallied him on his apprehensions ; but fortunately on the third visit an attack came on in my room.   He was gradually cured by cutting down a too abundant dietary, promoting his excretions, and reforming his engagements and vacations.   The third case of recovery I saw in April 1909, in Exeter, in a gentleman aged sixty-three, and of good constitution.   Some weeks before he had been thrown violently down, and his leg was injured.   This injury did well ; on my visit he had virtually recovered from it.   But since the accident he had suffered daily, or almost daily, from severe paroxysms of tachycardia, some of them lasting two or three days.   Sometimes 180 was exceeded, but the usual acceleration was about 150.   When I first felt his pulse it was very rapid ; ten minutes later the seizure had passed, so that I could examine his heart critically.   There was no perceptible defect.   As the malady arose from a transient cause we hoped, with continued rest and general amendment, that it would disappear ; and, happily, after some grave relapses, time has justified our hopes.   In the majority of patients as they advance in years, the return of the attacks is postponed ; the intervals are longer, the attacks more tolerable, and there is more time for rally.

Treatment.—Unfortunately we have no trustworthy means either to

cut short the attacks or to prevent them; but, as I have said, the attacks may get less both in number and severity with advancing years, or may pass altogether away; and, in some cases, we may prevent them by warding off contingent causes. The attack itself may be arrested by some empirical device, often discovered by the patient himself; and the device may in the individual case be as frequently successful as in other cases it may utterly fail. I have just said that in one of my cases Nativelle's granules proved, to my surprise, to be promptly curative. And in a few others I have found the tincture of digitalis in a little brandy certainly serviceable; the brandy seems to mitigate the nausea which in tachycardia the foxglove is especially apt to set up. However, brandy or no brandy, this drug is too often of little effect, and patients give it up. If, however, digitalis does not modify the rate of the heart, it seems to add to the diuresis, while in valvular disease digitalis only too often fails in this respect. Deganello and Baccelli report cures by daily intravenous injections of $\frac{1}{2}$-1 mgr. strophanthin; or of $\frac{1}{10}$ mgr. thrice daily. In other cases the camphor-oil injection seems to have proved efficacious. The hypodermic use of morphine, very cautiously controlled both as to dose and as to the danger of habituation, may help in surmounting a bad attack; but it is less successful than might be expected. Were the cause accelerator irritation it ought to be more efficacious. Dr. Herringham relies upon the subcutaneous use of atropine, and Dr. Rolleston has found it useful. One of my patients still clings with faith to a prescription of salicylate of sodium and sodium bromide which I gave her many years ago; she assures me that it is of much service in mitigating and abbreviating the seizures. I gave it on a strong hint of goutiness in her family. This patient has had a fibroid tumour for some years, but the attacks are of older date; there is no evidence that the fibroid has affected her tachycardia in any way for good or evil. I usually recommend compression of the abdomen with a binder, but I think it is seldom well applied; a trained midwife should be engaged to instruct the patient in the proper use of it. The bromides are of no considerable value, but, like valerian, in full doses, and the monobromide of camphor, may be of temporary service. Wood's patient was relieved by drinking iced water and strong coffee. Cold affusion, either to the chest or over a larger surface of the body, ice compresses over the heart, or, as some advise, on the nape of the neck, or very hot stupes, arrest the attacks in some cases (Eames, Macmillan), but in others fail as conspicuously. The graduated douche or the wet sheet are more appropriate for prevention. To torment the already tormented by emetics and other such violent means could only be justified by a success with which they cannot be credited. The application of electric currents, of this kind or that, to the vagi in the neck, or digital pressure on these nerves (bilateral compression should surely be very cautiously attempted?), have answered occasionally, but have usually disappointed expectations. Schlesinger, however, has recorded a curious case in which pressure on the right vagus arrested attacks, but failed to do so on the left side; at

the necropsy neuritis of the right vagus was discovered. We should have supposed that irritation of the vagus would have retarded the pulse, and that pressure on a paretic nerve would have had scarcely any effect. Finally, it is said that a compression of the chest by the patient himself sometimes succeeds in stopping an attack. I have not had a good opportunity of seeing this method properly tried. It is to be essayed as follows :—The patient will thrust his feet as hard as he can against the foot of the bed ; then, pressing his arms closely into his sides, he will take a long inspiration ; in the next place, closing the glottis, he will make a strong expiratory effort, thrusting hard the while against the walls of the chest with the upper arms, and clasping them with the forearms. In this way it is said that the rate of the heart may be directly controlled. By such a method an old friend of mine used to make his heart intermit. Such an expedient may give a sudden jar or pull to the circulatory system, a jar which, like the shake of a kaleidoscope, may dislocate the cardiac system into another attitude of equilibrium—from a temporary and unstable equilibrium back to the more stable centre of health.

During the intervals of quiescence persevering efforts must be made to nourish and invigorate the system. The digestion and the excretory organs are to be vigilantly watched and corrected, and all means adopted to secure serenity of life and a wholesome and regular occupation. I have referred more than once to cases in which these hygienic means sufficed for a permanent cure ; one of these cases, however, had lasted only for a year or two, another for a few weeks. One of my tachycardiacs began to ride a bicycle with much advantage ; the bicycle is better than horse exercise, for a horse may, and often does, make a sudden demand on the rider's nerve. " Heart massage " seems to me to be no more than ordinary massage plus suggestion ; but ordinary massage is useful in emaciated or podgy people ; in more vigorous patients Swedish gymnastics may be cautiously used with advantage, to open the way to more interesting forms of exercise. It will be remembered that as any over-exertion or stress may bring on an attack, the treatment must be trimmed between the extremes of indolence and fatigue. To dwell upon soothing body and mind, upon avoidance of worry or sudden effort, and so forth, would be to dwell upon platitudes which bear no more upon this than upon all cardiac maladies. The alleged curative effects of moral control, psychic ascendancies, and so forth probably derive their vogue from errors of diagnosis, upon a confusion between paroxysmal tachycardia and more vulgar storms in neurotic vessels.

RETARDED PULSE.—The researches of Gaskell, Wenckebach, Mackenzie, and others indicate that the name Bradycardia, which, like tachycardia, was invented as a bit of finery, may be retained and turned to a useful purpose to signify a reduced conduction, temporary or permanent, of the " auriculo-ventricular conductive bundle." If so, the label must not be attached indiscriminately to toxic cases ; as of jaundice, of vagus

interference, of high arterial pressures, and so forth. In this section " heart-block " with its train of nervous symptoms (Stokes-Adams disease) will not be discussed. Even if it be true that bradycardia with all the Stokes-Adams train, or at any rate up to syncope, may depend on transient causes, and in some cases perhaps may consist only in " lost systoles "—systoles, that is, which fail to open the semilunar valves—yet notwithstanding we must for the present carry all these cases on to another chapter, to the class of Bradycardia, to which they essentially belong. Here we have to discuss only the retardations which may occur independently of organic disease, at any rate of the heart itself. Digitalis, anaemia, certain infections and other similar contingencies may retard auriculo-ventricular conduction ; or, as a whole, the heart may be retarded by a vagus pull on the auricle, an interference which the jugular pulse and the atropine test would distinguish. I saw a case recently of considerable retardation in which no isolated auricular beats could be detected. Indeed the x-rays have demonstrated that the heart may descend in rate to 20-25 beats with a regular atrio-ventricular order. Vagus retardation alone probably does not fall below 45-40, and it should be succeeded by a phase of reinforcement.

It is now more generally recognised that slowing [1] of the pulse is by no means invariably due to the control of the vagus on the one hand or to general " fatty degeneration " of the left ventricle on the other ; though in these cases it may be hard to say how much of the retardation may be due to its vigilant nursing. In such states as senile bronchopneumonia, where the tendency is to atonic dilatation, the action of the vagus, whatever its immediate protection, is apt to turn to ill, even in the heart itself ; for vagus action reduces the work as well as the rate of the heart, so that, unless amply reinforced by the quiescence, the organ may not overtake its arrears. Of the kinds of retardation these convenient classes may be made :—(i.) Rise of blood-pressure, as seen in its simplest form in the " expiratory diminution of rate " ; or, conversely, in the temporary suspension of vagus action by continual sipping, or even by one act of swallowing (Waller ; and James Mackenzie's notable case H. D.) ; (ii.) intrinsic poisons, such as carbonic acid or those of uraemia and jaundice ; or extrinsic poisons, such as lead, tobacco, digitalis ; or bacterial products, as in diphtheria, some of which, like carbonic acid, act directly on the vagus or its centre, some on the heart itself ; (iii.) reflexes from eccentric derangements ; such as those arising in the heart, in the gastro-intestinal canal (dyspepsia, gall-stone, etc.), in the pelvic organs, in the throat or ear, in a distended bladder, and so forth ; (iv.) the infrequent pulse of children ; (v.) that of hysteria, melancholia, and other psychical disorders ; (vi.) that of " exhaustion," as in convalescence, or after great fatigue ; (vii.) and that of pain (as illustrated by experimental irritations

---

[1] It is not improper to speak of an infrequent pulse as " slow." Slow is not a technical term, it is a common adjective, not of precision. It may be used of the rate of procession as well as of the terms of the series. " How slowly do the Hours their numbers spend ! " One may cross the street slowly by slow paces, or by a slow succession of them.

of a sensory nerve). The alterations of pulse due to cerebral, bulbar, cervico-spinal, and other exocardial disease, fall outside our subject. A marvellous case of retarded pulse (of category ii. or vi.) in which the heart-beat, during the worst phase of it, was reduced to 15, I saw repeatedly with Mr. Teale at Manningham many years ago. The patient was a middle-aged man of enormous energy, and no less enormous business adventures; and he indulged himself freely in the pleasures of the table. Under some obscure but temporary high intracranial vascular pressure, not haemorrhagic nor obviously uraemic, he became semi-comatose; Cheyne-Stokes respiration persisted in a severe degree for some days, and his pulse fell lower and lower in number. I cannot say what part the auricles took in the retardation; I have no notes of the case. He made a complete recovery, and threw himself into life again as ardently as ever, and apparently with impunity.

Retarded pulse is said to be frequent in childbirth, and certain continental physicians attribute it, with little probability, to a temporary excess in the blood-mass. Prof. Osler agrees that the pulse is retarded in parturition, whether premature or at term; the rate, he says, may decline from 60 to 44, and has sometimes fallen as low as 34.

As regards functional retardation and epileptiform attacks, I have never had my finger on the pulse of an epileptic just before or at the very initiation of an attack; but scores of times, as for instance in the wards of lunatic asylums, I have felt the pulse as the seizure became manifest : I have never, however, found any peculiar change in the rate. I find that Sir W. Gowers makes the same remark. In the cases of association of slow pulse with epileptiform convulsion, as in Stokes-Adams disease, uraemia, or severe haemorrhage, it still seems probable that the slackening of the pulse is the essential antecedent, though the consequence is far from invariable. A crucial instance is that of Lépine and Porot, in which a pulse of 26-34, with syncopes, proved to be due to narrowing of the occipital foramen (6 mm. in all diameters). Lépine had published a similar case twelve years previously. In Holberton's classical case a slight transient injury of the cervical cord was accompanied by a pulse-rate of "$7\frac{1}{2}$ per minute" (*vide* p. 145).

In some persons an infrequent pulse pertains to their normal state. Of "normal slow pulse" we see many examples; but we have to assure ourselves of what is normal to the individual. I cannot agree with Riegel, Eichhorst, and others that a pulse under 60 is presumably morbid. In athletes in good training the pulse-rate is less frequent than is generally supposed; in them during rest and recumbency the rate often falls below 60 (*vide* "Over-stress of the Heart," p. 200). In the Radcliffe Infirmary, during the Michaelmas examination for the M.B. degree in 1897, my colleagues and myself noted, in a vigorous, cheerful old man, a pulse of 28. By a little exercise or excitement it could be raised to 32 or 33. As is usual in weather-beaten labourers over sixty years of age, his arteries were somewhat thickened. Of the rate of his pulse in former years he knew nothing: he had never noticed his pulse until we called

attention to it. It may have changed gradually as he grew older. He had been admitted for some trivial ailment, but the candidates found nothing else to report of him; and Dr. Samuel West, Dr. Mallam, and myself found him free from any other malady than that of advancing years. He felt very well, and was vastly amused by our determination to find some fault in him. A pulse of 50 is no very uncommon rate in healthy persons, rather in men, perhaps, than in women; in a friend of my own a pulse of 55 or less, sometimes slowing down on fatigue to 45, or rarely to 42, has proved consistent with great nervous and muscular activity up to years which are now more than mature. For him a pulse of 80 is fever; it never rises over 100 or thereabouts, except during active muscular exertion, when it will reach 110. Corvisart's record of Napoleon's pulse as habitually 40 is well known; Sir William Broadbent, I believe, recorded the case of an athlete with a pulse of 36; but a pulse under 40 is strongly suggestive of some morbid condition. It is scarcely necessary to say that in all cases of infrequent radial pulse the number of the cardiac revolutions must be counted at the centre; for some of the waves may fail to reach the periphery; or auricular waves may not be propagated in due time. Or an apparent fall, even to 40, may be the effect of extra-systoles failing to raise the semilunar valves ("frustrate contractions"). Hearts naturally infrequent are more subject to extra-systoles. Roy used to say that in man a healthy heart might drop six beats and recover; but can a deteriorated organ cross such an abyss of time? Yet we read of pulses of 20—nay, of 12 a minute; of stops of 15 seconds' duration—in one instance an arrest of 30 seconds is alleged. I have counted a pulse-rate in Stokes-Adams disease of 8 at the ventricles, but between these beats auricular beats were occurring in some frequency. Moreover, ventricular beats, if very feeble, may be inaudible. An infrequent radial pulse due not to extra-systoles but to failure of ventricular projection is always a grave symptom, and in the course of acute diseases is of sinister omen. It is virtually the same as pulsus alternans.

All I know definitely about "hysterical slow pulse" I have found in von Noorden and Buchholz. If I have seen it I have made no note of it. The reference may be to the vaso-vagal cases (p. 515), which are not peculiar to hysteria. In melancholia the pulse is often retarded, and of high pressure. In respect of poisons we know that some of them, such as lead, may act indirectly by perverting the metabolism of the body, and thus generating intermediate toxins; uraemia and jaundice are often associated with a slow pulse. Within a few weeks before writing these words I have twice seen pulses under 50, at the heart, in undergraduates of sound constitution but suffering under attacks of catarrhal jaundice. Two years ago I witnessed a like reduction in the catarrhal jaundice of a young man aged twenty-five, who, however, made a complete recovery. It does not seem to occur in the jaundice of carcinoma, nor indeed in the infectious kinds. I allude to these cases because some doubt has been thrown upon this effect of jaundice, noted by Galen,

re-noted by Avicenna, and emphasised by Bouillaud.   In its later stages the pulse quickens, as in icterus gravis, and thus forbodes evil.   Perhaps these toxic substances irritate the vagi, centrally or peripherally.   The poisons generated by bacteria—the infections—not infrequently begin by stimulating the vagi, so that the pulse is slowed ; then as the vagus is exhausted the pulse quickens, and in later stages may be much accelerated.   In convalescence generally the bulbar centres seem unstable, so that the pulse is retarded or hastened by influences which in the normal state are indifferent.   That muscarine slows the pulse is a familiar laboratory demonstration ; and the accelerating effect of its antidote atropine is more familiar still.   Tobacco, again, probably by the rise of arterial pressure, stimulates the vagi at first.   Rise of blood-pressure may retard the pulse remarkably, and this apparently without any nervous mediation, as all clinical observers and physiologists are aware ; high pressure and over-full chambers seem to retard conduction ; but the rule that the rate of the pulse is inversely as the blood-pressure is open to many contingencies ; it holds only when other things are equal.   In the slow pulse of exhaustion blood-pressures are presumably low.   In vomiting, again, the pulse slows while pressure is falling.   If vagus irritation be its cause, the low pressure is due in part at any rate to reduction as well as delay of the impulse ; the residual blood in the left ventricle is more.   A succeeding phase of reinforcement ought to follow.   I have seen vagus retardation fall to 45 in persons whose pulse in the normal state was of ordinary frequency.   In some cases of temporarily retarded pulse with "nervous exhaustion" the voice becomes hollow or feeble, the vision may be dim, and the ears may sing.   In one such case I remember the patient, partly in timidity perhaps, intimated that he was too much exhausted to speak above a whisper.

Sexual exhaustion may reduce pulse-rate.   Just before writing on this subject in the first edition, a patient was sent to me with a slow pulse, about 40, attended by a sense of depression, almost melancholic, especially of a morning.   It was a great effort for him to get up to breakfast ; although after he had got to work or play the sensation would wear off.   At the times of slow pulse the temperature would fall to 95°.   He was in business, but in an easy one ; he had no cares, his habits appeared to be correct, and he had had no troubles.   He was fond of physical exertion, and could and did ride, shoot, and so forth, even to the full, without being the worse for it.   His age was 40.   On examination of his heart nothing abnormal was to be found.   His own medical man had cut down his tobacco (usually $2\frac{3}{4}$ ounces a week) without much relief.   I ascertained that he gave himself up to excessive marital intercourse, even to daily indulgence.   The prescription of a separate bedroom soon cured him.

Some of the cases of slow pulse in children may be due to self-abuse ; but by no means all.   To find a pulse of 50 or even 45 in a little boy or girl, perhaps with some extra-systolic arrhythmia, used to alarm me, lest there were some cerebral irritation ; but as, often enough, nothing

happened, I gained confidence ; and am now, if still on my guard, not prophetic of evil. In some cases worms may be the cause of the retardation ; but antidotes for worms do not always prove the connexion. Nevertheless, as some arrhythmia may be present, and some heaviness or drooping manner may be exaggerated by anxious parents, these cases for a few days may give reason for some uneasiness. "Gastric catarrh " is among the causes of it ; in the child the vagus centre, like the temperature centre, is more susceptible than in later years. Moreover, a little watching will shew that the rate is much under the influence of the breathing. The ages of such patients run from four or five to fourteen or fifteen ; the child may be languid and out of spirits, or dyspeptic, when the state of the pulse is found out, as it were accidentally. Irritation of the vagi cannot be the invariable cause. Slow-pulse children are usually·of unstable nervous constitution, at any rate in that stage of their development.

The lagging pulse of convalescents from fevers and other exhausting diseases, already referred to, is a common event, and is sometimes suggestive of cerebral complications, especially in children ; this again may be due to vagus irritation, to myocardial exhaustion, or even to poisoning of the cardiac muscle. I have seen it in severe bronchitis with distended right ventricle, much residual blood, and greatly overcharged veins. Intermittence by extra-systoles in these cases may point to vagus protection ; but unequal cardiac contractions may signify a much graver state of affairs.

The slow pulse of pain is no unfamiliar phenomenon, and one full of interest : it is surely due to reflex stimulation of the vagus, with or without consciousness, and can be produced by experiment ; for instance, by the inhalation of irritating vapours. Sir Richard Douglas Powell mentioned an interesting case of this kind at the meeting of the British Medical Association in 1894. The patient was subject to neuralgia, and to palpitation ; but not simultaneously, for an attack of pain might stop the cardiac disturbances. Sciatica is perhaps the pain most efficient in producing inhibition ; but almost any sudden paroxysm of pain of sufficient severity may be reflected in the pulse. Its chief interest lies in the peril of it to an enfeebled heart, as in renal or hepatic calculus, or in angina pectoris. How far a lagging pulse may be due to a failure of the accelerators we cannot tell ; but in cases of "exhaustion " such may be the case, entirely or in part. Of the intimate relations of the intra-cardiac ganglia to the functions of the heart we know little, or nothing more than that they are of motor affinity ; Dr. Gaskell regards them rather as survivals of the nervi vasorum than as dominant factors in mammalian cardiac function. Of retardation due to disease in the bulbar area we have many records : such as Brissaud's quasi-Stokes cases—the one of syphilitic lesion at the origin of the vagus, the other of lesion involving with it certain other nerves (Bell's palsy, trigeminal neuralgia, etc.). To the retardations of concussion, meningitis, uraemia, a passing allusion will suffice. The same brief allusion will suffice for encroachment on the vagus by local disease in or near it.

Retardations of the pulse are often associated with extra-systoles and other arrhythmias, and depend on a "dyspepsia" which a few doses of calomel will often dispel; and prevention will consist in diet and careful mastication of the food.   Such disturbances are often worse after meals, and are attended with flatulence; when, rightly or wrongly, they are called gouty.   They are commonly associated with the intermittence of extra-systole, or with flutters.   Momentary efforts may aggravate the condition, but in a sound heart persistent exercise removes it, for the time.   The urine must, of course, be minutely and repeatedly examined, and signs of cardio-arterial degeneration duly appraised; remembering, however, that, if due to atrophic or fibrotic deterioration of the myocardium, especially if for some while it reduces the value of the auriculoventricular bundle, other signs of gravity may be absent, or very indefinite. In all such cases of doubt the physician by means of the stethoscope, the x-ray screen, the state of the jugular pulse and so on, will test the time-relations of the auricles and ventricles.   In most if not all the cases under present consideration these relations will prove to be normal, the auricles being retarded *pari passu* with the ventricles and without alteration of rhythmical departure.   A ventricular rate of 40 will duly succeed an auricular of the same rate.   Yet even in cases of degenerate auriculo-ventricular bridge the synchronism may persist for a time.

<div align="right">CLIFFORD ALLBUTT.</div>

REFERENCES

1. ALLBUTT, Sir C.   "Cardiac Arrhythmia," *Med. Chron.*, Manchester, 1901-2, 4th ser. ii. 321.—2. BALFOUR, G. W.   *The Senile Heart*, London, 1894.—3. BINSWANGER, O.   *Neurasthenie*, Jena, 1896.—4. BOUVERET, L.   "De la tachycardie essentielle paroxystique," *Rev. de méd.*, Paris, 1889, ix. 753, 837.—5. BROADBENT, Sir W.   "Irritable Heart and Transient Murmurs as a Cause of Rejection of Candidates for the Army and Public Services," *Lancet*, London, 1897, ii. 1260.—6. BUCHHOLZ. *Beitr. z. Kenntniss der Vagusneurosen*, Berlin, 1892.—7. DE LA CAMP.   "Beitr. zur Physiologie und Pathologie der Zwerchfellathmung, einschliesslich der zugehörigen Herzbewegungen," *Ztschr. f. klin. Med.*, Berlin, 1903, xlix. 411.—8. DA COSTA, J. M. "Cardiac Asthenia, or Heart Exhaustion," *Amer. Journ. Med. Sc.*, Phila., 1894, cvii. 361.   8a. *Idem.*   "On Irritable Heart," *Ibid.*, 1871, lxi. 17.—9. DETERMANN. "Herz- u. Gefässneurosen," *Samml. klin. Vortr.*, Leipzig, 1894, N.F. (M.) Nr. 30.— 10. DIETLEN, H.   "Orthodiagraphische Beobachtungen über Veranderungen der Herzgrösse bei Infektionskrankheiten," *München. med. Wchnschr.*, 1908, lv. (ii.) 2057, and other papers, 1905-8.—11. FARQUHARSON, R.   "Case of Unusually Rapid Action of Heart," *Brit. Med. Journ.*, 1875, i. 770.—12. FOXWELL, A.   "Causation of Functional Heart Murmurs," *Lancet*, London, 1899, ii. 1209.—13. GERHARDT, C. "Ueber einige Angioneurosen," *Samml. klin. Vortr.*, Leipzig, 1881, (M.) No. 70. 13a. GIBSON, G. A.   "Miners' Heart in Nervous Diseases of Heart," 1904.—14. GORDON, W.   "Posture and Heart Murmurs," *Brit. Med. Journ.*, 1902, i. 636.— 15. GOWERS, Sir W. R.   "Vagal and Vaso-vagal Attacks," *Lancet*, London, 1907, i. 1551.—16. HERING, H. E.   "Zur experimentellen Analyse der Unregelmässigkeiten des Herzschlages," *Arch. f. Physiol.*, Bonn, 1900, lxxxii. 1.—17. *Idem.*   "Das Wesen des Herzalternans," *München. med. Wchnschr.*, 1908, lv. (ii.) 1417.— 17a. *Idem.*   *München. med. Wchnschr.*, 1909, lvi. 845. — 18. HERZ, L.   "Das schwache Herz" (weakened heart), *Wien. med. Wchnschr.*, 1893, xliii. 1761. —19. HOFFMANN, A.   "Acute Herzdilatation und Cor mobile," *Deutsche med. Wchnschr.*, Leipzig, 1900, xxvi. 306.—20. *Idem.*   *Herzneurose*, Wiesbaden, 1901. 21. JANOWSKI.   "Le diagnostic functionnel," Art *Cœur, L'œuvre med. Chir.*, 1908, No. 5.—21a. KIRKLAND.   "Inherited Tachycardia," *Lancet*, London, 1909, i.—22. KRESS.   "Zur Frage der acuten Herzdilatation," *Neurol. Centralbl.*, Leipzig, 1905, xxiv. 882.—23. MACKENZIE, JAMES.   *Diseases of the Heart*, Oxford,

1908.—24. MANN, J. D. "Some of the Effects of Excessive Smoking," *Brit. Med. Journ.*, 1908, ii. 1673.—25. MERKLEN, P. "Capacité fonctionelle du cœur," *Presse méd.*, Paris, 1903, xi. 197.—25a. *Idem.* "Troubles fonct. du cœur," Paris, 1908.—26. MITCHELL, J. K. "Notes on Irritable Heart in Neurasthenic Cases, and the Effect of limited Muscular Action on the Heart in Health and Disease," *Trans. Coll. of Phys. of Phila.*, 1892, 3rd ser., xiv. 132.—27. NOORDEN, C. VON. "Ueber hysterische Vagusneurosen," *Charité-Ann.*, Berlin, 1893, xviii. 249.—28. OSLER, W. *Practice of Medicine*, London, 6th edit., 1905.—29. POWELL, Sir R. D. "Functional Diseases of the Heart," *Brit. Med. Journ.*, 1894, ii. 1034.—30. *Idem.* *Treatment in Diseases and Disorders of the Heart*, 1899, H. K. Lewis, London.—31. ROLLESTON, J. D. "Diphtheritic Paralysis," *Practitioner*, London, 1909, lxxxii. 110.—32. RUDOLF, R. D. "Functional Heart Murmurs," *Intern. Clinics*, Phila., 1905, 14th ser., iv. 104.—33. SANSOM, A. E. "The Irregular Heart : A Clinical Study," *Trans. Med. Soc.*, London, 1893, xvi. 100.—34. *Idem.* "Functional Disease of the Heart," *Brit. Med. Journ.*, 1894, ii. 1040.—35. *Idem.* "The Irregular Heart after Influenza," *Ibid.*, 1897, ii. 1090.—35a. SNELL, S. "Miners' Heart," *Trans. Sanit. Inst.*, 1895.—36. STARCK, H. "Zur Frage der akuten Herzdilatation," *München. med. Wchnschr.*, 1905, lii. 302.—37. STEWART, G. N. "Researches on the Circulation Time, and on the Influences which affect it : IV. The Output of the Heart," *Journ. Physiol.*, Cambridge, 1897-98, xxii. 159.—37a. WEISS und JOACHIM. *Arch. f. Physiol.*, Bonn (Pflüger's), 1908, cxxiii. 341.—38. WENCKEBACH, K. F. "Ueber pathologische Beziehungen zwischen Atmung und Kreislauf," *Samml. klin. Vortr.*, Leipzig, 1907, N.F. (M.) Nr. 140-1.—39. *Idem.* *Die Arythmie als Ausdruck bestimmter Functionsstörungen des Herzens*, Leipzig, 1903.

Tachycardia : 40. BACCELLI. *La Via delle Vene*, Rome, 1907.—41. BARR, Sir J. "Clinical Lecture on Paroxysmal Tachycardia," *Brit. Med. Journ.*, 1904, ii. 109.—42. BENSEN, R. "Ein Fall von Innervationsstörung des Herzens" (Case cured by pressure in the neck), *Berlin. klin. Wchnschr.*, 1880, xvii. 248.—43. BOUVERET, L. "De la tachycardie essentielle paroxystique" (Cases and digest), *Rev. de méd.*, Paris, 1889, ix. 753, 837.—44. BOWLES, R. L. "Unusually Rapid Action of the Heart" (Two cases), *Brit. Med. Journ.*, 1867, ii. 53.—45. BRIEGER, L. "Beitrag zur Lehre von der anfallsweise auftretenden Tachycardie" (Case, with necropsy), *Charité-Ann.*, Berlin, 1888, xiii. 193.—46. BRISTOWE, J. S. "On Recurrent Palpitation of Extreme Rapidity in Persons otherwise apparently healthy" (Cases with criticisms), *Brain*, London, 1888, x. 164.—47. BUCKLAND, F. O. "A Case of Rapid Heart" (Case in a child after measles), *Trans. Clin. Soc.*, London, 1892, xxv. 92.—48. BUNZL-FEDERN, E. "Ein Fall von Tachycardie und Augenmuskellähmungen" (Case, with ocular palsy), *Prag. med. Wchnschr.*, 1891, xvi. 496.—49. CAVAFY, J. "Unusually Rapid Action of the Heart," *Brit. Med. Journ.*, 1875, ii. 294.—50. COTTON, R. P. "Notes and Observations upon a Case of unusually Rapid Action of the Heart" (232 per minute), *Brit. Med. Journ.*, 1867, i. 629 ; "Additional Notes," *Ibid.*, 1869, ii. 4.—51. COWAN, J., MACDONALD, D.. and BINNING, R. I. "The Venous Pulse in Paroxysmal Tachycardia," *Quart. Journ. Med.*, Oxford, 1909, ii. 146.—52. DEBOVE et BOULEY. "De la tachycardie paroxystique essentielle" (Case), *Bull. et mém. Soc. méd. des hôp. de Paris*, 1890, 3me sér., vii. 953.—53. DEGANELLO, M. "Sopra un caso di tachicardia parossistica essenziale," *Policlin.*, Roma, 1908, xv., sez. med. 57 ; [abstr. in] *Brit. Med. Journ.*, 1908, i. Epitome 89.—54. ECCLES, A. S. "Paroxysmal Heart-hurry associated with Visceral Disorders" (Cases), *Lancet*, London, 1891, ii. 118.—55. EICHHORST. *Handbuch*, 6th edition, i. 245 *et seq.*—55a. FAISANS. "Tachycardie essentielle paroxystique" (Cases in a family with malaria), *Bull. et mém. Soc. méd. des hôp. de Paris*, 1890, 3me sér., vii. 964.—56. FRAENKEL, A. "Zur Lehre vom weakened heart nebst Bemerkungen über das Symptomenbild des kardialen Asthma und dessen Behandlung" (Case after compound fracture, with post-mortem), *Charité-Ann.*, Berlin, 1878, v. 273.—57. FRÄNTZEL. O. "Ein Fall von paroxysmaler Tachycardie" (Case), *Ibid.*, 1889, xiv. 357.—58. *Idem.* "Ueber Tachycardie," *Deutsche med. Wchnschr.*, Leipzig, 1891, xvii. 321.—59. FREYHAM. "Ueber paroxysmale Tachycardie" (Cases ; argues that the disease is a neurosis), *Ibid.*, 1892, xviii. 866.—60. GERHARDT, C. "Ueber einige Angioneurosen," *Samml. klin. Vortr.*, Leipzig, 1881 (M.) Nr. 70.—61. HAMPELN, P. "Ueber einen Fall habitueller und paroxystischer Tachycardie mit dem Ausgange in Genesung" (Curious case after pericarditis), *Deutsche med. Wchnschr.*, Leipzig, 1892, xviii. 787.—62. HERRINGHAM, W. P. "Concerning Paroxysmal Tachycardia," *Edin. Med. Journ.*, 1897, N.S., i. 366.—63. HIRSCHFELDER. "Obser-

vations on Paroxysmal Tachycardia," *Johns Hopkins Hosp. Bull.*, Balt., 1906, xvii. 337; and 1908, xix. 322.—64. HOCHHAUS, H. "Beiträge zur Pathologie des Herzens: III. Ueber Tachycardie" (Case, with necropsy), *Deutsch. Arch. f. klin. Med.*, Leipzig, 1893, li. 17.—65. HOFFMANN, A. "Neue Beobachtungen über Herzjagen," *Ibid.*, 1903, lxxviii. 39.—66. HUBER, A. "Ein Fall von paroxysmaler Tachycardie" (Case, with hysteria), *Ibid.*, 1891, xlvii. 13.—67. HUPPERT, M. "Reine Motilitats-Neurose des Herzens," *Berlin. klin. Wchnschr.*, 1874, xi. 223, 237, 247, 261.—68. KELLY, A. O. J. "Essential Paroxysmal Tachycardia. Report of four Cases "), *Med. and Surg. Reporter*, Phila., 1896, lxxv. 513.—69. KIRSCH, T. "Herzkrank oder Magenkrank? Ein Beiträg zur Kenntniss der Innervationsstörungen des Herzens" (Digest, with argument for reflex origin), *Deutsche med. Wchnschr.*, Leipzig, 1892, xviii. 726.—70. KLEMPERER, G. "Ueber Tachycardie," *Ibid.*, 1891, xvii. 335.—70a.—LEWIS, T. "Paroxysmal Tachycardia," *Heart*, London, 1909, i. 43. (This article appeared while the volume was in the press and, unfortunately, too late for consideration.)—71. LOESER, H. A. "Ueber paroxysmale Tachycardie," *Virchows Arch.*, Berlin, 1896, cxliii. 648.—72. MACKENZIE, JAMES. "Diseases of the Heart," 1908.—73. *Idem.* "Abnormal Inception of Cardiac Rhythm," *Quart. Journ. Med.*, Oxford, 1908, i. 39.—74. *Idem.* "The Extra-Systole," *Ibid.*, 1908, i. 131, 481.—75. MARTIUS. *Par. Tachycardie*, Stuttgart, 1895.—75a. MERKLEN. *Thèse de Paris*, 1902.—76. NUNNELEY, F. B. "Observations on Palpitation of the Heart and its Treatment," *Lancet*, London, 1871, i. 228, 265.—77. OLIVER, T. "A Case of Tachycardia or Rapid Heart successfully treated by Electricity and Large Doses of Belladonna" (Case, after injury), *Brit. Med. Journ.*, 1891, i. 217.—78. OSLER, W. *Principles and Practice of Medicine*, London, 6th edit., 1905. —79. OTT, A. "Zur Kenntniss der Ganglienzellen des menschl. Herzens," *Prag. med. Wchnschr.*, 1887, xii. 159.—80. *Idem.* "Topographischen Verhältnisse der Ganglien am menschlichen Herzen," *Berlin. klin. Wchnschr.*, 1889, xxvi. 291.—81. *Idem.* "Beiträge zur Kenntniss der normalen und patholog. Verhältnisse der Ganglien des menschlichen Herzens," *Ztschr. f. Heilk.*, Prag, 1888, ix. 271.—81a. PAWLOW. *Arch. f. Anat. u. Physiol.*, 1889.—82. PREISENDÖRFER, P. "Ueber reflectarische Vagusneurose," *Deutsch. Arch. f. klin. Med.*, Leipzig, 1880, xxvii. 387.—83. PROEBSTING, A. "Ueber Tachycardie" (Critical digest, based on Gerhardt's cases), *Ibid.*, 1882, xxxi. 349. —84. REYNAUD, G. "Tachycardie symptomatique paroxystique et gastro-entéroptose," *Rev. de méd.*, Paris, 1908, xxviii. 172.—85. RIHL, J. "Analyse von fünf Fällen von Ueberleitungsstörungen," *Ztschr. f. exper. Path. u. Therap.*, Berlin, 1905, ii. 88.— 86. ROSE, U. "Ueber paroxysmale Tachykardie," *Berlin. klin. Wchnschr*, 1901, xxxviii. 713, 744.—87. ROSENFELD. "Zur Behandlung der paroxysmalen Tachycardie" (Treatment by compressing the thorax), *Verhandl. d. Kong. f. innere Med.*, Wiesbaden, 1893, xii. 327.—88. SCHLESINGER, H. "Ueber die paroxysmalen Tachykardie," *Samml. klin. Vortr.*, Leipzig, 1903, N.F. (M.) Nr. 105; *Centralbl. f. innere Med.*, Leipzig, 1903.—89. SCHMOLL. "Paroxysmal Tachycardia" (Treatment by baths), *Amer. Journ. Med. Sc.*, Phila., 1907, cxxxiv. 662.—90. SPENGLER. "Ein interessanter Fall von paroxysmaler Tachycardie," *Deutsche med. Wchnschr.*, Leipzig, 1887, xiii. 826.—91. STRUBELL, A. "Ueber funktionelle Diagnostik und Therapie der Herzkrankheiten," *Ibid.*, 1908, xxxiv. 1842.—92. TALAMON. "Épilepsie cardiaque et tachycardie paroxystique" (Case after fall on head; argues it epileptic), *Semaine méd.*, Paris, 1891, xi. 13.—93. TAYLOR, S. "The Relation of Functional Disorders of the Heart to Diseases of the Abdominal Viscera" (Critical article, with brief cases), *Practitioner*, London, 1891, xlvii. 18.—94. TRECHSEL. "La tachycardie paroxystique" (Case and criticism), *Rev. méd. de la Suisse Rom.*, Genève, 1893, xiii. 119.—95. TUCZEK. "Ueber Vaguslähmung" (Case), *Deutsch. Arch. f. klin. Med.*, Leipzig, 1878, xxi. 102.—96. WATSON, Sir T. "On a Case of unusually Rapid Action of the Heart" (Case, with necropsy), *Brit. Med. Journ.*, 1867, i. 752.—97. WEINSTEIN, H. "Gastroptosis a Causative Factor of Tachycardia," *New York Med. Journ.*, 1907, lxxxv. 119.—98. WEST, S. "Paroxysmal Hurry of the Heart" (Cases; argues for myocarditis), *Trans. Med. Soc.*, London, 1890, xiii. 318.—99. WILLIAMS, P. W. "Paroxysmal Tachycardia," *Bristol Med.-Chir. Journ.*, 1897, xv. 122.—100. WOOD, H. C. Quoted by Osler, *loc. cit.*, 836.

Retardation :—101. HOLBERTON, T. H. "Case of Slow Pulse with Fainting Fits," *Med.-Chir. Trans.*, London, 1841, xxiv. 76.—102. LÉPINE, R. "Pouls lent; épilepsie bulbaire," *Lyon méd.*, 1884, xlv. 347.—103. LÉPINE, R., et POROT. "Pouls lent; syncopes; rétrécissement du trou occipital," *Ibid.*, 1906, cvi. 231.

C. A.

DISEASES OF THE BLOOD-VESSELS AND LYMPHATICS

ARTERIAL DEGENERATIONS AND
DISEASES.

ANEURYSM.

PHLEBITIS.

THROMBOSIS.

EMBOLISM.

DISEASES OF THE LYMPHATIC
VESSELS.
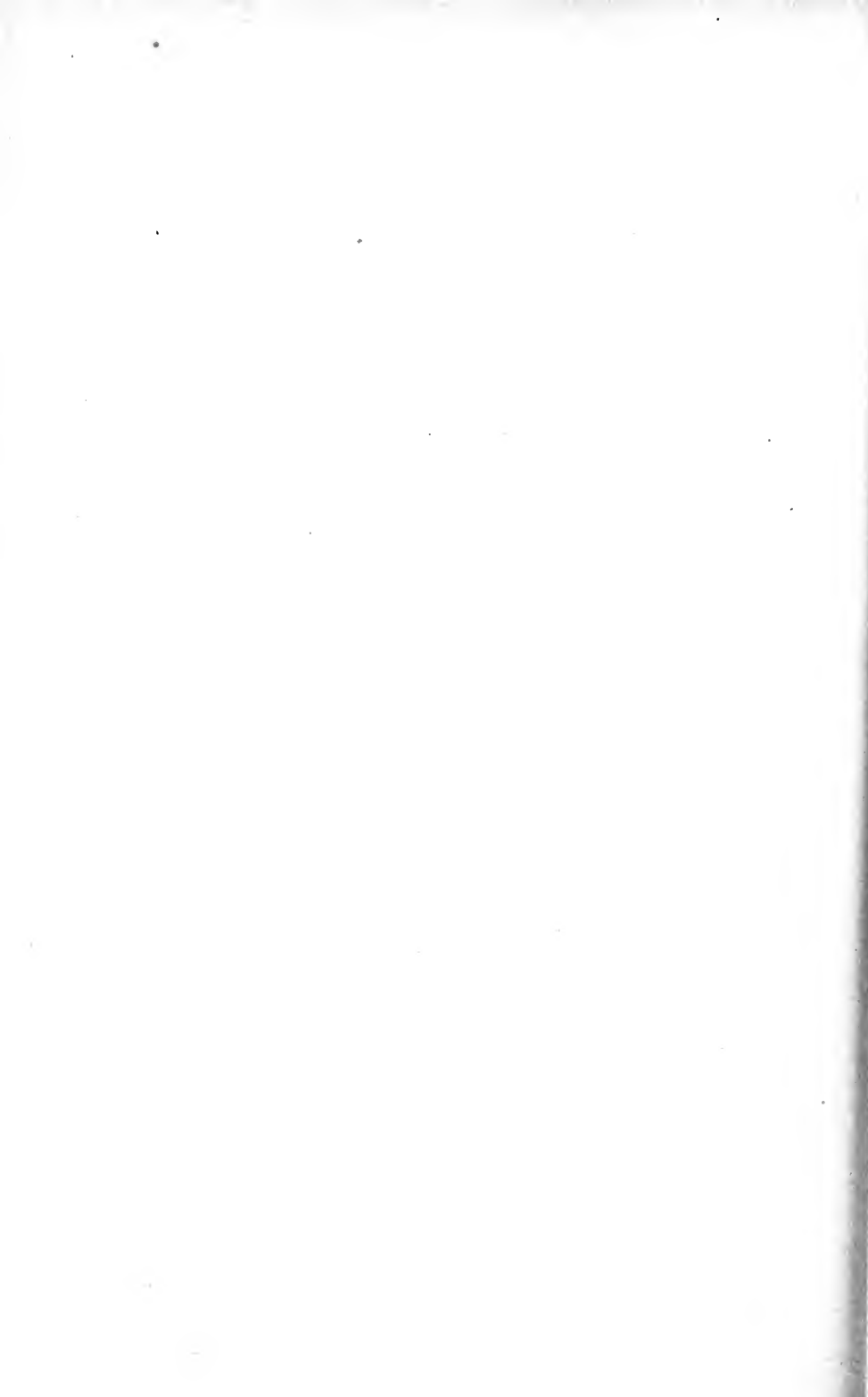
ARTERIAL DEGENERATIONS AND DISEASES

By F. W. MOTT, M.D., F.R.S., F.R.C.P.

Introduction.—The aphorism "A man is as old as his arteries" suggests more than its literal meaning; it implies that the majority of morbid processes known as degenerations are associated with diseases of the coats of these vessels, or are even directly due to them. In a large proportion of male adults over forty years of age death is due to cerebral haemorrhage, cerebral softening, cardiac degenerations, or aneurysm, and it is safe to assume that disease of the arteries plays a very important part in these results. The morbid process may be universal, or it may be local. It may affect primarily the large or the small arteries, and it may affect all the coats or one of them only. Among the most important determining causes of arterial disease are strain, syphilis, lead, alcohol, the gouty diathesis, and laborious occupations, to all of which males are more exposed; no wonder then that men are more frequently the subjects of arterial disease than women.

Certain arteries of the body are more prone to particular morbid processes than others. Gout and Bright's disease, for example, are associated with changes in the walls of the small arteries, and, since the morbid matter is eliminated by the kidney, this organ especially suffers. Although syphilis may attack any of the arteries, it has a predilection for the vessels at the base of the brain, whilst the effect of mechanical strain due to laborious occupations is felt more particularly by the aorta and large vessels.

Before passing to a full description of the various diseases of arteries it will be well to recall a few of the more important points relating to their distribution and structure.

Distribution of Arteries.—The more active the function of an organ, the greater the arterial supply. In every part of the body there is some relation of a special and useful nature between the arterial distribution and the structures which it serves, as is seen, for example, in the circle of Willis, which is a provision against temporary or permanent suppression of the blood-supply to the important cerebral arteries which arise from it; in the renal arteries, which arise at right angles to the aorta and break up within the kidney in such a way as to favour high

blood-pressure in the glomeruli and velocity in the arteriae rectae ; and in the coronary arteries, which arise in the sinuses of Valsalva, but, instead of running into the substance of the heart, are contained in grooves surrounded by loose fat, so that, while their place of origin ensures the necessary high blood-pressure, their ready distension during systole is unimpeded.

Another important consideration relating to the site and effects of arterial disease is the *dichotomous mode of division*, which explains the frequency of disease at the points of bifurcation. *Tortuosity* is another important factor in localising disease from internal strain. In certain organs, such as the brain, lungs, kidney, and spleen, the arteries are terminal, or virtually so ; consequently in the defect of anastomosis with other branches, occlusion cuts off the area supplied by that branch from its blood, and the tissue undergoes necrosis.

General Structure of Arteries.—The arteries of the body may be roughly divided into large, middle-sized, and small ; they possess three coats—internal, middle, and external.

(i.) The *tunica intima*, or inner coat, consists, from within outwards, of three distinct layers :—

(*a*) An endothelial lining, in contact with the blood, made up of delicate nucleated cells joined together by a cement substance, and arranged like a mosaic pavement. This endothelium, slightly modified in different situations, lines the whole cardiovascular apparatus, and its integrity is of the greatest importance in preventing the coagulation of the blood in the living vessels.

(*b*) A subendothelial layer of branched connective-tissue corpuscles with intervening cement substance.

(*c*) A continuous layer made up of a felt-work of fine elastic fibres with small openings therein, the fenestrated membrane of Henle. This elastic lamina in the empty contracted artery has the appearance of a crinkled, bright yellow line.

(ii.) The *tunica media*, or middle coat, consists, in the large arteries, of alternate layers of elastic fibres and unstriped muscle-fibres arranged circularly. The larger the artery the more does the elastic element predominate, whereas in the arterioles the muscular coat is, relatively to the size of the vessel, much better developed, and elastic tissue is not present. If we consider the dynamics of the circulation this difference of structure is at once explained ; the large arteries by their elasticity help to convert the intermittent force of the heart into a continuous pressure ; the muscles of the small arteries, by a general tonic contraction under the control of the vaso-constrictor nerves, maintain the peripheral resistance to the outflow of blood, keeping the arterial system always over-full, whilst the elastic-buffer action of the large arteries, continually tending to overcome this resistance, causes a steady flow through the capillaries. (For further particulars, see p. 18.)

(iii.) The *tunica adventitia*, or external coat, consists of connective tissue possessing a large number of interspersed elastic fibres, together

with blood-vessels, lymphatics, and nerves. The blood-vessels—vasa vasorum—serve to nourish the walls of the vessels ; probably they enter the middle coat of the larger vessels ; at any rate, it is not disputed that the tunica media is nourished by the blood of the vasa vasorum, and it is highly probable that even the inner coat is nourished by the transudation of lymph from them. I have pointed out (22) that the openings of the fenestrated membrane probably serve the purpose of allowing nourishment to enter from this source. The lymphatics are numerous, and in certain situations, as in the central nervous system, they form distinct perivascular sheaths. Vasomotor nerves run by the side of the vasa vasorum (Fig. 59), and over many of the large vessels important plexuses

Fig. 59.—Photomicrograph. Section of small artery of great toe of healthy human subject shewing the thick media. The adventitia with vasa vasorum injected. Magnified 30 diameters.

of nerves exist ; for example, the carotid plexus of the sympathetic and the cardiac plexus round the aorta.

Local Variations in the Structure of Arteries.—A full account may be found in the work of Messrs. Ballance and Edmunds, who explain the variable thicknesses of the outer coat in different situations as an adaptation to resist the pressure of joints, and visceral and muscular movements ; as examples they cite the common femoral, superior mesenteric and facial, all of which have relatively thick outer coats.

Charcot says the arterioles of the encephalon present the same characters as those of the large arteries at the base, namely, abundance of muscular elements, a relative paucity of elastic fibres, and remarkable tenuity of the adventitia. He also points out the importance of the perivascular lymphatic sheath enveloping the small blood-vessels, especially the arterioles, and containing a transparent fluid.

Developmental Defects of Arteries.—A few examples will be cited under this heading: such as (*a*) defect of certain arteries with non-development of the corresponding organ ; (*b*) doubling of the aorta ; (*c*) obliteration of the aorta, which may occur at the junction of the ductus arteriosus, or at the isthmus of the aorta (junction of pulmonary artery with the aorta) ; (*d*) congenital smallness of the arterial system described by Virchow in 1870 as the aortic hypoplasia of chlorotic girls. In the last mentioned all three coats of the aorta are greatly thinned, and the calibre of the vessel is that in a child ; the internal coat presents a reticulated appearance, and scattered yellowish lines and patches indicating fatty change are often seen. Such a vascular system is sufficient until puberty ; but the additional requirements of the blood entailed by the development of the reproductive and other parts render it relatively insufficient. Virchow attributes the frequency of palpitation and cardiac hypertrophy in chlorotic women to these congenital arterial defects (Vol. V. p. 689). Beneke, in 1867, began a series of researches on cadavers of different ages ; he measured the volume of the organs and the circumferential measurement of the arteries ; his average results shew that the size of the arteries, contrary to what one observes in the case of the heart, increases in a regular ratio to the age. The heart is twelve times less voluminous in the infant than in the adult ; it increases regularly in size up to five years ; increases less up to the time of puberty, and then again rapidly increases in size.

The subjects of arterial hypoplasia are not always of the " infantile type." They are well developed, but are pale, as in chlorosis ; the two conditions peculiar to them are *imperfect development of the hair and of the reproductive organs.* The face, armpits, and pubes are smooth, and, in the male, the penis and testes are incompletely developed (*vide* art. " Infantilism," Vol. IV. Part I. p. 487).

Such patients in rare instances have been known to die with symptoms of asystole, yet without any lesion discoverable at the necropsy except arterial stenosis and cardiac dilatation. Both Beneke and Virchow have pointed out that subjects of this affection are prone to endocarditis ; occasionally albuminuria may exist, and cases have been recorded of associated nephritis. Lee Dickinson described two specimens of ruptured aneurysms associated with hypoplasia of arteries ; and, as he found no degeneration of the vessels, but extreme tenuity of their walls, he attributed the formation of the aneurysms to congenital delicacy of the vessels. Arterial hypoplasia is sometimes associated with haemophilia, and the haemorrhagic diathesis may be the explanation of the various extravasations of blood which are found in these cases, but cannot be traced to any aneurysm or ruptured vessel.

The subjects of arterial hypoplasia seldom attain an advanced age ; they generally succumb either to asystole, to nephritis, or to some infectious disease ; according to Beneke, especially to tuberculosis and typhoid fever.

Acute Arteritis may be of extravascular origin, or may result from

general infections or intoxications. The former may be due to the
extension of the inflammation from the surrounding tissues, or to external
injury of the vessel walls.

Local Acute Arteritis.—Infective inflammation in the neighbourhood of
an artery may set up a periarteritis and a subsequent endarteritis. The
coats of the vessel become the seat of an infective inflammation charac-
terised microscopically by cell-infiltration. Necrosis of the tissue and a
rupture of the vessel wall may ensue ; for example, caries of the petrous
portion of the temporal bone has been followed by perforation of the
carotid artery and death from haemorrhage. Secondary haemorrhage is

Fig. 60.—Photomicrograph. Experimental arteritis. Section of the carotid artery of a dog shewing
great thickening of the inner coat at one spot, causing a projection and considerable diminution
of the size of the lumen of the vessel. Careful examination shews bright lines running in the
media of the healthy part of the arterial wall ; these have completely disappeared in the swollen
inflamed part. The elastic laminae have been ruptured by the profuse inflammatory cell-proli-
feration. Just below this spot the coats of the artery gave way and an aneurysm was formed. The
acute arteritis was produced by touching the sheath of the artery with lunar caustic. Magnifica-
tion, 15 diameters.

frequently due to infective inflammation of a ligatured artery. In some
experiments, which I performed many years ago (not published), I
exposed the carotid artery in animals and produced an acute local
inflammation by touching the vessel on one side with a nitrate of silver
stick ; the result was an acute periarteritis at this spot with a corre-
sponding endarteritis and enormous thickening of the inner coat from
proliferation of the subendothelial tissue (*vide* Fig. 60). The proliferation
caused rupture of the elastic layer of the intima, and in one case
produced an acute aneurysm, probably due to secondary infection of the
wound.

A local infective arteritis may occur in infective endocarditis ; it is
not an uncommon cause of aneurysm, and, as first was pointed out by

Prof. Osler and Dr. Hughlings Jackson, is almost the invariable cause of cerebral haemorrhage in young people. I have examined several cases; and in one of acute aneurysm of the ulnar and posterior tibial arteries, the coats of the aneurysms were attacked by an acute infective inflammation. We can understand that a small particle dislodged from a calcified valve might by its rough edges easily damage the wall of the artery, and if it carried infection with it, or the blood itself contained infective organisms, would bring about the very condition necessary to produce an acute arteritis experimentally.

General Acute Arteritis.—This condition arises from general infections or intoxications; of special importance is the implication of the aorta, to which the term acute infective aortitis has been given.

Experimental Evidence.—Acute arteritis has been produced experimentally in animals by several observers.

Crocq *fils* came to the conclusion, after inoculation with cultures of *B. coli* and *Streptococcus pyogenes*, that an acute arteritis does not necessarily follow microbial infection; it depends rather upon the nature of the microbe, and on other unknown factors. The experiments of Gilbert and Lion, Thérèse, Boinet and Romary, have shewn that acute arterial lesions may be produced in animals by the inoculation of various pathogenetic micro-organisms and also of their toxins, both with and without previous injury to the internal coat. Among the organisms used were *B. typhosus, B. coli*, streptococci, and staphylococci, and the toxins of diphtheria, cholera, and of the staphylococcal groups. The lesions produced by Boinet and Romary are stated to have exhibited the microscopic characters of acute endarteritis with cellular infiltration around the vasa vasorum.

Etiology.—Acute infective arteritis may result from certain fevers, chief of which are enteric, typhus, and rheumatic fever, diphtheria, influenza, puerperal infection, pneumonia, small-pox, scarlet fever, gonorrhoea, and malaria. Prof. Osler says that recent endarteritis was present in 21 out of 52 cases of enteric fever in which the aorta was examined. Thayer considers arteritis a more frequent complication of enteric fever than is generally recognised, and the French authors particularly refer to this as a cause. According to Leyden, influenza is more likely to be followed by arteritis than is enteric fever; such, at any rate, was his experience during the influenza epidemic 1889-90.

The arteritis may appear during the height of the malady or as a complication during convalescence. Acute infective arteritis may also attack those who are the subjects of gout, subacute or chronic rheumatism, syphilis, or chronic lead or alcohol poisoning. In such cases it is due to a complex of factors in which a secondary or terminal microbial infection plays an important part. Sudden muscular strain as a primary agent in the production of acute aortitis must be of very rare occurrence, but a case has been described by Sir Clifford Allbutt in which sudden physical exertion seemed clinically to have been the primary factor. The disease may arise primarily in a vessel previously healthy, or it may be an

infective process occurring in vessels already the seat of a chronic endarteritis.

Distribution of the Lesions.—The lesions may be distributed generally throughout the arterial system, or may be localised to the great vessels and the aorta; the ascending and transverse portions of the arch are especially liable to suffer. Acute arteritis has a predilection for the limbs, especially for the lower limbs. Barié found that in 11 out of 13 cases the lower limbs were attacked, once the hand, once the face; the posterior tibial was especially liable to it—eight times in 11. Leyden and Guttmann found the same proportion in influenza; in 8 cases, the popliteal was affected five times, a femoral artery once, a brachial once, and once a cerebral artery.

Morbid Anatomy.—The appearance of the vessels differs according to the severity of the disease and the situation of the lesions. The most characteristic appearances are seen in the vessels of large calibre, such as the aorta, which may shew the following conditions : (1) A reddening may be confined to the intima or it may extend through all the coats ; this must be distinguished from the diffuse red staining which is found after death in the aorta and large vessels in infective diseases. In the former the intima will be found to have lost its smooth polish and may shew some roughness, whilst a thin film of fibrinous exudation may be spread over the surface. The tunics may be swollen, and microscopic examination will shew a cellular infiltration of the coats especially marked around the congested vasa vasorum. (2) Gelatinous plaques are the most characteristic feature of acute aortitis ; they are caused by thickenings and elevations of the intima. They vary from 1 millimetre to 1 or more centimetres in diameter. Their appearance varies according to the duration of the process. At first soft, succulent, pinkish-white in colour, they later become greyish-yellow and tough in consistence. These gelatinous plaques may be found scattered throughout a vessel already the seat of all grades of atheromatous degeneration. The patches on microscopic examination are seen to be due to an infiltration of the sub-endothelial tissue with embryonic round cells, spindle-cells, and stellate cells arranged in layers. They are always associated with a corresponding area of periarteritis and mesarteritis ; in fact these varying sized patches are due primarily to inflammatory changes in the vasa vasorum (*vide* Fig. 1, Plate II.). (3) Vegetative aortitis is usually found near the root of the aorta, and is due to an infective fibrinous deposit on a patch of aortitis or atheroma ; as in ulcerative endocarditis, it may be a source of infective embolism. (4) Suppurative aortitis is rare, and may be due, as Oettinger supposes, to pyogenetic infection of the vasa vasorum. Pus may then be found between the external or middle coats, and may burst inwards.

Charlewood Turner has described two cases of ulcerative aortitis—one mycotic, the other septicaemic—in which there were thrombi in the branches of the pulmonary arteries and septicaemia. This condition may be produced by an infective and calcareous aortic valve which, partially

broken off at its attachment and striking against the wall of the aorta with every systole, damages the endothelium, and leads to secondary infection of the vessel.

Symptoms.—Acute arteritis may come on during the progress of the primary disease or during convalescence. The first symptom is spontaneous localised pain in the limb, exaggerated by movement and pressure. When thrombosis occurs, a hard painful cord can be felt, and there is enfeeblement and abolition of the pulse, associated with numbness and tingling, followed by anaesthesia, coldness of the skin, and swelling generally unaccompanied by oedema. The local temperature is lowered and the process frequently ends in gangrene, the extent of which varies according to the seat and extent of obstruction (*vide* p. 561).

Acute aortitis is often difficult to recognise, owing to the absence of characteristic symptoms, and mild cases, especially at the commencement, are particularly liable to escape notice. Occurring during the course of an infectious disease, the symptoms produced by the implication of the aorta may easily be masked by the phenomena due to the primary malady, or by concomitant endocardial and myocardial disease. Pain, dyspnoea, vertigo, and a tendency to syncope, are the principal features, but they are by no means constant. Pain in the form of anginal attacks, may be the first symptom, and is of valuable diagnostic importance ; and in its absence, according to the late Sir W. Broadbent, a diagnosis of acute aortitis can rarely be made. Its intensity varies with the position and extent of the aortic lesion, being especially marked when the base of the aorta and the orifices of the coronary arteries are involved. It may vary in intensity from a sense of constriction and oppression beneath the sternum to pain of an alarming and agonising character which may radiate along the brachial plexus and intercostal nerves of the left side, proceeding down the shoulder and inner side of the arm, as far even as the extremity of the little finger. Syncope and sudden death may follow severe attacks. Dyspnoea has, at its commencement, according to Boinet, the character of a *dyspnée d'effort*, being aggravated by movement or by the assumption of the left lateral or dorsal position. In mild cases there may be merely a feeling of temporary respiratory oppression ; later this may become more prolonged and severe, with long and intense paroxysms. Spontaneously, or under the influence of fatigue or emotion, a condition resembling spasmodic asthma may be set up, characterised by sudden oppression, orthopnoea, and extreme respiratory embarrassment. Expectoration is absent or slight ; it brings no relief, and does not contain Curschmann's spirals. Adventitious sounds in the lungs are usually absent, or only slight in degree. Retrosternal pain and cardiac palpitation, when present, will help further to differentiate the condition from that of simple spasmodic asthma. Dizziness and vertigo of aortic origin may be the first symptom of aortic implication. According to Boinet they are more frequent when the aortitis is complicated with insufficiency of the aortic valves. They may occur spontaneously or more frequently when the patient

raises himself or lifts his head. Their causation has been attributed by some to sudden oscillations of the blood-pressure in cases of dilatation of the aortic arch; by others, to a reflex mechanism, which, originating from the aortic lesion, is capable of producing a spasm of the capillaries of the central nervous system and cerebral ischaemia. François-Frank states that he has produced similar effects by experimental irritation of the endarterium of the aorta.

Fever is not a constant feature, and, according to Léger, may be entirely absent throughout the whole course of the disease. Sir William Broadbent stated that the temperature is usually only raised as a result of the primary infection, of which the aortitis may be a complication. A rise of temperature occurring during the height of an infective fever, or during the convalescent period, if it cannot be explained by the detection of some other complication or irregularity, should arouse a suspicion of the possible onset of acute aortitis. An intermittent pulse occurring in the course of enteric fever, according to Landouzy and Siredey, may herald the advent of acute aortitis. Boinet states that aortitis may reveal itself during the course or convalescent stage of small-pox by a temperature oscillating between 100·5° and 101·5° F. Aortitis of rheumatic origin may commence with a similar oscillation of temperature.

Physical signs in acute aortitis are often absent or are very indefinite. They are in most cases only obtained in chronic disease of that vessel. The sudden onset of aortic dilatation is a most important diagnostic feature, especially in the case of young adults who are not the subjects of chronic arterial disease. Its discovery is the more valuable if care has been taken at the commencement of the disease to map out the area of aortic dulness. Throbbing of the vessels in the neck and visible pulsation in the suprasternal notch may afford the first indication, but may be due to many conditions other than a dilated arch. The pulsation of the right subclavian may be rendered visible or palpable by depressing the right shoulder. Pressure symptoms, such as spasmodic closure of the glottis and some distension of the branches of the superior vena cava, may result from dilatation of the arch. A skiagram may assist in the diagnosis, but the most important means is percussion of the area of aortic dulness. Potain's sign, which applies to dilatation, whether temporary, as in acute aortitis, or permanent, as in chronic cases, is thus described by Sir Clifford Allbutt:—"According to the degree of dilatation, a dull area may be delineated occupying an area including the manubrium sterni, and extending thence towards the second space and third cartilage on the right. As the upper part of this dull area extends from the base of the breast bone in a segment of a circle to the right, it has been likened to the crest of a fireman's helmet."

The absence of the aortic reflex is regarded by Cherchevsky and Rondot as a valuable sign of the onset of acute aortitis. According to these observers, if some twenty or more light percussion strokes be applied to the right second intercostal space near the sternal border in a

healthy subject, the limit of aortic dulness enlarges for 2 cm. on an average, and the arterial blood-pressure is raised two or three degrees; then the aortic enlargement disappears at the end of some minutes, and there is a retraction inside the normal limit of 0·5 cm., with an accompanying loss of pressure of one degree.   They state that the abolition of this reflex, with fixation of the vascular dulness at the right border of the sternum, is often the initial symptom of the acute process, and is of value in the diagnosis of the more chronic forms.

Auscultatory phenomena are uncertain and inconstant.

Systolic and diastolic murmurs have been described, but it is difficult to say how far these may be due to a dilated and diseased aorta, or to associated disease of the sigmoid valves.   Sir Clifford Allbutt draws attention to a physical sign which is occasionally present with aortitis; *i.e.* the friction-sound of a dry basic pericarditis.   The condition, he states, is very liable to be associated with angina.   The aortitis is usually of the subacute or chronic variety.   Moreover, its proneness to affect the base of the aorta and coronary arteries, renders the patient very liable to crises of dyspnoea and cardiac angina.   For further information the reader is referred to Sir Clifford Allbutt's *Cavendish Lecture*, 1903, and to Roger, Gouget, and Boinet's *Diseases of the Heart and Arteries*, 1907.

The *diagnosis* of the syphilitic origin of aortitis will be substantiated by evidence of previous syphilitic infection.   A valuable test for antecedent syphilis is afforded by the Wassermann and Neisser-Bruck serum reaction.   It is stated by some authorities that this reaction may prove of even greater diagnostic value than the agglutination test for typhoid, but it would appear that this statement applies especially to general paralysis and tabes.   Nevertheless, in a recent communication Bruck and Stern obtained a positive reaction in 48·2 per cent of primary syphilis, in 79·1 per cent of secondary syphilis, and 57·4 per cent of tertiary syphilis.   Consequently, although a negative reaction would not exclude antecedent syphilis, a positive reaction would undoubtedly help to confirm the syphilitic origin of the affection.

Results.—Acute infective arteritis, and especially acute infective aortitis, are diseases of very considerable gravity.   Yet, of the latter, Sir Clifford Allbutt says : "It is not always the perilous disease we are disposed to presume it to be."   It is true that certain of the cases may recover without any apparent damage to the internal economy, but it is very liable to be followed by very serious sequels and accidents, such as acute aneurysm, dissecting aneurysm (either of which may rupture and cause sudden death), and acute infective embolism without obvious valvular disease.   Again, death may occur from occlusion of the coronary arteries by gelatinous plaques situated at their orifices or in their further course.   Obliteration of the peripheral vessels may lead to gangrene, the extent of which will depend upon the vessel affected, the possibility of a collateral circulation being established, and the vitality of the tissues. Infective thrombo-arteritis may lead to a local aneurysmal dilatation. Lastly, acute arteritis is often the precursor of arterial sclerosis.

REFERENCES

1. ALLBUTT, Sir CLIFFORD. Cavendish Lecture, *Lancet*, 1903, ii. 139 ; and *Lancet* Editorial, 1903, ii. 243.—2. BALLANCE and EDMUNDS. *Ligation in Continuity*, 1891. —3. BARIÉ. "Contribution à l'histoire de l'artérite aiguë," *Rev. de méd.*, 1884, iv. 1 ; *Presse méd.*, 1905, iv. 124.—4. BOINET et ROMARY. *Arch. de méd. expér. et d'anat. path.*, Paris, 1897, ix. 902.—5. BROADBENT, Sir W. H. "Heart Disease and Aneurysm of the Aorta," 1906.—6. BROADBENT, W. "Acute Aortitis," *Lancet*, 1905, i. 1412.— 7. BROUARDEL. "Études sur variole : lésions vasculaires du cœur et de l'aorte," *Arch. gén. de méd.*, 1874, ii.—8. BRUCK und STERN. *Deutsche med. Wchnschr.*, 1908, xxxiv. 504. — 9. CHARCOT. *Senile Diseases*, p. 286. — 10. CORNIL et RANVIER. *Manuel d'Histologie pathologique*, 1907, tome iii. — 11. CROCQ, *fils.* "Contributions à l'étude expérimentale des artérites infectieuses," *Arch. de méd. expér.*, Paris, 1894, vi. —12. DICKINSON, W. L. "Aneurysms associated with Hypoplasia of Arteries," *Trans. Path. Soc.*, London, 1894, xlv. 52.—13. FORT, EDMUND. *Thèse de Paris*, 1901. —14. GILBERT et LION. *Compt. rend. Soc. biol.*, Paris, 1889, i. 583.—15. *Idem. Arch. de méd. expér. et d'anat. path.*, Paris, 1904, xvi. 73. —16. GUTTMANN und LEYDEN. *Die Influenza Epidemie*, 1889-90.—17. HUCHARD. *Maladies du cœur et de l'aorte*, tome ii.—18. LANDOUZY et SIREDEY. *Rev. de méd.*, Paris, 1885, v. 843 ; 1887, vii. 928.—19. LÉGER. *Étude sur l'aortite aiguë*, Paris, 1877.—20. LEGROUX. *Soc. méd. des hôp.*, 1884.—21. MARTIN. *Rev. de méd.*, 1881, i. 369.—22. MOTT. "Cardiovascular Nutrition, its Relation to Sudden Death," *Practitioner*, 1888, xli. 161.—23. OSLER. *Practice of Medicine*, 1905, 6th ed. 69.—24. POYNTON, F. J. *Lancet*, 1899, i. 1352. —25. RIST et RIBADEAU-DUMAS. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1905 ; *Lancet* Editorial, 1906, i.—26. ROGER, GOUGET, et BOINET. *Maladies des artères et de l'aorte*, Paris, 1907.—27. THAYER. "The Cardiac and Vascular Complications and Sequels of Typhoid Fever," *Johns Hopkins Hosp. Bull.*, Balt., 1904, xv. 323 ; also for references on this subject, *New York State Journ. Med.*, 1903, iii. 21.—28. THÉRÈSE. *Thèse de Paris*, 1891 ; and *Rev. de méd.*, Paris, 1893, xiii. 123, and 1898, xviii.—29. TURNER, F. C. *Trans. Path. Soc.*, London, 1886, xxxvii. 174.

Obliterative Arteritis.—This disease was first described by Friedländer, in 1876. It is often accompanied by neuritis, and, before complete obliteration, intermittent claudication may occur, associated with cyanosis and coldness of the extremities, thus giving rise to a condition resembling that of Raynaud's disease.

Etiology.—The disease is more frequent in men than in women ; out of Mr. Pearce Gould's 9 cases 7 were males and 2 females. It affects adults between thirty and sixty. The earliest date of onset in Mr. Pearce Gould's cases occurred at nineteen years of age. The causes are unknown. It has not been definitely associated with any particular diathesis, nor with any acquired disease, such as syphilis, alcoholism, malaria, albuminuria, or diabetes. I have, however, seen a case of symmetrical gangrene of the lower extremities in a middle-aged man suffering from alcoholic neuritis, the cause of the gangrene being arteritis and thrombosis. Syphilis has been suggested as the chief factor in some of the cases, and, according to Mr. Pearce Gould, is perhaps the most certain of all known causes. Influenza, alcoholism, erythromelalgia, cold, contusion, and previous thrombosis or phlebitis are regarded by him as more or less important factors in originating the disease. Vascular hypoplasia, especially of the arteries of the lower extremities, has also been suggested as one of the possible disposing factors. Dr. F. Parkes Weber has pointed out the

comparative frequency of its incidence among male Jews living in the East End of London, some of whom have been in the habit of smoking a large number of cigarettes daily. He suggests that unwholesome food and racial factors may play a part in the etiology. J. Israel has also drawn special attention to the occurrence of idiopathic gangrene in adult Russian Jews.

Thoma does not agree with Friedländer that there is a special form of obliterative endarteritis, and this opinion has the support of many observers at the present day ; neither does he support the statement of Billroth and von Winiwarter that gangrene in both old and young subjects
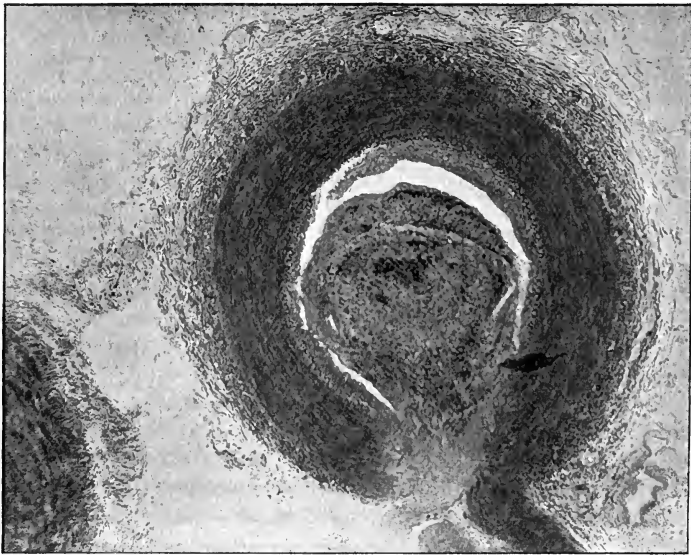
Fig. 61.—Photomicrograph. Section of anterior tibial artery in a case of obliterative endarteritis. This specimen shews the thickening of the inner coat and the existence of a thrombus which extends into a collateral. This is really an example of arteriosclerosis producing thrombosis and gangrene. The specimen was kindly given to me by Dr. Marinesco, whose case is referred to in the text. Magnification, 20 diameters.

is due to this condition. He considers that these authors have mistaken for it a thrombus transformed into connective tissue in an artery affected with arteriosclerosis, and certainly the photograph of the specimen (Fig. 61) supports this view. Hoegerstedt and Nemmser described three cases of constriction and closure of large arteries. The condition is rare. Syphilis in some of the cases appears to have been the principal etiological factor, in others arteriosclerosis with or without syphilis and strain. In these cases a number of large arteries of the trunk and limbs have been constricted, and even gradually occluded and converted into fibrous cords. It begins with a thickening of the arterial wall in the form of arteriosclerosis, or as a syphilitic endarteritis ; it terminates in thrombosis

and occlusion. The symptoms are various, according to the arteries affected and the rapidity of the process of occlusion ; if it be gradual there is time for the establishment of collateral circulation, and there is no functional defect.

Microscopical examination reveals thickening of the walls of the arteries, due to cellular proliferation of the endarterium and hypertrophy of the middle and external coats, development of vasa vasorum in the middle and external coats, and inflammatory thickening of the small vessels which may have led to complete occlusion. The obliteration of the lumen of the artery may be due to thrombosis or proliferative endarteritis. The coats of the veins may become inflamed, and these vessels may be blocked (*vide* Fig. 3, Plate II.). Dr. Marinesco, to whom I am indebted for the specimen of obliterative endarteritis shewn in Fig. 61, found in his case a degeneration of the muscles of the limb, whilst the nerves remained unaffected. Dr. Parkes Weber also mentions in one of his recorded cases the absence of transverse striation and the presence of marked fibrillation of the muscle-fibres without any visible changes in the nerve elements.

The latest contribution to the pathology of this condition is by Leo Buerger, who states that there are two prevailing views with respect to the nature of the obliterative process, namely : (1) that of von Winiwarter and Friedländer, who attribute the closure of the vessels to proliferation of the intima ; (2) that of Weiss and von Manteuffel, who believe that the extensive occlusion of the vessels in this disease is dependent upon a peculiar type of arteriosclerosis in which desquamation of the endothelium in the popliteal artery leads to the formation of parietal white thrombi and to occlusion of the arteries by direct peripheral extension of the primary focus. Buerger, as the result of a careful study of the vessels obtained from the amputated limbs of 19 cases, cannot agree with either of these views, but concludes that the condition is due to a thrombotic process in the arteries and veins followed by organisation and canalisation, and not to an obliterating endarteritis. He suggests that the names endarteritis obliterans and arteriosclerotic gangrene should be abandoned in favour of obliterating thrombo-angiitis of the lower extremities.

Symptoms.—Like all anatomical modifications of the lumen of the arteries of the limbs which end gradually in occlusion, it may engender various premonitory symptoms long before it culminates in *gangrene*. These symptoms are pain in the limbs frequently occurring in crises, intermittent cyanosis, cramps, coldness, and numbness ; — conditions which, transitory at first, afterwards instal themselves permanently. Sooner or later the pulse is no longer felt in the course of the arteries, and the temperature of the part is lowered, indicating the approach of gangrene. Ecchymotic patches appear at one or several points of the extremity of the limb. Eschars arise, and the gangrene, some-times moist, sometimes dry, spreads with more or less rapidity. The lower limbs are affected more often than the upper, but it may begin

in the hands; the affection is frequently, but by no means necessarily, symmetrical. In the amputations that have been practised it has been noticed that the arteries do not bleed, and the wound heals with difficulty unless the amputation has been high above the seat of mortification.

REFERENCES

1. ARKWRIGHT, J. A. *Lancet*, London, 1902, ii. 737 and 753.—2. BOILEAU. "Sur le rétrécissement généralisé des artères," *Thèse de Paris*, 1887.—2a. BRAMWELL, B. *Lancet*, London, 1908, ii. 229.—2b. BUERGER. "Thrombo-angiitis obliterans," *Am. Journ. Med. Sc.*, Phila., 1908, cxxxvi. 548.—3. BUROW. *Berlin. klin. Wchnschr.*, 1885, xxii. 507.—4. DUTIL et LAMY. *Arch. de méd. exp.*, 1893, v. 102.—5. FRIEDLÄNDER. "Arteritis obliterans," *Cent. f. med. Wiss.*, 1876.—6. GOULD, PEARCE. *Trans. Clin. Soc.*, London, 1884, xvii. 95, and 1891, xxiv. 134; also Lettsomian Lectures, *Lancet*, 1902, i. 717.—7. HEIDENREICH. *Semaine méd.*, 1892.—8. HOEGERSTEDT und NEMMSER. "Ueber die krankhafte Verengerung und Verschliessung vom Aortenbogen ausgehender grossen Arterien," *Ztschr. f. klin. Med.*, 1896.—9. ISRAEL. *Deutsche med. Wchnschr*, 1904, xxx. 1828.—10. JOFFROY et ACHARD. *Arch. de méd. exp.*, 1889, i. 229.—11. MARINESCO. "Sur l'angiomyopathie," *Semaine méd.*, 1896.—12. MICHELS and PARKES WEBER. *Brit. Med. Journ.*, 1903, i. 566, and *Trans. Path. Soc.*, London, 1905, lvi. 223.—13. RIEDEL. *Centralbl. f. Chir.*, 1888, 554.—14. ROUTIER. *Bull. Soc. de chir.*, Paris, 1887.—15. THOMA. *Textbook of General Pathology*, vol. i. Translated by Alex. Bruce, 1896.—16. WEBER, PARKES. *Lancet*, 1908, i. 152 (for extensive bibliography).—17. WIDERMANN. *Beitr. f. klin. Chir.*, 1892, xi.—18. WILL. *Berlin. klin. Wchnschr.*, 1886, 268.—19. WINIWARTER, VON. *Arch. f. klin. Chir.*, 1879, xxiii. 202.—20. ZOEGE-MANTEUFFEL. *Deutsch. Ztschr. f. Chir.*, 1898, xlvii. 461.

Syphilitic Arteritis.—*History and Introduction.*—The discovery of the pathology and symptomatology of this affection has been one of the most important advances in modern medicine; many grave nervous diseases, which were formerly not even diagnosed, are now curable, or amenable to treatment.

At the end of the seventeenth century both Lancisi and Albertini recognised syphilis as a cause of aneurysm; and Morgagni, in his remarkable work *De Sedibus Morborum*, describes a necropsy upon a syphilitic patient thus:—" Cor laxum. In una ex arteriae magnae valvulis Arantii corpusculum multo majus quam aequum esset. Sub eoque in ea facie qua valvula valvulas spectabat, membranae laminae ex quibus illa fiebat, ad modicum tractum ita sejunctae, ut quia hiabant specillum immittere inter utramque potuerim. Ipse autem proximus arteriae truncus albidis intus maculis passim distinctus, nec satis laevis, imo nonnihil inaequalis. Mox autem ad curvaturam *in Aneurysma distentus.*" In another place he states : " Quod saepe observavi in aliis cadaveribus, eorum praesertim, qui Syphilide laborarunt et ad *Aneurysma Aortae* vel ad pectoris hydropem sunt dispositi." Again in the same work he remarks, in describing the necropsy of a syphilitic patient : " Sed in tenui meninge *arteriarum trunci omnes*—omnesque item earum rami, iique praesertim qui plexum choroidem versus contendunt, multo erant crassiores aequo et duriores ; exsiccatique osseam pluribus in locis naturam ostenderunt. Quinetiam duram meningem idem ferme in ejus arteriis quae crassiusculae ipsae quoque factae erant conspectum est." Farther on

he describes swellings on all the large arteries and their branches, namely, the carotids, subclavians, and even the coronary arteries of the heart.

The investigation of arterial disease in syphilis was allowed to slumber, mainly owing to the teaching of John Hunter, until Dittrich, in 1849, described a case of inflammation and blocking of the right internal carotid and middle cerebral. Ziemssen considers the Danish physician Steenberg to be the discoverer of the connexion of this form of arteritis with syphilis, although before the appearance of his work, in 1860, isolated observations of disease in the larger arteries of the brain had been published by Virchow, Bristowe, and others. Sir S. Wilks was the first author in this country to call attention to syphilitic disease of the arteries, in 1863. He ascribed to syphilis certain nodules found on the cerebral arteries, and also the constricted lumen of these vessels, in a woman aged thirty-eight, who, five years previously, had been infected with syphilis, and who died from an apoplectic seizure. In 1868 Sir Clifford Allbutt made a microscopical examination of the vessels, and described *for the first time* the histological changes in a case of "Cerebral Disease in a Syphilitic Patient" in the following words :—" Both the long and cross sections shewed great inequality in the thickness of the walls and of the several coats. This change was due to a chronic arteritis with great nuclear and cellular proliferation, and affecting all the coats to some extent, but especially the middle and inner coats. The distinction between the coats was in many places lost." Then again he noted that there was no atheroma. He found also the most minute arteries affected. This important observation did not attract the attention that it deserved, and it was not until 1874 that Heubner published his work upon the microscopical appearances of syphilitic arteritis affecting the cerebral vessels, and this work, so practically important and interesting, may be said to have laid the foundation of our knowledge of the subject. About this time Dr. Julius Mickle published a number of cases of syphilitic arteritis. In 1877 Sir T. Barlow demonstrated a similar condition of the cerebral arteries in infants, the subjects of hereditary syphilis.

Although Heubner considered endarteritis invariably primary, yet it is now amply proved that in some cases the disease starts as a periarteritis ; and this condition was, according to Dr. A. Bruce, first described by Sir John Batty Tuke in 1874. In Sir Clifford Allbutt's case, however, there was undoubtedly periarteritis accompanying the endarteritis.

Although syphilis has a special predilection for the cerebral arteries, giving rise to characteristic clinical symptoms, yet other arteries are affected by endarteritis and even periarteritis ; but, with the exception of the aorta itself and the coronary arteries, the symptoms presented by arterial disease of the organs due to syphilis are not distinguishable from general syphilitic affection of the organ.

Brain syphilis is an affection of the arteries in one form or another. Even gummas start in the pia-arachnoid around the vessels, although

in many instances apparently situated within the brain substance. It was formerly taught that syphilitic arteritis is usually a late secondary or a tertiary symptom ; now most authors concur in believing that it may arise in the early stage of the secondary period, or at any subsequent time. It is now recognised that brain syphilis may and frequently does occur in the first year ; indeed the statistics of Hjellmann shew that it is most frequent in the first year, and that the numbers diminish with each successive year. Of 30 cases of brain syphilis which I have had under my care, with a sure history of the time of infection, one-half occurred within the first four years. Three cases occurred during the first year ; four during the second year ; five in the third year ; and three in the fourth. Pathologically, syphilitic disease of the arteries falls into three groups, but the groups may be associated : (*a*) obstruction of blood-supply of an organ ; (*b*) irritation ; (*c*) weakening of the arterial walls.

Causes.—A tendency to disease of the cerebral vessels may be heredi-tary : I have seen two brothers affected at the same time with syphilitic arteritis cerebri. Blows on the head often precede the onset of the disease. Probably, however, the most important cause is neglect of specific treatment. Nearly all authors agree that syphilitic arteritis is much more likely to occur in persons who have not been specifically treated. Toxic influences, such as chronic alcoholism and plumbism, may also be important factors, especially the former. Excesses " in Baccho et in Venere " are often followed by symptoms of arterial disease.

It seems probable also that those blood dyscrasias which raise arterial pressure would favour internal strain of these vessels, especially the aorta and coronary arteries, and render them more liable to disease. In the case of the aorta and coronary arteries physical exertion plays a most important part in the symptoms and complications that may arise in con-nexion with syphilitic disease—such as sudden rupture of the aorta, formation of false, dissecting, and true aneurysms. I recall very few cases of aneurysm of the aorta in men from whom I had not been able to obtain or detect a specific history. Maclean pointed out that in soldiers the most important cause of aneurysm is syphilis. Welch, in 1876, made a number of observations on soldiers, and shewed that in 34 cases 17 were undoubtedly syphilitic, and 8 were probably so. He found further, in necropsies on 56, the subjects of syphilis, that 60 per cent had disease of the aorta. Malmesten attributes 80 per cent of aortic aneurysms to syphilis. Likewise a large proportion of cases of aneurysm of the cerebral arteries, and of the large vessels of the body, are of syphilitic origin. A patient died at Claybury Asylum who was supposed to have been suffer-ing from general paralysis. At the necropsy I found an aneurysm on each internal carotid outside the skull the size of a large cobnut ; there were multiple aneurysms on the circle of Willis and the main branches. The patient had signs of syphilis on the body, and a history. He was only thirty-eight, but looked sixty years of age. Microscopic examina-tion shewed infiltration with lymphocytes and plasma-cells around the vessels of the middle coat in the wall of the aneurysms.

Pathology.—Syphilitic arteritis may affect, simultaneously or success-ively, a number of arteries of the body, and in some instances it gives rise to a general affection of the small arteries and arterioles (*vide* Peri-arteritis nodosa, p. 579). Again it may be limited to the aorta, or even to the coronary arteries, but by far the most frequent and important seat of the disease is the brain. The disease falls especially upon the arteries about the base, namely, the vertebrals and the basilar, together with the carotids—the vessels which enter into the formation of the circle of Willis and its branches. The arteries in the Sylvian fissure are very liable to the affection, and on several occasions I have found obliterative endarteritis of the opto-striate branches which enter the island of Reil and the anterior perforated space. Why it should affect these parts of the arterial circu-lation, and spare the vessels of the hemispheres, has yet received no adequate explanation. It may be that the basal vessels are surrounded with a large quantity of cerebrospinal fluid which possibly contains the syphilitic toxin. It is becoming generally accepted that the *Treponema pallidum* (*Spirochaeta pallida*) is the infective agent of syphilis ; but this organism has not been found in the cerebrospinal fluid, although occasionally successful inoculation of monkeys has been obtained by the use of cerebrospinal fluid. The organism may possibly exist in some modified form, to account for this, and for the great difficulty experienced in demonstrating its presence in syphilitic lesions excepting the primary sore, mucous tubercles, and congenital syphilitic tissues. Hoffmann states that Reuter and Schmorl have demonstrated spirochaetes between the fibrils of the proliferated intima in syphilitic aortitis. Moreover, Benda has proved the existence of typical spiral, straight, and granular forms of the *Treponema pallidum* in the external layers of the media.

Morbid Anatomy.—Scattered over the aorta are raised, oval, or roundish plaques which, in the early stages of their formation, have a greyish gelatiniform appearance, and are more or less soft. Later, as the cell-infiltration undergoes transformation into dense fibrous tissue, these plaques become tough and fibrous and of a pearly-white colour. As a rule, but not necessarily, they are free from atheromatous change. They occur in any part of the aorta, but a not infrequent situation is just above the sinus of Valsalva (*vide* Fig. 1, Plate I.). Arteritis affect-ing the cerebral or coronary vessels presents the appearance of small greyish-white opaque nodules or plates visible in the walls ; they are firm and of a stiff cartilaginous consistence. The lumen, in cross section, appears narrower, like a half moon ; but as the growth proceeds there may be circular constriction of the lumen so as almost to obliterate it (*vide* Fig. 62). In the universal syphilitic arteritis, which is often mis-taken for general paralysis of the insane, the small as well as the large arteries are affected ; the small arteries look like stiff, coarse threads of a dirty-white colour, and on section their walls appear to be greatly thickened, which accounts for their firmness when rolled between the fingers.

As in Fig. 62, the vessels are frequently the seat of thrombosis, and

eventually they may be changed into firm and solid cylinders; this affection of the intima may be the sole naked-eye appearance of disease; but it may be associated with gummas around the vessels, and gummatous meningitis; or, in the generalised form of syphilitic meningitis, there may be periarteritis and endarteritis affecting all the great and small arteries (*vide* Fig. 63). Hitherto I have been referring to the vessels of the brain; but I have seen cases of obliterating endarteritis affecting the

Fig. 62.—Photomicrograph. Syphilitic endarteritis and thrombotic occlusion of the opto-striate branches of the left middle cerebral, producing a defect of speech and right hemiplegia. There was softening of the island of Reil and of the internal capsule and basal ganglia. Patient, aged forty-seven, was certified as a case of general paralysis. Magnification, 50 diameters.

coronary arteries (*vide* Fig. 64) with or without associated aortitis. Little fibrous nodules, caused by a swelling of the intima, obliterated one or both arteries and produced fatty degeneration of the heart. Generally the orifice is so affected, but in one case—a young man who had had syphilis and was suffering with Bright's disease and lead poisoning—the right coronary one inch beyond the orifice was almost completely obliterated.

Out of 50 cases of cardiovascular disease, which I investigated in the wards and post-mortem room of Charing Cross Hospital, I found three cases

of syphilitic coronary stenosis. The appearance of the intima was very much the same as in the cerebral vessels ; little fibrous nodular swellings encroached upon the lumen, and in one case to such an extent was the vessel blocked that a large bristle could not be inserted (*vide* Fig. 65). I have also seen most extensive proliferation of the intima in the renal vessels of a syphilitic subject.
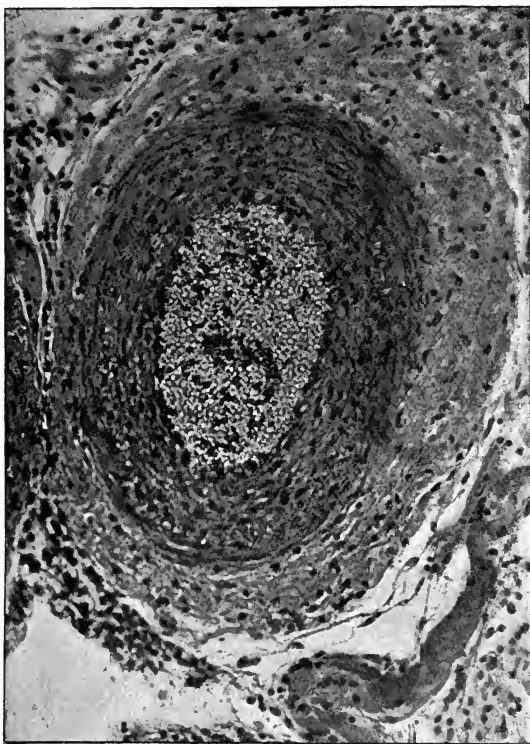
Fig. 63. — Photomicrograph. Cerebrospinal syphilitic meningitis and periarteritis. The outer and middle coats are infiltrated with leucocytes. The blood in the vessel contains a great excess of leucocytes. All the arteries of the brain and cord were affected ; in some situations there was extensive endarteritis, and the whole of the left fossa Sylvii was filled up with a gummatous mass. The case was diagnosed as one of general paralysis of the insane. Magnification, 200 diameters.

It may be asserted, as a general rule, that syphilitic endarteritis is a distinct process from atheroma. Heubner asserts that the new cell-formation of the inner coat always goes on to fibrosis and never undergoes caseation or calcification. Huber argues against this too restricted doctrine of Heubner, and cites a case of a prostitute, infected six months before death, at whose necropsy extensive endarteritis was found in the aorta, and many of the patches were undergoing caseation and calcification. The observation of Admannson, confirmed by Birch-Hirschfeld

FIG. 64.—Photomicrograph. Endarteritis obliterans of the right coronary one inch from its origin, probably syphilitic, in a man aged twenty - nine the subject of chronic Bright's disease and lead poisoning. The patient died from cardiac failure. A large thrombus was found in the left ventricle adherent to the apex, also an aneurysm of the abdominal aorta. There was marked fatty change in the muscular fibres of the right ventricle. Magnification 20 diameters.
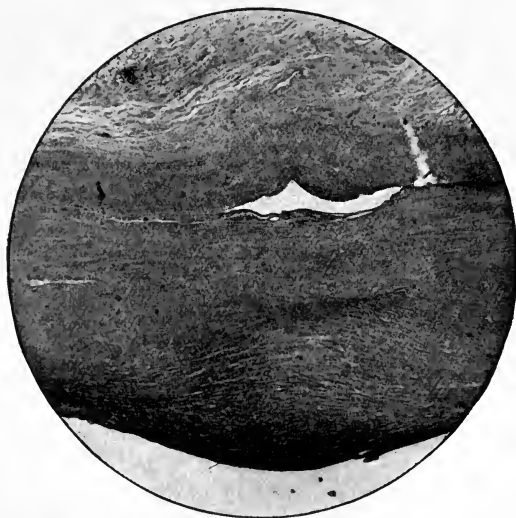
FIG. 65.—Photomicrograph. Section of a nodular endarteritis of the left coronary artery. There is no tendency to caseation or calcification. The patient was aged thirty-eight. There was atheroma of the aorta, but little or no affection of the aortic valves. The patient died with obscure symptoms of extreme fatty change of the heart-muscle, occasioned by almost complete obliteration of both coronary arteries. Magnification, 75 diameters.

PLATE I

(1) The aorta, from a case with a well-marked history and signs of syphilis on the body, shewing a raised pearly fibrous nodular plaque just above the aortic valves, leading to some puckering of the two cusps where they join one another, and interfering with the competency of the valve. Both coronaries are patent at their orifices, but there was some fibrotic thickening of the intima in patches along their course, not, however, leading to occlusion.

(2) Pearly fibrosis around the orifices of the intercostal arteries in a case of arteriosclerosis, with well-marked signs and history of syphilis; below is a calcareous plaque.

(3) A pearly fibrous plaque in the aorta, from the same case as (2). Microscopically it was seen to be caused by dense fibrous proliferation of the intima.

PLATE I.

Fig.1.
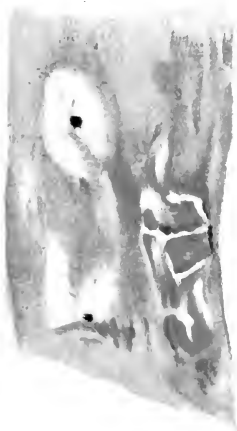
Fig. 2.

Fig. 3.

PLATE II

Fig. 3.

Fig. 2.

Fig.

PLATE II

(1) A section through a plaque from a case of acute syphilitic aortitis in which sudden death occurred from the rupture of a dissecting aneurysm. In the adventitia two vessels are seen infiltrated with lymphocytes and fibroblasts. One is seen with a branch passing right up into the media, but it has given way, causing a haemorrhage into the middle and inner coats. There is a marked mesarteritis, and the plaque is closely related to the inflammatory changes around the *vasa vasorum*. × 40.

(2) Obliterative arteriosclerosis of the posterior tibial with the two *venae comites*, from a case of symmetrical gangrene of the feet in a woman suffering with general arteriosclerosis. The nose also in this case became blue, nearly to the root, before death. The thick-walled artery is seen in the middle, contracted on a small thrombus. There is a homogeneous hyaline appearance of the wall. The veins are filled with blood-clot. × 6.

(3) A section through the wall of one of these veins shewing haemorrhages into the deeper layers of the media, and also into the adventitia. The muscle-fibres are swollen and indistinct, and the nuclei imperfectly stained. × 150.

and others, upon the atheromatous changes in the umbilical vessels and in the arteries of fetuses, stillborn owing to syphilitic infection from the parents, supports this conclusion; but the rule is nevertheless as Heubner states it.

In several cases of young women with a syphilitic history who have died in the asylum at Claybury, I have found well-marked general endarteritis cerebri with equally well-marked atheroma of the aorta. Indeed I regard atheroma of the aorta in young people as strong presumptive evidence of syphilis.

Prof. L. Rogers has reported several cases of extensive atheroma and dilatation of the pulmonary arteries without marked valvular lesions or any marked disease of the systemic vessels as a not very rare cause of fatal dropsy in Bengal. He states that it produces extreme hypertrophy and dilatation of the right cavities which may be so great that the right ventricle forms both the apex and the whole of the anterior surface of the organ, and in consequence these cases have been diagnosed clinically as due to left-sided valvular disease, with the exception of one case, which he recognised during life. They occur nearly always between the ages of twenty and forty years, and are almost certainly syphilitic in origin. The most remarkable feature is the preponderance of the affection among females. Palpitation, dyspnoea, and great dropsy due to tricuspid regurgitation are the principal symptoms, but these may subside for a time under appropriate treatment. Death may occur in a fairly early stage from severe strain such as child-birth, but many of the fatalities result from a terminal hydropericardium of insidious onset and accompanied by dilatation of the coronary veins of the heart.

Microscopical Appearances.—The inner coat is greatly thickened by proliferation of the subendothelial layer, so that the elastic lamina will be found separated from the lumen by a great development of newly-formed tissue consisting of spindle and stellate cells or, in a later stage, of fibrous material. The wall is usually affected unequally, being more thickened on one side than the other, and, when the vessel is cut transversely, the patch of disease is often crescentic in shape. There is very little tendency to degeneration; but as the lesion grows older, it becomes denser, firmer, and cicatricial. Not infrequently the vessel is blocked with a recent or organised thrombus. The elastic coat is sometimes ruptured, sometimes stretched so as to lose its crinkled appearance, and the muscular fibres of the middle coat have often undergone degeneration. The elastic lamina is frequently split, and, according to Heubner, a new elastic lamina may be formed; but this is denied by Cornil. In the case of the aorta and large vessels I have generally found an accompanying periarteritis and mesarteritis. The nutrient arteries of the large vessels are greatly thickened, and extensive inflammation is aroused, the vessels being surrounded with leucocytes (*vide* Figs. 66, 67, and 68). In the case of the aorta, the inflamed vessels often penetrate the inner coat, and the inflammation may be so intense that haemorrhages may occur into the middle and inner coats, as in the

case of the aorta (*vide* Fig. 1, Plate II.). With such intense inflammation it can easily be understood how sudden strains may cause a rupture or dilatation of the wall at the diseased spot, and the formation of an aneurysm. As I have already said, there is little tendency in the proliferated subendothelial tissue to undergo either caseation or calcification. This rule applies to the cerebral vessels, the most frequent seat of syphilitic disease ; but in the case of the aorta it is quite possible that degenerative changes may occur from blocking of the vasa vasorum. Many authors consider that syphilis is the most important cause of arteriosclerosis, and certainly there is no reason that arteriosclerosis, a degenerative process, should not be induced by the devitalising influence of syphilis. A syphilitic arteritis, if it does not lead to occlusion, may not be accompanied by any defined symptoms unless universal. Therefore, a widespread arteritis may proceed to a fibrosis or sclerosis, and later on in life give rise to symptoms. I have now examined many cases of cerebral softening occurring especially about the basal ganglia, giving rise to seizures, a progressive paralysis, mental enfeeblement, and emotional instability ; sometimes hemiplegia, or triplegia, indicative of multiple small coarse lesions affecting the internal capsule and basal ganglia. In the majority of these cases there is a history or evidence of syphilis. The symptoms commence between forty-five and fifty-five, and the cases are frequently diagnosed as general paralysis or senile melancholia with paralysis. The appearances presented by the small vessels of the brain, especially the terminal arteries of the basal ganglia, are those of arteriosclerosis. The thickening is mainly due to a fibrosis often limited to the inner coat, and quite consistent with a syphilitic origin.

Strümpell has recently called attention to the frequent association of tabes dorsalis with diseases of the heart and blood-vessels. He also points to the frequent association of arterial disease with general paralysis. His conclusions are : (1) It is not an uncommon event to find aortic insufficiency, sclerosis of the aorta, and aneurysms associated with rudimentary or developed tabetic phenomena ; (2) it is frequent to find these vascular changes associated with well-marked symptoms of tabes ; (3) these frequent combinations of disease indicate a similar causation ; they are sequels of a previous syphilitic infection.

Hereditary endarteritis syphilitica presents the same microscopical characters as that of acquired syphilis (Barlow, Chiari, Hawkins).

The relation of syphilis to the pathology of aneurysm of the aorta, large vessels, cerebral vessels, and heart will be considered elsewhere (pp. 597, 625).

Symptoms.—The symptoms of syphilitic arteritis may be divided into two categories : (A) obliterative and (B) ectasial arteritis.

A. Obliterative Arteritis.—The obliteration may be thrombotic or there may be occlusion, generally partial, owing to the lateral projection of the proliferated coat.

B. Ectasial arteritis may be divided into : (*a*) Simple aneurysmal dilatation with the phenomena of irritation or compression of adjacent

structures ; (*b*) aneurysmal dilatation with rupture, ending in cerebral haemorrhage.

FIG. 66.—Photomicrograph. Periarteritis nodosa. Mesarteritis and endarteritis of the aorta. The black nodules in the external coat consist of masses of leucocytes, so also the little black patches in the media. The patient, a man aged forty-seven, died from aortic valvular disease of long standing. Magnification, 10 diameters.
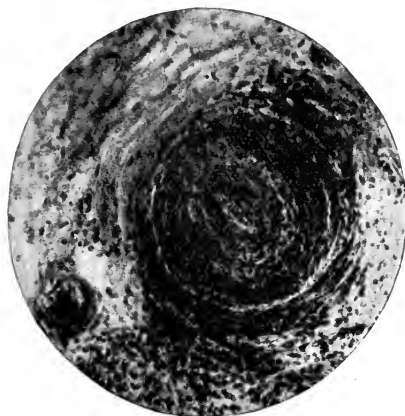
FIG. 67.—Photomicrograph. Endarteritis obliterans of the vasa vasorum of the aorta, from a case of acute syphilitic arteritis with rupture, formation of dissecting aneurysm, and death in a man aged thirty-one. Magnification, 150.

In the first subdivision there may be all possible varieties of hemiplegias, monoplegias, ocular palsies, and aphasia. With complete obliteration a clinical picture may be presented varying with the calibre

and seat of the artery. Whereas in partial obliteration the symptoms may be temporary and curable, complete obliteration must necessarily be permanent, and in a measure irreparable. The symptoms arising from the ensuing softening of brain substance depend entirely upon the size of the vessel blocked, the situation and area of brain substance supplied by the vessel, and, lastly, upon the possibility of collateral circulation.

Arteritis without accompanying gummatous meningitis, and without thrombotic occlusion, may occur; but it will be gathered from what has been said that a number of vessels of the circle of Willis must be affected before any pronounced symptoms arise; and, according to Heubner, only when two large adjacent branches of the circle of Willis are affected do circulatory disturbances in a hemisphere arise. The result of endarteritis syphilitica of the brain is a disturbance of the circulation of
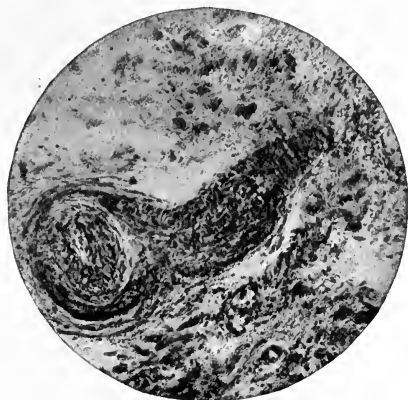
FIG. 68.—Photomicrograph of small nutrient artery of the aorta from a case of chronic Bright's disease, lead poisoning, and (probably) syphilis. Endarteritis obliterans. In the immediate neighbourhood there was nodular atheroma. Examination of sections from this and other cases leads me to believe that the nodular character of the disease in the aorta is due to changes in the vasa vasorum, such as are represented in this and the other photomicrograph. Magnification, 150 diameters.

the whole brain, and specially of the hemispheres, causing, according to the extent of the disease, slight or severe disturbance of the functions of this organ. The result may be psychical disturbances, which Heubner asserts were never absent in the cases observed by himself. These are slowness and difficulty in thinking, loss of decision, weakness of memory (amnesia), apathy, stupor, irritability, various anomalous moods, and sleeplessness. These and other allied phenomena can be ascribed to the defective circulation in the hemispheres. It can be understood that if one or more of the large arteries are in great part occluded, considerable variations of the blood-pressure in the hemisphere may result; and this would account for fainting fits and losses of consciousness like true apoplectic seizures.

The transitory character of the early phenomena of syphilitic arteritis (before thrombosis has occurred) is a very important feature. The narrowing of the lumen may be very well marked, but still some blood can

get through ; or, at any rate, there is time for a collateral circulation to be established, and this is the key to the peculiarities of the symptomatology. As Oppenheim in his valuable work points out, there may be a hemiparesis lasting perhaps a few minutes, a few hours, or a few days ; then it disappears, again to return, and eventually ends in permanent hemiplegia. It may begin with a transitory monoplegia, which gradually extends and affects arm, leg, or face ; thus becoming hemiplegic in character, and often associated with aphasia, especially when the hemiplegia is on the right side. Triplegia may occur and is especially characteristic of syphilis. This is brought about by a combination of a transverse focal myelitis and cerebral softening in one hemisphere from arterial disease. Charcot pointed out that one of the most characteristic signs of syphilitic arteritis is aphasia. The transitory trouble in speech may occur several times in the day. There is in other cases temporary word-blindness, disordered vision, vertigo, word-deafness, and all forms of speech defects, amnesia, alexia, etc. We can easily understand how these losses of function arise. There is a temporary disturbance of the circulation in the various parts of the brain which are concerned with these various functions ; but collateral circulation through other vessels supplies the necessary nutrition, and with it the return of function comes back. The experiments of Dr. Leonard Hill throw considerable light upon this subject. He has ligatured both carotid and both vertebral arteries in the dog ; the animal is then for some days demented, paretic, and suffers (probably) with psychical blindness. At the end of a week it recovers. He has found the anterior spinal arteries dilated to the size of vertebrals, and these restored the circulation to the hemispheres. The neurons had not died from the temporary cutting off of the blood-supply, but they were incapable of exercising their functions. I have examined the brains of these animals microscopically, and I find the cells are for the most part quite normal in structure. If, however, a monkey or cat be used for this experiment, a sufficient collateral circulation is not established soon enough, and the animal usually dies, frequently with convulsions, at the end of twenty-four hours. The cells of the brain in these latter animals exhibit most marked degenerative changes, indicating that recovery would be impossible, and that permanent loss of function must have occurred. The gradual obliteration caused by syphilitic endarteritis allows of gradual re-establishment of the circulation by collateral branches, and the brain substance is not, therefore, completely deprived of nutrition. The disturbance of circulation is temporary, and the loss of function is also temporary ; but, if the disease be not treated, the vessels which allowed the re-establishment of the circulation become similarly blocked, and then softening of the area of brain supplied by the diseased vessels ensues, and the loss of function is permanent. It is, therefore, of the greatest importance to recognise the disease in the earliest stage—that of the temporary loss of brain function, for it is often very amenable to treatment. Mercurial inunction or injections combined with large doses of iodide of potassium will save a man from softening of the brain, incurable

paralysis, and mental failure.   Probably the therapeutic action is not so much the opening up of the old obliterated or partially obliterated vessels as the prevention of thrombosis and of the extension of the disease to vessels either little affected or not at all, vessels which are therefore able to maintain the circulation.

Sometimes the patient can describe exactly the onset of the paralysis or aphasia ; but usually at the time there were giddiness and dulness of perception, or somnolence, symptoms which sometimes persist even after the paralysis has passed off.

It is much more common to find loss of motor power than loss of sensibility ; but various sensory phenomena may occur, such as pain, paraesthesia, hemianaesthesia, hemianopsia.   Frequently there is paralysis of cranial nerves, generally associated with hemiplegia.   I have recorded a case of complete paralysis of the fifth nerve with enophthalmos and hemiplegia, all on the right side.   The most frequent paralysis is some form of *ocular paralysis*, which may be due to a lesion of the nerve or the nucleus.   I have recorded a case of paralysis of both third nerves with marked paresis in both lower extremities, and to a less degree in the upper ; complete recovery occurred under treatment (*vide* " Disease of Cranial Nerves " in Vol. VIII).

Oppenheim points out that dysarthria is not infrequently met with in association with difficulty of swallowing and bulbar symptoms indicating disease of the basilar or vertebrals.   Alternate hemiplegia is another symptom of basilar disease.   I have met with various difficulties of speech : namely, a curious drawling staccato speech without any distinct paralysis of the muscles of articulation, due to patchy softening of the hemispheres of a widespread nature.

Sir J. Hutchinson relates a case of Anderson's in which, for two years before the onset of severe paralytic symptoms, from occlusion of the basilar, prodromes occurred in the form of headache and psychical disturbances. A case recently came under my notice of a congenitally defective woman, aged fifty-one, who was admitted to the Asylum suffering from auditory hallucinations and delusions, but with no paralytic symptoms.   Quite suddenly she was seized with faintness and severe vomiting.   She then became unconscious, Cheyne-Stokes breathing developed, and she died $1\frac{1}{2}$ hours after the onset of symptoms.   Old syphilitic aortitis was found and *old endarteritis of the basilar* with an *organising thrombus which was canalised in the centre*.   The disease is apt to be mistaken for general paralysis of the insane, as I have many times seen.   Both diseases are insidious in origin and progressive in character.   In general paralysis, however, the speech defects are not transitory, the tremor is finer, coarse paralyses are very rare, Argyll-Robertson pupils are very common, Babinski's sign is very seldom met with and clonus is rare.   The mental symptoms are usually more marked and more characteristic, and the disturbance of consciousness and mental symptoms generally are much less given to ebb and flow than in syphilitic arteritis (pseudo-general paralysis).   In both diseases probably abundance of lymphocytes would be found in the

cerebrospinal fluid. According to Plaut, the syphilitic antitoxins would more likely be present, as tested by the Wassermann method in general paralysis, than in syphilitic brain disease. He obtained the reaction 94 times out of 95 cases of the former disease. Inasmuch as specific treatment is, if not positively harmful, of no use in general paralysis, but is highly beneficial in syphilitic arteritis, the diagnosis is of the greatest importance. Early treatment decides the prognosis in many cases, and a complete recovery occurs in some. But, as a rule, even in the most favourable circumstances, the patient is not quite the same as before. There is often slight loss of expression in the face, a little slowness and hesitancy in speech, a loss of memory, an inability to undergo mental fatigue, slight weakness of grasp, dragging of the leg or legs, a little spastic rigidity with exaggeration of knee-jerks and clonus. Frequently I have seen patients go on for years apparently well, and then a fresh attack has occurred.

Syphilitic arteritis affecting the coronary arteries may give rise to severe symptoms of cardiac degeneration (*vide* p. 125).

If, as in Figs. 64, 65, the arteries are almost completely blocked the heart will undergo acute fatty change from insufficient nutrition. I have seen this occur in several cases. In two of the cases, previously referred to, of coronary obstruction due to syphilis, there was absolutely no valvular lesion. The patients suffered with symptoms of cardiac dilatation ; the pulse was hardly to be felt at the wrist, though, when one hand was placed over the cardiac area, the impulse was forcible, and diffused over a considerable surface ; the other hand upon the pulse detected the fact that many of the beats were not forcible enough to produce a pulse-wave in the radial artery. Such cases are generally rapidly fatal ; the patients first complain of breathlessness on exertion without obvious cause, of fainting feelings from cerebral anaemia, especially on assuming the erect posture, of giddiness and vertigo. These cases are of extreme importance from the point of view of life insurance, as men between twenty and forty, who have had syphilis but who have no signs of valvular disease, may yet exhibit symptoms of cardiac degeneration most difficult to account for. Often there is arrhythmia, and the pulse may sometimes be slow, sometimes quick. Anginal spasms frequently occur.

Syphilitic arteritis followed by thrombosis may affect the branches of the coronary arteries, and cause necrosis of patches of myocardium. The degenerated tissue may yield, and an aneurysm of the heart result ; but more commonly there is a gradual process of coagulation-necrosis of the muscle and fibrous substitution, thus accounting for many cases of the so-called fibroid heart of syphilitic origin.

It is probable that some cases of so-called Raynaud's disease—symmetrical gangrene—are due to syphilitic arteritis.

Congenital Syphilitic Arteritis.—The *symptomatology* has a close resemblance to that of the acquired disease of adults ; namely, convulsions,

headache increased in severity at nights, irritability, paralysis, and speech defects of various kinds. Sir T. Barlow states that the result has been sometimes softening, sometimes sclerosis of the brain ; more frequently the latter. If the Rolandic area or the pyramidal system has been affected in any part, descending degeneration of the crossed pyramidal tract occurs, with spastic rigidity of the limbs and contracture ; and he concludes his admirable account, based upon a number of cases, by contrasting the brain disease due to hereditary syphilis with that due to the acquired disease, as follows :—" We find that amentia, in association with eclampsia and spastic limbs, are to be regarded as typical of hereditary syphilis ; hemiplegia, with or without unilateral convulsions, as typical of acquired syphilis in the adult. The morbid anatomy of the former consists mainly of chronic meningitis and endarteritis, with cortical sclerosis and atrophy, whereas the common lesions in acquired syphilis are gummata and softening from arterial disease and thrombosis."

Congenital Syphilitic Aortitis. — " Wiesner has studied the arterial changes in ten undoubted cases of congenital syphilis and found constant characteristic lesions in the arteries. The aorta, with its larger branches and the pulmonary artery, were the most frequent sites of the pathological conditions which he found. In these vessels he distinguished a boundary zone between the media and adventitia, in which, as is also the case in the arterial lesions of acquired syphilis, the primary alterations in the tissues are to be looked for." A constant hyperaemia of the vasa vasorum, sometimes with thrombosis and haemorrhage, was found. In congenital syphilitic children several weeks old Wiesner found a perivascular fibrosis replacing the cellular infiltration around the vasa vasorum and, in some cases, causing obliteration. (Oscar Klotz.)

Periarteritis Nodosa. — In 1866 Kussmaul and Maier described a hitherto unknown disease which they observed in a tailor who was attacked with Bright's disease and rapidly progressive muscular atrophy.

The disease began with diarrhoea, shivers, sweating, and a feeling of numbness in the fingers. The patient was anaemic, but during the progress of the disease the temperature was generally normal, and the heart and the pulse not noticeably changed ; the urine was diminished, and contained blood, much albumin, and many epithelial cells and casts. Paralysis began in the index finger and some muscles of thumb, and spread later to the other muscles ; there was severe pain in the muscles, both spontaneous and on pressure ; parts of the skin were anaesthetic, while others were hyperaesthetic ; pains of a colicky nature occurred in the hypochondriac region ; there was sometimes constipation, sometimes diarrhoea. Four weeks after admission little nodules, the size of a split pea, were found beneath the skin of the abdomen and chest. At the necropsy little nodular swellings, varying in size from a poppy-seed to a pea, were found on the small arteries of all the muscles, except those of the face, and on most of the subcutaneous arteries. With the exception of the pulmonary, most of the arteries of the body were affected.

When examined microscopically, an acute inflammation of the media and adventitia was found.

Cases of a somewhat similar character have been described since. Three of these cases have been associated with multiple aneurysms. A résumé of the literature of cases of this disease up to 1907 is given by Dr. W. Carnegie Dickson.

Etiology.—The male sex appears to be more prone to the affection than the female. Longcope states that of 26 cases in which the sex was stated 21 were males and only 5 females. Though young persons may be affected, the disease seems to occur most frequently in adult life. Six cases were observed in persons under twenty years of age, 12 in adults between twenty and forty, and 8 in individuals between forty and sixty. The youngest case on record is that of a boy $2\frac{1}{2}$ years of age, recorded by Krzyszkowski, whilst the oldest is that of a man fifty-seven years of age reported by Benda. The cause of this disease is very obscure. Although everything points to some general infective agent, nothing has been discovered. The mode of onset of the disease, its course, the great wasting together with fever, and the presence of a leucocytosis in the few cases in which the blood has been examined, support this opinion. Weichselbaum considers this form of arteritis to be of syphilitic origin, von Kahlden does not. Benda is of the opinion that the lesions in peri-arteritis nodosa are essentially unlike any syphilitic process affecting blood-vessels. Schmorl and Benedict have suggested that the improvement of their cases under iodide of potassium is in favour of the syphilitic origin of the disease. A definite history of antecedent syphilis has been obtained in a few of the cases; in others it was considered that syphilis could be definitely excluded. The whole course of the disease from the beginning to the end usually takes about a few weeks or months. This is not like syphilis. Nor does the localisation suggest this infection, especially in regard to the comparative rarity with which the cerebral vessels are affected. No micro-organisms have been found in sections, and no culture preparations have been successful in substantiating the cause of the disease; moreover, the search for spirochaetes has so far been attended with negative results. From a study of the published cases of this disease I am of the opinion that they must be divided into two classes, distinguished by the presence or absence of syphilis, as this disease cannot be accepted at present as the only etiological agent.

Fig. 69.—Periarteritis nodosa. *a,* Node-like swellings. (Vessels taken from the mesentery of the small intestine: natural size.)

Morbid Anatomy. — Dr. Dickson in his valuable monograph is of the opinion that two distinct diseases have been described under the heading "Periarteritis Nodosa." These should be differentiated from one another, and classified under different names.

(a) *Periarteritis Nodosa.*—A true periarteritis, nodular in its distribution; the majority, if not all, of the cases of which are syphilitic in nature.

(b) *Polyarteritis Acuta Nodosa.*—Characterised by the formation of small localised nodules upon the smaller and medium-sized arteries.

According to Dr. Dickson the earliest discoverable changes consist in a proliferative inflammation of the adventitia in the form of leucocytic infiltration, cloudy swelling of the endothelial cells lining the vasa vasorum, and occasionally considerable fibrin-formation. The process rapidly spreads inwards causing destructive lesions in the muscular coat. "These are accompanied by local inflammatory changes, and are followed by the giving way of the internal elastic lamina and the other coats of the vessel-wall. Thrombosis of the contents of the lumen and of its aneurysmal dilatation is an almost constant accompaniment of the lesion, as are also proliferative changes in the outer and inner coats of the vessel. Secondary changes such as infarction, necrosis, haemorrhage, etc., may occur in the organ or tissues supplied by the affected artery."

Symptoms.—The onset of the disease is usually sudden, with fever and shivers, and its course is soon characterised by progressive marasmus and great anaemia. It is important to observe the absence of a relation between the enormously high pulse frequency and the relatively low temperature during the subsequent course of the disease. Not less characteristic are the violent pains which occur in the various parts of the body; they are especially frequent and violent in the hypochondria, and sometimes are limited to this situation. Cramps and tenderness in muscles are important symptoms, and may be the first to be noticed. They may be accompanied by weakness of the extremities, which may be transient or may progress almost to a stage of paralysis. Sensory changes which have been ascribed to disease of the peripheral nerves have been noted, such as anaesthesia, hyperaesthesia, and formication. Enteritis or nephritis generally accompanies the above phenomena; they are probably due to thrombotic occlusion of numerous small arteries, giving rise in the mucous membrane of the intestine to haemorrhagic infarctions which later lead to ulcers, and in the kidney to multiple ischaemic necroses. A part of the degenerative change of the renal epithelium may be due to the anaemia; the oedema, which is generally present, may be dependent upon nephritis, anaemia, or the changes in the arteries or to a combination of these changes. The nervous symptoms are probably due to changes in the arteries supplying the nervous structures of the spinal cord, the spinal ganglia, and the nerves. The appearance of subcutaneous nodules during life is a symptom of the utmost importance, but they are not a constant feature. Kussmaul and Maier, Müller and others have reported cases. Benedict and Schmorl have diagnosed the disease by the examination of an excised nodule. Longcope states that the presence of these nodules has been recorded in 7 cases. This writer also states that "A combination of symptoms which have been noted frequently, and which might reasonably lead one to suspect periarteritis

nodosa, are : pain, cramps or tenderness of the muscles, especially of the limbs, attacks of epigastric pain, albuminuria and haematuria, tachycardia, possibly dyspnoea or syncopal attacks, fever, leucocytosis, and the presence of subcutaneous nodules. Without the subcutaneous nodules the diagnosis during life is practically impossible."

A syphilitic nodular periarteritis of the central nervous system has been described by Baumgarten, Alex. Bruce, Gilbert and Lion, and Lamy. Really this disease is one of multiple gummas, with general cerebrospinal peri- and end-arteritis.

Dr. Alex. Bruce divides the cases into three groups :—(*a*) The outer coat is infiltrated more or less uniformly with round cells, but without any marked tendency to degeneration (*vide* Fig. 63). (*b*) The outer coat shews a nodular and diffuse cellular infiltration with commencing caseation. (*c*) The outer coat shews a distinct formation of caseous gummas as well as diffuse periarteritis.

I have observed all these conditions in the same case, and they represent successive stages in the formation of a gumma.

No case has yet been recorded in which a definite periarteritis has been seen more than four years after the primary infection. In fact, the earlier the occurrence of severe cerebral or spinal symptoms following infection the more likely is it to be due to this form of the disease. In one case which I examined the symptoms appeared within six months of the infection. Gilbert and Lion speak of the symptoms appearing even before the primary stage had passed away. The symptoms may be intense headache (worse at night), giddiness or pains in the head and neck, optic neuritis, vomiting, symptoms of mania, convulsions, unconsciousness, palsies of muscles supplied by cranial nerves, hemiplegia, monoplegia, paraplegia, and aphasia.

Tuberculous arteritis affects especially the medium-sized and small arteries and arterioles, leaving the large arteries free. The situations in which tuberculous affection is especially apt to occur are the cerebral arteries, the lobular branches of the pulmonary artery, and the renal arteries.

Specific cerebral arteritis is found in tuberculous meningitis. It starts in most instances in the perivascular lymphatic sheath, and the new formation proceeds from without inwards, invading the middle and inner coats successively. It is especially likely to occur in the middle cerebral and its branches. Damage of the endothelium leads to thrombosis and obliteration of the lumen already considerably constricted, and as a result there is extensive softening of the cerebral substance (*vide* Fig. 70).

Tuberculous lesions of the pulmonary artery belong to the history of excavation in the lungs, and the reader is referred to the article on "Pulmonary Tuberculosis," Vol. V. p. 326, for fuller information. The mechanism is of the same character in all cases ; but, according to its seat and distribution, it produces different morbid changes. Along with

the peribronchial affection, ending in caseation, there is invasion of the arterioles that accompany the terminal ramifications of the bronchi. In excavation all the tissues are destroyed except the artery and bronchus which are left exposed; the bronchus, surrounded by the caseous tuberculous material, ulcerates, the artery is at first irritated, and as a result there is proliferation of the inner coat. Thrombosis may occur, or the tuberculous process may invade the wall of the artery on the surface towards the cavity, and soften the coat; the blood-pressure in the vessel then causes dilatation with formation of an aneurysm.

In the tuberculous kidney, more especially in the miliary form of the affection, systematised bacillary lesions occur in the course of the radiating arteries. The microbial infection is embolic in origin. The
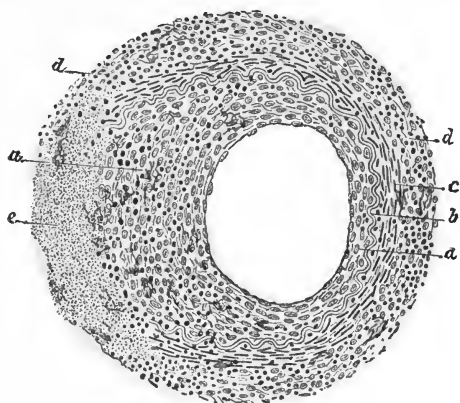
Fig. 70.—Tuberculous arteritis. *a*, Intima, *a*₁ proliferous intima infiltrated with cells and containing tubercle bacilli; *b*, inner elastic lamella; *c*, media; *d*, proliferous adventitia infiltrated with cells and containing tubercle bacilli; *e*, caseous portion of the vessel-wall. (Preparation stained with fuchsin and methylene blue, and mounted in Canada balsam: ×100, but the bacilli have been sketched under a higher magnifying power.)

consecutive infective thrombosis can be recognised by the naked eye, and the result is that tracts of caseous material appear along the course of the pyramids of Ferrein; even cones, like infarcts, may be found sometimes (Letulle). Tuberculous arteritis is usually produced by extension of a periarterial focus into the arterial wall; but it may occur primarily from embolic infection, as in the kidney.

To the naked eye the arteries present circumscribed thickenings, at first firm and grey, then friable and yellow. In pulmonary tuberculosis two forms are met with—(*a*) nodular aneurysmal, (*b*) obliterating.

Examined microscopically, the walls of the arteries are found infiltrated with round cells, islets of which are found in the adventitia around the vasa vasorum; giant cells may exist. As in syphilis, there is proliferation of the endothelial layer of the intima, and thickening of the same by a new formation consisting of spindle-shaped and stellate cells; the intensity of the inflammation may cause a rupture of the

elastic lamina and an incursion of leucocytes in great numbers. The rupture of the elastic lamina with the degeneration and death of the muscular fibres leads to weakening of the wall, and may result in the formation of an aneurysm. Again, as in syphilis, thrombosis or caseation of the granulation tissue may occur, and, if the vessel has not been blocked by a thrombus, it often ruptures and so gives rise to haemorrhage; should rupture not take place, bacillary infection of the blood-stream may occur.

A more favourable termination is fibrous hyperplasia of the adventitia with cicatrisation and occlusion.

Degeneration of the Arterial Walls. — *Fatty degeneration* is seen principally in the aorta just above the semilunar valves, where, in almost all adults, will be found opaque whitish spots or lines scarcely if at all elevated; it is not common, however, in the medium-sized arteries, and is, as a rule, of little clinical importance. Fatty degeneration, however, of the small vessels of the brain in general paralysis and in various blood diseases, such as pernicious anaemia, leukaemia, scurvy, or purpura, may occasion the symptoms of rupture and haemorrhage. In the large arteries the process begins with fatty degeneration of the endothelial cells of the intima; these become filled with fat-globules which stain black with osmic acid. The endothelium may become detached, and a shallow breach of the surface result; but, as a rule, it leaves the subjacent structures unchanged; yet sometimes there is an extension of the fatty degeneration to the subendothelial layers, and a proliferation of the fixed cells in the neighbourhood, which not infrequently take the products of fatty disintegration into their interior. The tunica media is sometimes the seat of fatty degeneration, the fat-granules are seen between the elastic fibres and laminae, and, when very profuse, the muscular fibres are no longer seen; a disappearance which is due to fatty degeneration of the muscle-fibres.

Calcareous degeneration is often associated with fatty changes in the media, especially in the arteries of the lower limbs; it may also be associated with hyaline degeneration or atheroma. Simple calcareous degeneration is a senile change found chiefly in the middle-sized arteries. It affects the muscular fibres of the middle coat, and appears as a band running partly around the vessel, which, at the same time, is dilated and loses its elasticity. If the change be very far advanced, the artery is converted into a tortuous rigid tube (gas-pipe artery). This form of degeneration is frequently followed by gangrene of the lower extremity owing to thrombotic occlusion (*vide* Fig. 78). The calcareous salts are deposited in small glistening granules, which coalesce to form compact plates. Sometimes these calcareous plates may present the appearances of true bone.

In the next section we have to consider a very important and widespread change in the arteries of which the senile condition forms one division.

REFERENCES

Syphilitic Arteritis : 1. ALLBUTT, Sir C. "Case of Cerebral Disease in a Syphilitic Patient," *St. George's Hosp. Rep.*, 1868, iii. 55.—2. BARLOW. *Trans. Path. Soc.*, 1877, xxviii. 287.—3. BRUCE, ALEX. "Syphilitic Nodose Periarteritis," *Medico-Chir. Soc. Trans.*, Edin., 1893, xiii. 190.—4. CHARRIER et KLIPPEL. *Rev. de méd.*, 1894, xiv. 771.—5. CORNIL et RANVIER. *Traité d'anatomie pathologique.*—6. DITTRICH. "Der syph. Krankheitsprocessen d. Leber," *Prager Viertelj.*, 1849, xxi. 21.—7. GOWERS. *Diseases of the Nervous System.*—8. HAWKINS, H. P. "General Arteritis in a Child, possibly the Result of Congenital Syphilis," *Trans. Path. Soc.*, London, 1893, xliii. 46. —9. HEUBNER. *Die luetische Erkrankungen d. Hirnarterien*, Leipzig, 1874.—10. HOFFMANN. "Die Ätiologie der Syphilis," 1907. (This contains a full account of the Etiology of Syphilis in relation to the recent developments since the discovery of the *Spirochaeta pallida* by Schaudinn.)—11. KLOTZ, OSCAR. "Congenital Syphilitic Aortitis," *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii. 11.—12. LANCEREAUX. *Dict. encyclopédique des sciences méd.*—13. LANG. *Vorlesungen über d. Pathologie und Therapie der Syphilis.*—14. MORGAGNI. *De Sedibus et Causis Morborum*, t. ii. 369.—15. *Idem. Ibid.* t. i. p. 297.—16. *Idem. Ibid.* t. i. p. 296.—17. MOTT, F. W. "Brain Syphilis in Hospital and Asylum Practice," *Archives of Neurology*, 1899, i.— 18. NONNE, MAX. "Syphilis und Nervensystem," 1902. (This work contains an admirable bibliography up to 1902.)—19. OPPENHEIM. *Die syphilitische Erkrankungen des Gehirns.*—19a. ROGERS, L. *Quart. Journ. Med.*, Oxford, 1909, ii. 1.— 20. STEENBERG. *Canstatt's Jahresb.*, 1861, 328.—21. STRÜMPELL. "Ueber die Vereinigung der Tabes dorsalis mit Erkrankungen der Herzens und der Gefässe," *Deutsche med. Wchnschr.*, 1907, xxxiii. 1931.—22. VIRCHOW. *Geschwulste*, Bd. ii. S. 444.—23. WILKS. *Guy's Hosp. Rep.*, 1865, 3rd ser., ix.—24. WUNDERLICH. *Syphilitic Diseases of the Brain and Spinal Cord.* German Clinical Lectures. Series ii. New Sydenham Soc.—25. VON ZIEMSSEN. *Syphilis of the Nervous System.* Clinical Lectures by German Authors, 1894. **Tuberculous Arteritis :** 26. CORNIL. "Tuberculose," *Journ. de l'anat.*, 1880, xvi. ; and *Pathol. Histology*, London, 1882.—27. GUARNIERI. (Tuberculous Meningitis), *Arch. p. le scienze med.*, 1884, vii. 233.—28. KIENER. (Tuberculosis of Serous Membranes), *Arch. de physiol.*, 1880, vii.—29. MARTIN. *Recherches sur le tubercule*, Paris, 1879.—30. MENETRIER. (Vascular Lesions in Phthisical Pulmonary Cavities), *Arch. de méd. exp.*, 1890, ii. 97.—31. NARSE. *Virchows Arch.*, 1886, cv.—32. WEIGERT. *Ibid.* 1882, lxxxviii. **Periarteritis Nodosa :** 33. BENDA. *Berlin. klin. Wchnschr.*, 1908, xlv.—33a. BOMBARD. *Virchows Arch.*, 1908, cxcii. 305.—34. DICKSON, CARNEGIE. "Polyarteritis Acuta Nodosa and Periarteritis Nodosa," *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii. 31 (for complete bibliography up to 1907).—35. EPPINGER. *Pathogenesis d. Aneurysmen*, Berlin, 1887 ; and *Arch. f. klin. Chir.*, Berlin, 1887, xxxv., suppl. 126.—36. FLETCHER, H. MORLEY. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1892, xi. 323.—37. KAHLDEN, VON. *Ibid.* 1894, xv. 581.—38. KUSSMAUL und MAIER. "A Peculiar Arterial Affection," *Deutsches Arch. f. klin. Med.*, 1866, i. 484.—39. LONGCOPE, W. T. *Bull. Ayer Clin. Lab. Pennsylvania Hosp.*, 1908, No. 5, p. 1.—40. MEYER, P. *Virchows Arch.*, 1878, lxxiv. 277.—41. WEICHSELBAUM und CHVOSTEK. *Allg. Wien. med. Ztg.*, 1877, xxii. 28.

ARTERIOSCLEROSIS

Introduction.—The name arteriosclerosis is applied rather loosely to any thickening and rigidity of the vessel-wall. The obvious naked-eye change in the large arteries, named by some authors atheroma, and by others endarteritis deformans, is included. Arterio-capillary fibrosis, a change first described by Gull and Sutton, in the walls of the small vessels, which only becomes obvious on microscopic examination, is regarded by some authors as the true arteriosclerosis. Thoma, who has studied the subject in the most systematic manner, considers it to be a change of the whole vascular system, which he proposes to name *angio-*

sclerosis. Atheroma or endarteritis deformans of the aorta may be associated with arterio-capillary fibrosis, and perhaps it would be better to adopt the term arterial sclerosis in accordance with the nomenclature of the Royal College of Physicians, implying thereby any form of arterial thickening.

Definition.—A local or general thickening of the arterial wall with loss of contractility and elasticity, occasioned by fibrous overgrowth mainly of the tunica intima, secondary and proportional to degeneration of the muscular and elastic elements of the media.

Etiology.—Old age consists in the set of conditions disposing to bodily decay. Of these arteriosclerosis is one of the most obvious manifestations; yet as the hair becomes grey, or falls out, at a comparatively early age, so the arteries are liable to degeneration in some people early in life, in others later (*vide* art. "Old Age," Vol. I. p. 182). Degeneration of the arteries is one of the most frequent of senile changes. All those causes which dispose to bodily decay and early ageing of the individual will tend towards arterial degeneration.

Although we do not yet know *how* arteriosclerosis is produced, clinical observation and experimental research have put us in a much better position to understand *why* it is produced, and therefore how best to avoid it; although, judging from its frequency, it seems to be steadily on the increase. Among the better educated of the public the word arteriosclerosis is well understood, and the serious complications arising in association therewith are fully appreciated, hence we should realise the necessity for caution in informing a patient that he is suffering from arteriosclerosis when the symptoms are but slight.

Certain diatheses favour this process. The chief of these are gout, rheumatism, and arthritis; but apart from these, there appears to be an hereditary tendency to arterial degeneration in some people. A number of diseases of infective nature tend to lower the vitality of the tissues of the body, including the arteries. I have already shewn that acute arteritis is apt to follow various infective diseases; that scarlet fever, by its damaging effects on the kidneys and resulting stress, may lead to permanent strain of the arterial system. Enteric fever also is a not infrequent cause of arteriosclerosis (Thayer). No disease is probably more productive of arterial degeneration than syphilis, and this in several ways: (i.) By causing endarteritis of the vasa vasorum, and defective nutrition of the walls of the large arteries: (ii.) By a syphilitic arteritis or aortitis, due to the action of the specific virus, generally terminating in either a general or a circumscribed patchy fibrosis of the small arteries and a nodular circumscribed fibrosis of the large arteries (*vide* Figs. 1, 2, 3, Plate I.). This may be regarded as a process of healing of a more or less acute pathological process: (iii.) By the devitalising influence of a toxin, long present in the body, producing anaemia and lowering the physiological margin of normal metabolism, so that, in case of injury or stress of a tissue, the equilibrium is not maintained and degeneration ensues.

Toxic Causes of Extrinsic Origin.—The most important of these are alcohol and lead. Dr. Dickinson has shewn that people engaged in the liquor traffic are, on the whole, more liable to arterial degeneration, and Sir Thomas Oliver has shewn that renal disease and arterial changes are induced by lead poisoning (see article "Lead Poisoning," Vol. II. Part I. p. 1060). It has long been known that lead may be an important factor in the production of gout, and it is probable that both lead and the toxic agents of gout cause defective metabolism. Chronic smoking and chewing, especially when associated with alcoholism and syphilis, are potent causes of arterial degeneration.

Dietetic Causes.—Excess of food and drink (high living) is especially injurious to the arterial system in people leading a sedentary life, and suffering from a gouty diathesis. The habitual ingestion of immoderate quantities of beer, which, by the state of plethora it determines and the gouty condition it favours, also a diet rich in meat and meat extractives contained in soups, gravies, etc., tend to the production of a continuous high pressure in the arteries. Vegetarians have pointed to the rarity of arteriosclerosis in the herbivora ; it has been rarely observed in very aged oxen and horses. Observers, however, have found that occasionally rabbits suffer with arteriosclerosis ; on the other hand, it is rarely met with in the dog.

At present we know very little about the injurious effects of leucomaines and ptomaines. It is probable that the former are produced in the body ; the latter may be absorbed from the alimentary canal, and it is conceivable that if they are not destroyed by the liver they are highly injurious. Altogether experience seems to shew that it is not only the quality and quantity of food contained in a diet which are of importance, but the individual constitution and the power of digestion and assimilation. Any food that was not digested would be liable to favour bacterial fermentation and putrefaction in the intestines ; toxins would be formed which neither the epithelial cells of the alimentary canal nor the liver could continuously intercept. Sexual excess and violent emotional disturbances may tend to arteriosclerosis by sudden hypertension of the arterial system, owing to a rapid rise of blood-pressure, especially when associated with a chronic intoxication. Moreover, prolonged psychical stress probably acts as a factor in the production of arterial degeneration by inducing chronic digestive disturbances and auto-intoxication.

Internal Secretions.—Arteriosclerosis is so often associated with chronic renal disease that it has been suggested that an excess of an internal secretion (renin), derived from the destruction of the kidney substance, is cast into the circulation, and that this excessively stimulates the neuromuscular mechanism of the blood-circulatory system and leads to continuous high blood-pressure. In discussing the experimental pathogenesis of arteriosclerosis, the importance of the suprarenal gland will be dealt with, together with the possibility that the well-known physiological effects of the internal secretion of this gland are responsible for the causation of high pressure (*vide* p. 602 *et seq.*).

PLATE III.

Fig. 1.

Fig.
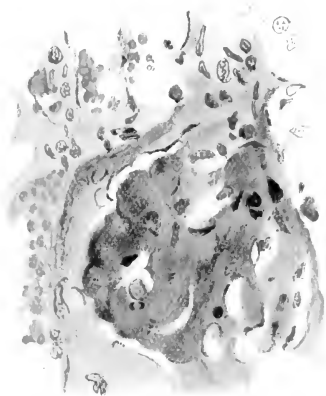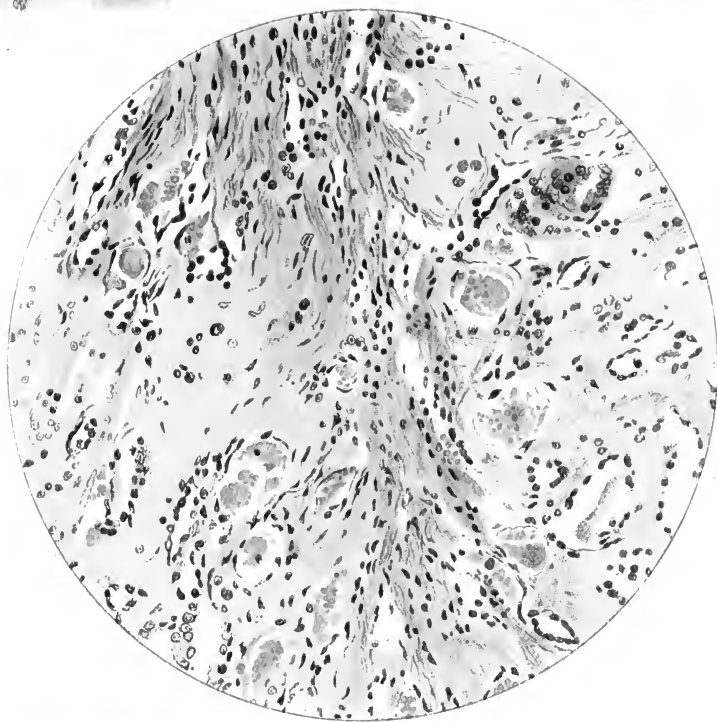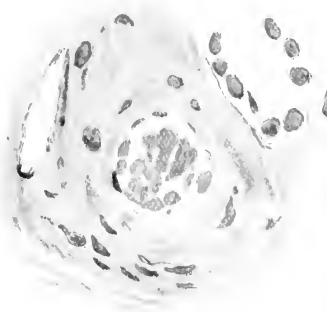
Fig. 5.

Fig. 3.

Fig. 4.
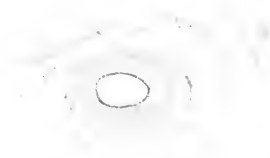
PLATE III

(1) A glomerulus from the kidney with hyaline degenerated vascular coil. × about 400.

(2) Small vessel surrounded by hyaline fibrotic material. No vessel-wall distinguishable. (A small portion of (5) more highly magnified.) × about 400.

(3) A small vessel, apparently a vein, in longitudinal section, shewing hyaline degeneration of the wall. From the anterior-median fissure of the spinal cord. × about 400.

(4) An arteriole with a thickened homogeneous hyaline degenerated wall, from the membrane of the anterior fissure of the spinal cord. × about 400.

(5) Section of kidney at the base of a pyramid shewing marked interstitial fibrosis, with fibrotic hyaline degeneration of the vessel walls, and atrophy of the parenchyma. Many of the tubules are denuded of epithelium, and contain homogeneous purple - stained casts seen in section. × 220.
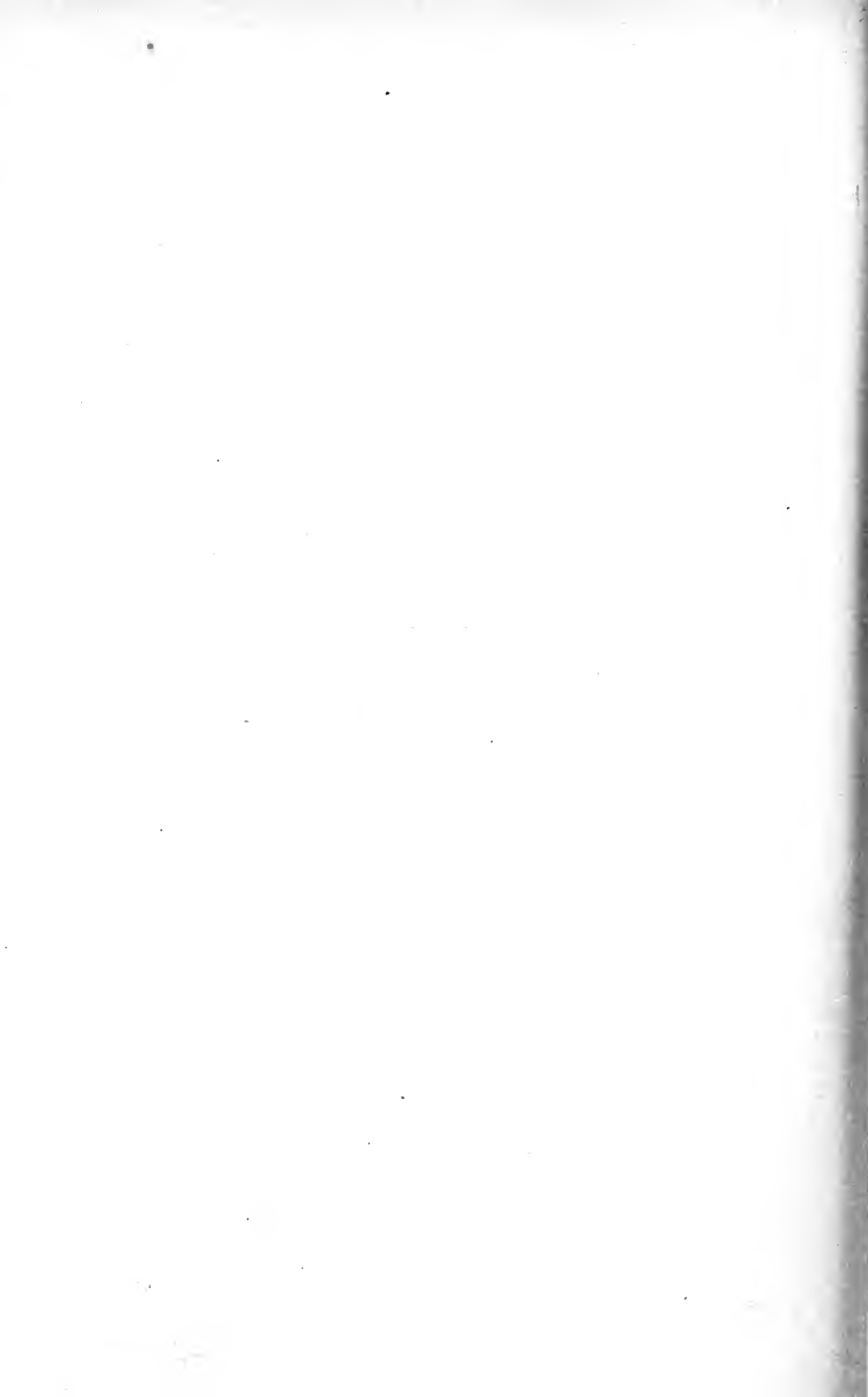
Occupation.—All causes which tend to increase the force and frequency of the heart's beat will increase the stress on the muscular and elastic structures in the large arteries, especially if this be accompanied by increase of the peripheral resistance. In particular, occupations involving continuous muscular exertion tend to degeneration of the large arteries ; navvies, blacksmiths, porters, labourers, soldiers are often the subjects of this disease early in life. It is said that workmen shew arteriosclerosis in the upper limbs, and that the arteries of the right arm suffer earlier and more extensively than the left. Important as is the fact that mechanical strain seems of itself competent to produce premature degeneration of the arterial system in a normal healthy man living under healthy conditions, yet, according to Bollinger, horses, oxen, and dogs used for traction purposes, and therefore subject to mechanical strain, do not suffer from atheroma. Tschigajew has studied the mean arterial blood-pressure in Russian peasants, while engaged in long and continuous work in the fields during the summer, and while at rest during the winter. During the period of work there was increase of arterial pressure, but this gradually disappeared in the winter. He compared these results with his observations upon labourers in the iron foundries, who work continuously under unhealthy conditions throughout the year ; in the latter class he found permanently high arterial pressure, and thickening of the arteries due to arteriosclerosis (see " Over-stress of the Heart," p. 193). Lastly, it may be mentioned that any occupation involving stress in a moist, cold atmosphere is said to be especially prone to produce arteriosclerosis.

Sex.—Arteriosclerosis is much more common in men under fifty than in women ; but after the climacteric period it is as common in women as in men, if not commoner. Men are much more subject, as a rule, to the causes I have already discussed, and when women are placed under the same conditions they are as liable to arterial degeneration as men. Jusserand has pointed out that at Lyons, where a large number of women employed in laborious occupations under insanitary conditions attend the hospital, the proportion of cases of arteriosclerosis is larger in the women than in the men. Dr. M'Crorie, on examination of the post-mortem records at Glasgow, found that the disease was as frequent in women as in men. Pregnancy appears to favour the development of arteriosclerosis and, according to Ascoli, the most marked aortitis occurs in women who have had the most pregnancies. Again, it is said that a prolonged and difficult establishment of the menopause favours the appearance of arteriosclerosis.

Morbid Anatomy and Pathology.—When arteriosclerosis affects the aorta and its large branches, it is usually named atheroma ; some authors, however, prefer the name endarteritis deformans. Of this there are two varieties—nodosa or circumscripta, and diffusa. Councilman, in a study of arteriosclerosis, divides the subject into three varieties—the nodular, the senile, and the diffuse. In the *senile* form he points out that there is atrophy of the liver and kidneys, and that the heart is often small ; in

half the cases he examined (7 out of 14) there was no cardiac hypertrophy. In the *diffuse* form the disease is widespread throughout the aorta and its branches, and in this vessel is often associated with *nodular sclerosis*.

Nodular Arteriosclerosis.—With the naked eye we see oval or circular projections of the inner coat of a more opaque or yellow colour than the surrounding tissue, varying in size from a hemp-seed to a shilling. These are scattered over the surface of the aorta, especially in the ascending part of the arch, affecting the concave surface rather than the convex. They occur also at the bifurcation of the aorta, whilst between these two situations the atheroma may be absent except around the orifices of the vessels.

The process begins by the formation of semi-transparent gelatinous patches. When the process is older the patches are found to be firm, dense, and of cartilaginous consistence. As a rule the endothelial lining of the vessel is intact, and the change is situated in the layer between the endothelium and the elastic lamina. The older patches are yellowish and opaque, owing to necrobiosis of the deeper layers of the patch where caseation, and frequently calcification, are taking place. When cut into, the former process is recognised by the fatty detritus—something like porridge, or fine meal, as the name atheroma implies—which is seen in the deeper parts. Calcification may be so extensive as to be quite obvious to the naked eye in the form of calcareous plates, producing some resemblance to the hide of a crocodile ; or, in the slighter degree, it may only be recognisable by the gritty character of the patch when cut into. Ossification in sclerotic vessels has been recorded. Virchow in his *Cellular Pathologie* has stated that although most of the so-called bony aortas and vessels were undoubtedly calcified, the formation of true bone does occur. In 1886, Mr. Paul demonstrated before the Pathological Society of London, a specimen from a sclerotic tibial artery in which there was a focus of bone-formation in the intima. Mönckeberg, in 1902, published an extensive research on bone-formation in sclerosed vessels. Out of 100 cases examined, he found true bone-formation in 10. Poscharissky in an examination of fourteen hearts found bone-formation on three occasions, the mitral valve being affected in 2 of these cases. C. H. Bunting reports a case of formation of true bone with cellular (red) marrow in a sclerotic aorta, and states that bone-formation has been found in the aorta six times, in the intima and media of medium-sized arteries twenty-two times, and in the cardiac valves eight times. For the bibliography of this subject the reader should consult Bunting's paper. It is probable that the cases which have so far been reported do not indicate the true frequency of this condition, and further study on this important subject is desirable. If atheromatous material be examined microscopically, crystals of cholesterin, fatty acids, oil-globules, and disintegrating cells may be observed. Microscopical examination of patches of atheroma will shew changes according to the age of the patch ; but usually all stages of the disease may be seen, the central portion being the oldest. The endothelial layer is bulged inwards by a great increase in the subendothelial layer, the cells of which

have undergone proliferation. These cells consist of branched, stellate and fusiform elements, and in the centre they are seen in all stages of necrobiosis. Crystals of fatty acids can generally be observed in the deeper and more central portions of a patch.

Examination of the middle coat shews that the muscular fibres present a hyaline swollen appearance, and occasionally the elastic fibres have been found ruptured. Now this would lead to a bulging of the vessel-wall were it not for the fact that a proportional compensatory thickening of the inner coat takes place by proliferation of the sub-endothelial layer already referred to. Thoma, by injecting the aorta and large arteries with paraffin wax at a pressure of 160 mm. of mercury (the mean pressure in the aorta), has shewn that the paraffin casts are quite smooth, and present none of the irregularities which the nodular plaques would have produced had they not exactly filled up the bulge in the vessel-wall produced by the weakened media at the spots where the intima is thickened. Certainly this is a strong argument in favour of the compensatory view.

Why should the proliferated connective-tissue cells of the sub-endothelial layer of the intima undergo degeneration in the central parts of the plaque ? I look upon it as a process in which the younger and more vigorous cells are able to take up nutriment, while the older, situated in the deeper and more central portions of the patch, perish and undergo fatty degeneration. The process of necrobiosis may extend to the whole patch, and the intima may give way and form an atheromatous ulcer. Dr. Ainslie Hollis attributes atheroma to an infective process. I think this possible when ulceration occurs.

According to Rokitansky the order of frequency with which other vessels are affected is as follows—splenic, iliac, femoral, coronary, cerebral, uterine, brachial, spermatic, and common carotid. The arteries of the stomach and the mesenteric are but rarely affected, and the pulmonary least of all.

The frequency with which the coronaries are affected we can easily understand from their situation, subject as they are to the highest arterial pressure. The splenic, again, is an artery which must be subject to variable arterial pressure, for, during the rhythmical contraction of the muscular tissue of that organ, there is a considerable increase of the peripheral resistance.

The relative infrequency of affection of the pulmonary artery, which occurs under such conditions as involve increased tension of its walls—as, for example, prolonged mitral stenosis—indicates the importance of internal strain as a factor in the degenerative process. The importance of syphilis has already been referred to on p. 569.

Pepper cites a case in which the pulmonary artery, as a result of prolonged right heart hypertrophy in mitral stenosis, was sclerotic and atheromatous to its minutest subdivisions. The aorta and the arteries of the systemic circulation were but little affected. Romberg, however, has described two cases of sclerosis of the pulmonary arteries without valvular disease, and without any marked

morbid change in the lungs. It is specially noteworthy that there was no morbid resistance to the pulmonary circulation. This is a very rare and peculiar form of disease, and till recently unknown. There was marked cyanosis, which was not related in a particular degree to congestion. The right heart was hypertrophied; probably the disease was congenital. The lumen of the pulmonary arteries was greatly dilated up to the second division; that of the smaller arteries notably diminished. There was marked sclerosis of the intima, the cause of which was not determined. These cases closely resemble those recently described by Prof. Rogers, which he ascribes to syphilis, (*vide* p. 569).

Diffuse Arteriosclerosis.—This affection is part of a widespread process
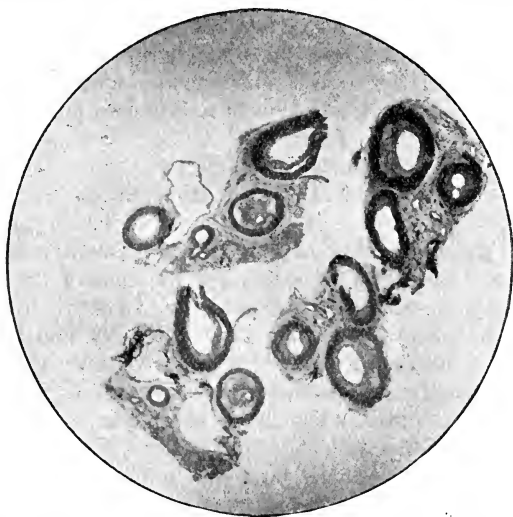
Fig. 71.—Transverse section of the arteries of the kidney from a case of general arteriosclerosis in a man aged fifty-six. The patient was a soldier and had had syphilis. He suffered from an attack of acute nephritis, with cardiac failure from dystrophic fibrosis. There was dropsy, albuminuria, and a petechial eruption. All the arteries were rigid, tortuous, and atheromatous. The photomicrograph shews an obliterative endarteritis of the branches of the renal artery at the hilum of the kidney. Magnification, 6 diameters.

affecting the arterio-capillary system and corresponds to the cases described by Gull and Sutton as arterio-capillary fibrosis. It occurs especially in strongly-built middle-aged men. The process begins in the small arteries and capillaries, especially those of the renal cortex, but also in those of the brain and heart. It is very frequently associated with nodular atheroma of the aorta, doubtless due to changes in the vasa vasorum (*vide* Figs. 67 and 68). The heart is usually greatly hypertrophied, and when the coronary arteries are involved, as they often are, cardiac dystrophic fibrosis is often associated with it. The kidneys may shew extreme fibrosis but sometimes the organs are increased in size; the capsule is adherent and the organ is tough. Microscopically the unstriped muscular fibres of the media exhibit hyaline swelling, fatty degeneration,

or atrophic changes, so that the muscular elements are often not recognisable. This is especially the case in the small arteries of the kidney, where the wall of the vessel may appear to consist only of a homogeneous hyaline tissue. Sometimes the degenerated atrophied fibres of the media can be made out, but nothing of the elastic lamina, the intima being thickened and represented only by a homogeneous hyaline material with but few nuclei. The capillaries of the glomeruli shew this hyaline change especially well, a change which may, and frequently does, lead to their obliteration (*vide* Figs. 71 and 73). A similar change is found in the vessels of the pia mater (*vide* Fig. 72). The results of these widespread changes are increased resistance to the flow of blood through the capillaries, hypertrophy of the left ventricle, dilatation of the larger arteries from degenerative changes in the muscular and elastic tissues of

FIG. 72.—Cerebral vessels from a case of arterio-capillary fibrosis. The small vessels of the pia mater shew large numbers of leucocytes around. In one vessel the lumen is completely obliterated, in others the walls are much thickened. *Vide* also figure 74 of miliary aneurysms which were obtained from this case. Magnification, 100 diameters.

the media, slowing of the circulation, and compensatory proliferation of the subendothelial layer of the inner coat. Disease of the kidneys is always detected if the organs are examined microscopically ; but to the naked eye the change is sometimes not so apparent, and may be overlooked. In all cases its degree depends upon the stage of the disease and the mode of death. The red granular contracted kidney is the most characteristic naked-eye appearance. The heart in most cases is greatly hypertrophied, but it is generally tough, and if examined microscopically, the muscular fibres are seen to be surrounded by a great excess of fibrous tissue, and the small arteries and capillaries are thickened and present fibrous hyaline degenerations, generally accompanied by arteriosclerotic changes in the coronary arteries. Both the semilunar and mitral valves may be thickened, opaque, and sclerotic.

Sclerosis of the pulmonary artery has been discussed on p. 587.

Sachs has microscopically examined the arteries in 100 cadavers, and the results of his observations shew that in diffuse arteriosclerosis the arteries of the limbs, especially of the lower limbs, are most frequently affected, the anterior tibial being the artery most often affected in the body.

Thoma points out further that the sclerotic process may affect the veins also—*phlebo-sclerosis.*

To Gull and Sutton is due the conception of *arteriosclerosis* as an independent affection, named by them *arterio-capillary fibrosis.* They proved that the red granular contracted kidney of chronic Bright's disease is but a part of a general vascular disease, and their observations were most valuable in demonstrating that what was looked upon as a disease of a single organ was, in reality, a widespread vascular change throughout the body, secondary to some other process which we now recognise as probably due to defective metabolism. Lancereaux came to the same conclusion, although under his title of *herpetism* his views and observations did not receive in this country the recognition they deserve. He shewed, however, that in 61 cases of generalised arteriosclerosis the associated conditions were—interstitial nephritis, 55 ; cerebral haemorrhage or softening, 12 ; pulmonary emphysema, 21 ; articular lesions, 14 ; friability of the bones, 10.

Gull and Sutton shewed that out of 35 cases of emphysema granular contracted kidney was present in 21, and this quite accords with my own experience. Mahomed made observations on 61 cases of chronic Bright's disease without albuminuria, but with high-pressure pulse and cardiovascular changes. He held that in the red granular contracted kidney the disease was primarily in the vessels throughout the system.

Dr. Dickinson argues that the disease of the kidney is the initial cause of the cardiovascular change. In 250 cases of granular contracted kidney he found cirrhosis of the liver in 37 instances only. It must be borne in mind, however, that, if excessive internal stress on the vessel-wall be the cause of degeneration, then we should not expect the liver to be affected, because we know that, owing to the free anastomosis of the hepatic and portal vascular systems, a rise of internal stress would not readily occur. It might be argued that the 37 cases of cirrhosis were due to alcohol or mechanical congestion. Dr. Dickinson says that in chronic renal disease there occurs a hypertrophy of the cardio-arterial system which is universal from its origin to its terminations, and affects not only the ventricles and the arterioles, but also the intermediate arteries of every size. He has also shewn that in cases of granular contracted kidney abnormal thickening may be demonstrated in the larger as well as in the smaller arteries. His researches shew that the aorta, the innominate and the femoral arteries are all increased in the total thickness of their walls, in the thickness of their muscular coat, and in their circumference. He holds, with Bright, that the peripheral resistance is mainly in the capillaries, and opposes the "stop-cock hypothesis" of the late Sir George Johnson ; but he agrees with Johnson that the muscular

coat is hypertrophied. A long controversy ensued between Gull and Sutton and Johnson on the causation of the cardiac hypertrophy and high arterial pressure. Johnson attributed the thickening of the arterioles to the hypertrophy of the muscular elements; Gull and Sutton to a fibrotic change of arteries and capillaries, beginning usually, but not always, in the kidney. There may be truth in both opinions. In the prodromal stages of the disease a spasm of the arterioles is probably brought about by irritation of toxic products, causing contraction of the muscular coat, increased peripheral resistance, and compensatory increased force of the heart's action; whereupon the arterial blood-pressure rises. Thus not only would the heart undergo hypertrophy, but the muscular coat of the arteries also. Sir Clifford Allbutt, however, regards this hypertrophy as due not to excessive contraction of the arterioles, which in other diseases, as in Raynaud's disease, does not produce it, but, as in the heart, to a compensation of the dilating pressure.

The late Sir Wm. Broadbent did not think that the primary obstruction is produced by contraction of the arterioles; he admits that the muscular coat of the arterioles is increased, but that this is secondary to the resistance in the capillaries. Dr. Dickinson points out that the capillaries, although containing no muscular fibres, are yet capable of contraction. He admits that, associated with the renal disease, there may be widespread changes in the arterio-capillary system of the whole body, but that the hyaline fibroid change described by Gull and Sutton is secondary to the renal disease.

Thoma made injection experiments upon cadavers with salt solution, observing the times of injection of a given number of litres at a given pressure, when arteriosclerosis existed, and when it did not. He found it took very much longer to inject the same amount of fluid in the case of sclerosis. Oedema of the lower extremities occurred when only four litres had been injected into a body affected with widespread angiosclerosis; whereas into the arteries of a body not so affected seventeen litres could be injected before leakage took place. These and other experiments shew that when arteriosclerosis is present the salt solution has to overcome much more resistance in the vessels of the lower extremities, although investigation shews that the lumen of the arteries is not greatly diminished. It may be concluded, therefore, that changes in the permeability of the capillary walls are also present in arteriosclerosis. Does it not also suggest that the capillary area generally is greatly diminished, probably on account of the fibrotic changes referred to?

Again, the researches of Hoffmann, Runeberg, and other pupils of Thoma prove that the fluid in angiosclerotic oedema is characterised by a small amount of albumin and low specific gravity, indicating hydraemia of the blood; to this may be attributed the defective metabolism, the abnormal permeability of the capillaries, and the degenerative changes in the muscular and elastic tissues which Rokitansky, Thoma, and most authors believe to be the initial factor in the thickening of the inner coat.

It can easily be understood that a widespread change affecting the capillaries and arterioles may cause an increased peripheral resistance in the circulation which has to be overcome by an increased force of the heart's action, resulting in hypertrophy of the ventricle. These two factors lead to increased stress upon the large arteries, to their eventual distension, laterally and longitudinally, to thickening of their walls, and to tortuosity.

Thoma, by a series of researches, has shewn that the thickening of the walls of the arteries, especially of the inner coat, is a process compensatory to the slowing of the current caused by the distension of the vessel. If so, the thickening of the intima may be looked upon rather as a compensatory fibrosis than an endarteritis, because, as Prof. Adami points out, although Thoma speaks of the process as a chronic inflammation, yet he states that a similar process occurs under physiological conditions ; for he and his pupils have shewn that a strictly analogous thickening of the intima, due to proliferation of the subendothelial layer, occurs at birth in the portions of the aorta between the ductus Botalli and the points of departure of the umbilical arteries, in the uterine arteries after parturition, to a less degree after menstruation, and in the arteries of an amputated limb.

General arterio-capillary fibrosis leads to thickening of the coats of the arteriae arteriarum, and I am inclined to believe, from many observations, that frequently degeneration of the media is primarily due to obliteration or obstruction of the vasa vasorum, and consequent defective nutrition of the muscular fibres ; but Prof. Adami argues that, if this be so, its influence on the intima is not apparent, for he cannot find evidence in healthy arteries, or in the earlier stages of arteriosclerosis, that any branches of these vessels pass into the intima. The reply to this argument is—(i.) for what purpose is the elastic lamina fenestrated ? (ii.) no vascular branches pass into the cornea, which consists of layers of branched connective-tissue corpuscles quite similar to the subendothelial layer of the arteries ; and (iii.) the nodular form of arteriosclerosis is only to be explained, I think, by supposing that an area of a vessel-wall has been damaged by some anatomical imperfection in the blood-supply of the vasa vasorum.

In the necropsies of the London County Asylums I have been struck with the frequency of arterial degeneration, and with the proportional infrequency of intracerebral haemorrhage, as compared with my experience and statistics obtained at Charing Cross Hospital.

I have therefore investigated the reports of 300 necropsies made at Claybury Asylum. Of 160 male cases, the great majority in persons after middle life, there was atheroma of the aorta in 113 ; generally speaking, the disease affected both the aorta and the cerebral vessels. The cerebral vessels were noted as diseased in 60 cases, in 40 markedly so. In 65 the kidneys shewed some degree of interstitial fibrosis, usually not very marked. The heart was moderately hypertrophied in a few cases, and in only 2 was it much hypertrophied. Atheroma was noted in

24 cases out of 86 in people aged forty-five or under ; 22 of these were the subjects of general paralysis, but of the total 86 cases, 60 were general paralytics. Thus it appears that atheroma of the aorta was present in about 1 in 3 cases of general paralysis and 1 in 13 in other cases of men who had died at forty-five years of age or under. Of 140 female necropsies, atheroma of the aorta was observed in 81. Of these, 34 were noted as "marked,"the remainder as "some atheroma." The cerebral vessels were noted as diseased in 49 cases ; in 35 marked, in 14 moderate or slight. The kidneys shewed moderate interstitial fibrosis in 19, slight in 21, fatty in 9 cases. The heart was moderately hypertrophied in 2 cases, and considerably hypertrophied in 2 cases. Aortic sclerosis was noted in 18 cases out of 53 in

FIG. 73.—Photomicrograph of kidney from a case of arterio-capillary fibrosis. In the centre is a small artery with thickened hyaline walls. The patient, aged forty-eight, died of cerebral haemorrhage. The "hair" vessels are shewn in the photomicrograph.

persons aged forty-five or under. There were 18 cases of general paralysis, and in 10 of these there was atheroma of the aorta and especially a form of nodular pearly fibrosis suggesting the influence of syphilis. In five cases some nodular endarteritis cerebri was discovered on very careful microscopic examination of all the cerebral arteries by pulling the vessels out, floating them in water, and selecting any opaque spots for microscopic investigation. In this way disease was often discovered which might otherwise have been easily overlooked. In one case of atheroma of the aorta no disease of the cerebral arteries was discovered, and there was one case of disease of the cerebral vessels in which the aorta was unaffected. So that more than half the cases among women exhibited arterial disease, just as in the men, and without evidence of long previous high arterial pressure ; for in none of these cases was there cardiac hypertrophy. The proportion of atheroma in cases other than general paralysis was 1 to 5 ; in nearly all cases it was slight, and the cerebral vessels were not affected.

Since this article was published in the first edition, I have collected the statistics relating to 1926 necropsies conducted at Claybury Asylum by my assistants or myself during the ten years, 1st September 1898 to 31st March 1908, with a view of ascertaining the percentage-incidence of intracerebral and subdural haemorrhage and deposit in the insane. The results are recorded in the subjoined table, from which the following conclusions may be deduced. The total percentage of intracerebral haemorrhage is 1·2 per cent. With the exception of two cases of

doubtful general paralysis, the haemorrhage occurred in the subjects of insanity other than general paralysis. In the case of subdural haemorrhage and deposit, especially in males, there appeared to be two separate morbid conditions: (*a*) Cases of undoubted subdural haemorrhage in elderly people associated with arterial degeneration and cardiac hypertrophy; (*b*) Cases of membrane formation of a similar nature to that found in general paralysis. Among the females the former class of case was not in evidence. Combining therefore the intracerebral and subdural types of haemorrhage we have a sum total of 35 cases out of 1926 necropsies, which is about 2 per cent. In practically every case of intracerebral and subdural haemorrhage there was associated chronic vascular and renal disease (*vide* Table); a condition markedly different from that found in cases of chronic pachymeningitis, in which the heart, together with the other viscera, usually shewed some wasting; especially was this the case in the subjects of general paralysis of the insane.

	Male.	Female.
Average weight of the insane heart in grams [1] . .	300	270
Average weight of the heart in the cases of intracerebral haemorrhage (M. 16; F. 9)	400	306
Weight of heaviest heart	600	440
Weight of lightest heart	310	200
Number of hearts above average weight . . .	16	5
Number of hearts below average weight . . .	0	4
Average weight of heart in cases of subdural haemorrhage other than general paralysis of the insane (M. 12; F. 1)	402	205 [2]
Number of hearts above the average weight . .	10	...
Weight of heaviest heart	540	...
Weight of lightest heart	290	...
Average weight of heart in cases of haemorrhagic pachymeningitis other than general paralysis (M. 7; F. 13)	275	238
Average weight of heart in cases of haemorrhagic pachymeningitis of general paralysis (M. 18; F. 2) .	290	257
Total number of deaths (out of 1926) occurring at 45 years and under	355	343
Number of cases of intracerebral haemorrhage occurring at 45 years or under	3	1
Number of cases of subdural haemorrhage and pachymeningitis other than general paralysis occurring at 45 years or under	1	1
Number of cases of haemorrhagic pachymeningitis in general paralysis occurring at 45 years or under .	11	1

[1] Obtained from an average of 100 male and 100 female consecutive necropsies.
[2] One case only.

TABLE shewing the incidence of Cerebral Haemorrhage and Chronic Pachymeningitis in the Insane.

	Males.	Females.	Total.
Total number of necropsies performed . .	937	989	1926
Number of cases of general paralysis . .	334	127	461
,, insanity other than general paralysis	603	862	1465
,, intracerebral haemorrhage in cases of general paralysis .	0(2 ?)	0	0
,, haemorrhagic pachymeningitis in cases of general paralysis	18	2	20
,, intracerebral haemorrhage in cases other than general paralysis	16	9	25
Percentage incidence of intracerebral haemorrhage on total number of necropsies . .	1·7%	1·0%	1·2%
Number of cases of subdural haemorrhage in cases other than general paralysis	12	1	13
,, haemorrhagic pachymeningitis in cases other than general paralysis . .	7	13	20
Combined total of cases of cerebral haemorrhage —intracerebral and subdural . . .	28 = 3%	10 = 1%	38 = 2%

Without laying too much stress upon these statistics, for I was not responsible for the notes made with varying degrees of care and discretion, I think they certainly shew that my impressions were correct as regards the difference between asylum and hospital experience. The conclusions I think we are warranted in drawing are : (i.) The relative infrequency of intracerebral haemorrhage in the insane with atheromatous cerebral arteries is probably due to the fact that they have not been the subjects of prolonged high arterial pressure, as shewn by the absence of cardiac hypertrophy. (ii.) The interstitial fibrosis of the kidney seldom assumes the degree present in the small red kidney of chronic Bright's disease. (iii.) Meningeal haemorrhage is due to rupture of small veins or capillaries. (iv.) There is a relatively high proportion of nodular aortitis, especially the pearly-white fibrotic plaques suggesting syphilis. (v.) The great proportion of arterial disease among the inmates of asylums is not as a rule connected with prolonged high arterial pressure and chronic Bright's disease, but is due to a general degenerative process in which the parenchyma of the kidney takes part. The infrequency of cardiac hypertrophy is probably connected with an infrequency of miliary aneurysms, and, consequently, a proportional infrequency of intracerebral haemorrhage. (vi.) Among asylum patients the frequency of cerebral softening due to arterial degeneration is striking.

To ascertain the relative frequency of atheroma, the reports of 1600 post-mortems made at Charing Cross Hospital were analysed, and of these 380, or nearly one-fourth, were found to exhibit a greater or less degree of atheroma. In these the males were to the females nearly as 3 to 1. Of the remaining 1200 cases there was a large proportion of subjects who had died in infancy and youth, of women, and of cases in which, for some reason or other, complete examination was not made. In 118 cases the atheroma was severe, in 104 moderate, and in the remainder slight. Neglecting the cases in which the reports state that the vessels were slightly affected, we may reckon that there were 222 cases of atheroma. Bollinger, in 1800 necropsies on adults, gives only 136 cases of sclerosis. The difference in the percentage between Bollinger's statistics and mine may probably be explained by the fact that a large number of our male patients, by their occupation and habits, are particularly prone to arterial degeneration. Many are porters at Covent Garden market, dockyard labourers, people engaged at the theatres and in the liquor traffic; in fact, every year a considerable number are brought in dead or dying by the police.

Another important point in connexion with the etiology and pathology of the disease is the high percentage of granular contracted kidneys and hypertrophy of the heart; thus shewing the close association between arteriosclerosis and prolonged high arterial pressure.

In these 1600 necropsies there were 60 cases of cerebral haemorrhage of all kinds—a percentage of 3·7 per cent, nearly double the percentage-incidence at Claybury Asylum. Thirty of these cases of cerebral haemorrhage were noted in subjects suffering from granular contracted kidneys and hypertrophied heart. Fagge states that the large majority of cases of cerebral haemorrhage are associated with granular contracted kidneys and hypertrophied heart. The large percentage of cases in which chronic Bright's disease was associated with atheroma would suggest that the naked-eye change is but a part of the pathological process, and that the microscope would reveal changes in the arterioles and capillaries throughout the body, in many instances localised particularly in the kidneys, which organs, for years past, had been endeavouring to rid the body of the waste products of a defective metabolism. Such a stress on the renal vessels and parenchyma produces in them a degenerative change of a gradually progressive character not limited to these organs, although they shew it more particularly.

The relation of atheroma to aneurysm is of considerable importance and will now be discussed. Of 34 cases of aneurysm of the aorta which occurred in the above-mentioned 1600 post-mortems made at Charing Cross Hospital, 7 were women, and in nearly all these 34 cases atheroma is recorded. These results correspond very closely with those of Coats and Auld, and of M'Crorie, as will be seen in the following table :—

Ages.	Mott.	Coats and Auld.	M'Crorie.
20–30	1	1	1
30–40	10	15	8
40–50	11	10	8
50–60	9	7	7
over 60	3		

In the three schedules there is only one case under thirty, and the majority occur in the two decades between thirty and fifty; this is precisely the period in which atheroma is most frequently met with.

Drs. Coats and Auld came to the conclusion that atheroma is the main cause of aneurysm. They consider that the thickening of the intima, with its subsequent degeneration, is a chronic inflammatory process; that the name *Endarteritis deformans* of Virchow is therefore correct; and that the changes in the muscular and elastic tissues of the media are brought about by pressure of the degenerated caseous or calcareous intima. I quite agree with them as to the very frequent association of atheroma with aneurysm; but whether the atheroma is an essential or an incidental event is disputable. The arguments of the above authors are that in the great majority of cases of aneurysm atheroma is present; that, like aneurysm, it affects men more than women; that it occurs in the two decades of life when aneurysm is most common. Their view is opposed to that of Eppinger, and in a measure to that of Thoma, who looks upon thickening of the intima as compensatory, and, as one would therefore suppose, rather preventive of aneurysm. Eppinger considers that rupture of the elastic and muscular fibres of the middle coat is the primary cause of ordinary aneurysm, and this may be true of dissecting aneurysm; but it seems a doubtful assertion in respect of sacculated aneurysm, for, apart from Coats' weighty arguments, Wagner was only able to find microscopic rents in the elastic tissue of the media in high degrees of atheromatous degeneration, and, as suggested by Coats, these might very well have been brought about by pressure and stretching. Such rents are indicated by the development of fibro-vascular tissue which cicatrises later; and the accepted mesarteritis of Köster appears to Wagner to be generally the result of laceration of the media. Yet I feel sure, from my own observations, that in cases of atheroma in the subjects of syphilis a simultaneous periarteritis and mesarteritis may accompany blocking of the vasa vasorum and acute changes in the media (see Fig. 68 and explanation).

Under syphilitic endarteritis I have already referred to the frequency of the association of syphilis and aneurysm, of aneurysm and atheroma, and of syphilis and atheroma. Amongst a large number of necropsies occurring in the London County Asylums I have occasionally had the opportunity of seeing advanced atheroma of the aorta in conjunction with typical syphilitic endarteritis cerebri in women under thirty, in whom

other evidences of syphilis were forthcoming. I cannot but think that an endarteritis obliterans of the vasa vasorum in the neighbourhood of the atheromatous patches is a convincing proof of the effect of syphilis in the production of atheroma of the aorta in comparatively young people, not the subjects of strain (see Figs 67, 68).

Fränkel and Much have employed the Wassermann reaction on serum obtained post mortem ; they assert that it offers a means of determining the syphilitic origin of a vascular or visceral disease, and it would be of interest to ascertain whether the reaction is negative in certain forms of arterial sclerosis and present in others. Since nodular fibrosis is very frequently seen in the aorta of patients dying of general paralysis, we might correlate this anatomical change with the constant positive reaction yielded by the serum of patients suffering with this disease.

I have already referred to the frequency with which we found granular contracted kidney and cardiac hypertrophy associated with cerebral haemorrhage. Gull and Sutton made most of their observations on arterio-capillary fibrosis on the vessels of the pia mater. It is rare to find cerebral haemorrhage in a person under forty who is not the subject of chronic Bright's disease, or of infective endocarditis. Of the 60 cases I have collected, seven were due to intracranial aneurysm, and four of these, all in young subjects, were caused by infective embolism ; the other three were due to atheroma or syphilitic arteritis. Two were due to secondary haemorrhage from tumours ; in all the remainder some affection of the kidneys was noted. In 80 per cent granular contracted kidney and cardiac hypertrophy were present. In the great majority of the cases the age was over forty-five. The proportion of males to females was 4 to 1. In 48 out of the 60 cases the seat of the affection was in the neighbourhood of the basal ganglia. In four cases the haemorrhage was meningeal ; in six meningeal and in the hemisphere, in two in the pons Varolii ; in one it was in the cerebellar hemisphere.

Miliary Aneurysms.—Atheroma of the larger vessels of the brain has but an indirect connexion with haemorrhage ; but it is a frequent direct cause of cerebral softening in old people, occasioning in them dementia and paralysis. Still, if the arteries be affected at the base only, it is remarkable how much compensation is possible by collateral circulation. We have already seen that atheroma of the larger vessels is generally associated with arterio-capillary fibrosis, and examination of the small vessels of the brain in cases of cerebral haemorrhage reveals marked changes in their walls. Charcot and Bouchard, in 77 successive cases of cerebral haemorrhage, found small aneurysms just visible to the naked eye (measuring from 0·2 mm. to 1 mm. in diameter), which they named miliary aneurysms. They may be found by washing away the brain substance from the vessels, and are more readily seen in the vessels of the pia mater of the convexity ; but, according to Charcot and Bouchard, the order of their frequency in different regions is as follows :—central ganglia, cortex, pons, cerebellum, centrum ovale, middle cerebellar peduncle, crus cerebri, medulla oblongata. This order pretty nearly coincides with the order of frequency of the seat of haemorrhage. They believe that the aneurysms are the result of a periarteritis.

According to Zenker, the primary change is in the inner coat ; but there is no reason to suppose that the process which originates arteriosclerosis is different in the small arteries of the brain from that of the rest of the body. It is much more likely that, as Sir William Gowers suggests, the effective element in the change is the loss of the contractile and elastic elements, with resulting fibrous overgrowth of the intima and adventitia. The photographs of the kidney and the cerebral vessels, and the figure of miliary aneurysms, well illustrate this point ; although the vessels of the brain undoubtedly shew evident signs of a periarteritis, as described by Charcot. The

Fig. 74.—Miliary aneurysms (*vide* Fig. 72).

aneurysms themselves appear like little red grains on the vessels ; sometimes they have a shiny aspect ; the colour depends upon the condition of the blood within them. They are sometimes extremely numerous, as many as 100 having been found in one case. It is easily understood how such aneurysms are formed if the muscular and elastic coats are degenerated ; the wall of the vessel yields to the pressure of blood, an

Fig. 75.—Photograph of an aneurysm at the junction of the left internal carotid and posterior communicating ; it is attached to a piece of the tentorium. It was the size of a large filbert ; the photograph is of half the aneurysm, and shews a round hollow cavity surrounded by a laminated clot. The only symptoms exhibited during life were paralysis of the third nerve on the left side, headache, and outbursts of passion. *Vide* also photomicrograph, Fig. 76.

immature fusiform or sacculated aneurysm takes place, and is liable to rupture at any time (*vide* Fig. 74).

In arteriosclerosis we have the two factors necessary for the production of miliary aneurysm, namely, weakening of the arterial wall and increase of blood-pressure. Probably the reason that these aneurysms are found especially in the brain is because the walls of the cerebral arteries are relatively thin ; there are but few muscle-fibres and vasomotor nerves. The arteries run in channels beneath the membranes, or in the substance of the brain, and, instead of being supported by solid tissues, they are surrounded by cerebrospinal fluid, which, although it distributes an even pressure to all the structures within the cranium, would offer less resistance to dilatation at a weak spot in the vessel-wall.

The Relation of Cerebral Aneurysm to Arteriosclerosis.—I refer now to

aneurysm, usually single, varying in size from a pea to a walnut. Primary degeneration of the vessel is an occasional cause in the second half of life (Gowers); this may be a fibroid change or a simple atheroma. Occasionally a simple weakening of the media, rupture of the elastic intima, and formation of an aneurysm may occur. This was so in a patient, a woman aged forty-four, who died suddenly. The photograph (Fig. 75) and photomicrograph (Fig. 76) shew, respectively, the aneurysm filled with laminated clot which was seated partly on the posterior communicating, partly on the internal carotid, and a section of the internal carotid just at its bifurcation; several little vesicular swellings were visible on the trunk and its branches in the neighbourhood of the aneurysm, and these, on section and microscopical examination, were found to consist only of the delicate adventitia, the media and intima having been ruptured. This

FIG. 76.—Photomicrograph. Transverse section of the internal carotid artery just at its division. At the places where the wall is extremely thin there existed little vesicular dilatations about the size of a large pin's head. As these were close to the sac of the aneurysm, it could not be discovered whether the fatal haemorrhage had been caused by the rupture of one of them or of the aneurysm itself. Magnification, 10 diameters.

no doubt explains the formation of the aneurysm. Another condition which sometimes gives rise to aneurysm is embolism from the debris of an atheromatous ulcer. This condition, however, is rare. The photograph (Fig. 77) shews an aneurysm at the bifurcation of the middle cerebral artery occurring in a woman aged thirty-eight, who died from hemiplegia caused by thrombosis extending from this aneurysm back into the middle cerebral trunk. The patient was free from valvular disease, but had general arteriosclerosis. Sections of the aneurysm shewed that a calcareous embolus had been driven into the vessel, and set up inflammation followed by dilatation.

Of the other arteries which are affected by arteriosclerosis, by far the most important clinically are the coronaries, disease of which may lead to imperfect nutrition and degeneration of the heart. Again, in the vessels of the limbs, as before seen, gangrene, especially of the lower limbs, is prone to come on in old people, owing to arteriosclerosis of the popliteal and tibial arteries, slowing of the circulation, and thrombosis

(Fig. 78). It will be gathered from these remarks on the pathology and the morbid anatomy of arteriosclerosis that it is a chronic, progressive, and cumulative disease of the whole vascular system, which in different individuals may shew a predilection for these particular vessels, or those, according to the various causes, immediate and remote. Many diseases which we recognise clinically as distinct maladies, are in reality a part of this general progressive change of vessels; namely, chronic Bright's disease, apoplexy, cerebral softening, senile dementia, fibroid heart, and sometimes fatty heart. To summarise: The pathology of arteriosclerosis is primarily defective metabolism and strain; physiological compensation—that is, increased functional activity of the left ventricle to overcome the increased peripheral resistance in the arterioles and capillaries —ensues and leads to hypertrophy of the muscular structures engaged, and to dilatation of the elastic aorta and large arteries. In the second
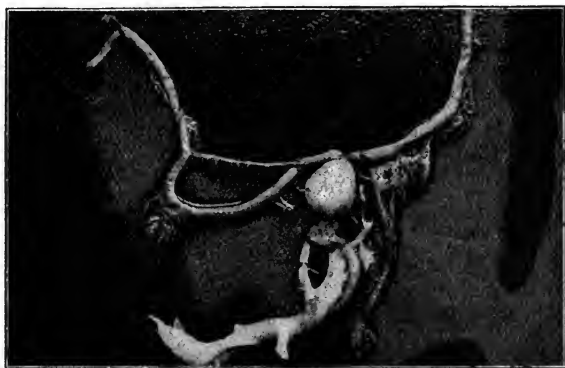
Fig. 77.—Aneurysm of the middle cerebral with thrombosis extending back into the internal carotid. Softening of the central region of the right hemisphere and basal ganglia, and left hemiplegia. The aneurysm was produced by impaction of an embolus from an atheromatous ulcer.

stage there is thickening of the vessel-wall, mainly of the intima, proportional and compensatory to degeneration of the muscular and elastic tissues. In the third stage the compensatory process fails, so that should the patient escape the danger of cerebral haemorrhage he may succumb in the final stage to blocking of his coronary arteries and consequent cardiac failure. Herein the general deficiency of nutrition, which alters the whole metabolism of the body, leads of itself to the failure of the physiological compensation which had been set up, and the inefficiently nourished muscular structure of the heart is unable to overcome the resistance in front. Dilatation of the left ventricle then follows, and mitral regurgitation, congestion of the lungs (frequently emphysematous), and dropsy, partly cardiac, partly due to changes in the capillary walls and the hydraemic condition of the blood, complete the vicious circle.

The Experimental Production of Arterial Degeneration.—Within the last few years attention has been directed to the production of experimental arterial disease in animals. Mention has already been made of

the acute local changes which I produced many years ago by the application of nitrate of silver to the external coat of the carotid artery in animals (p. 553). This line of research has been recently followed by Sumikava and by Harvey. The former has described the early changes obtained by similar experiments, the latter has found calcareous changes and bone-formation in the aorta of rabbits which have been allowed to survive some months after the application of nitrate of silver solution to the external coat.

As has been already mentioned, acute arterial changes have been produced in animals by the injection of bacteria and their toxins.
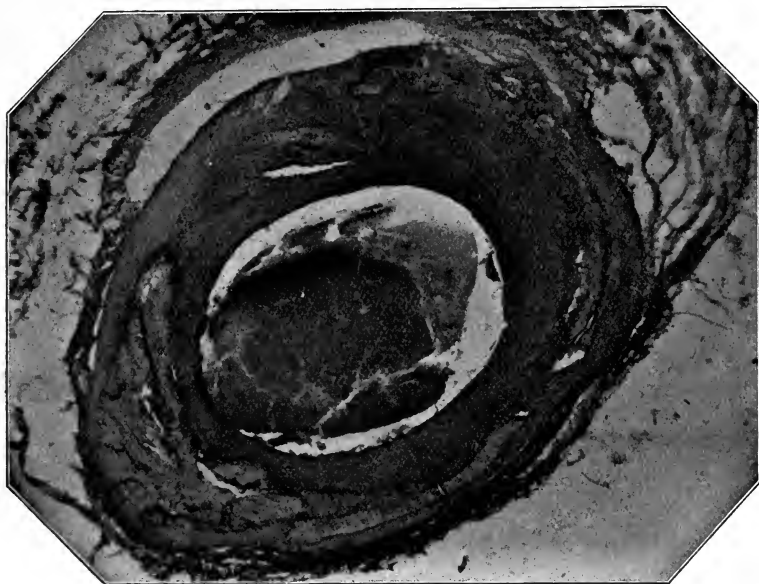
Fig. 78.—Photomicrograph. Anterior tibial artery from a demented patient who suffered from senile gangrene of the leg. A large calcareous plate can be seen in the middle coat, and the lumen of the artery is blocked by a recent thrombus.

Further, Gilbert and Lion, by the adoption of similar methods, have obtained changes which they consider are comparable with the chronic changes seen in the human aorta. In 1903 Josué published an account of the experimental production of arterial lesions in the aorta of rabbits as the result of repeated intravenous injections of adrenalin ; these he regarded as somewhat similar to those of human arteriosclerosis. His work has been confirmed by a large number of observers using adrenalin, and also several other drugs which have the property of raising the blood-pressure, such as digitalin, nicotine, barium chloride, squill, hydrastin. Adrenalin, however, appears so far to have given the most constant results. The single injection of 3 minims of a 1 in 1000 solution of adrenalin into the vein of a rabbit's ear, may be followed by difficult and rapid respiration,

collapse, and death ; at necropsy there may be found dilatation of the heart, oedema of the lungs, serous and cerebral haemorrhages. If the animal survive one or two dozen injections, very definite lesions may be found, consisting of distinct patches or plaques, pearly-white in colour, and often slightly depressed. Calcification of these areas is common, especially in the old lesions, and there may be aneurysmal dilatations. Microscopically, the earliest changes consist in granular degeneration of the muscle-fibres of the middle coat, the elastic fibres lose their wavy or crenated appearance and become stretched and frequently broken. Necrosis is followed by the deposition of calcium salts. The changes may be well advanced in the course of two or three weeks. The majority of observers are agreed that the primary changes begin in the media, and that the intimal changes, which are seldom seen, are secondary to the medial. Josué, and Baylard and Albarède, however, consider that the intimal changes are of primary importance. Pearce and Stanton have failed to find fatty degeneration by histological examination, and this appears to be the general experience. Oscar Klotz, however, states that the primary change in the media is fatty degeneration of the muscle-cells followed by a similar change in the elastic fibres. His opinion has been recently upheld by Bennecke, who concludes that fatty degeneration of the epicardium and myocardium is the forerunner of the aortic change, and that the first stages of the aortic lesion consist of fatty degeneration of the intima and media.

Loeb and Githens endeavoured to neutralize the rise of blood-pressure caused by the adrenalin, by the simultaneous injection of amyl nitrite. They considered that the arterial changes were due to the toxic action of the former, and not to the rise in blood-pressure, because the combined injection of the two drugs did not prevent the production of the lesion. Prof. Dixon, however, contends that they were wrong in their assumption, as he has shewn that the simultaneous injection of adrenalin and amyl nitrite does raise the blood-pressure, though the duration of the rise is less than when adrenalin alone is injected. Bennecke has used spermine in the place of amyl nitrite to counteract the blood-pressure-raising properties of barium chloride and hydrastin. He has likewise obtained arterial lesions, but in a smaller percentage of cases than by the use of either barium chloride or hydrastin alone. In the face of such controversial opinions caution must be exercised in interpreting results obtained from these intricate experiments. The difficulty of calculating the precise amount of the two drugs which will produce an exactly neutral effect as far as the blood-pressure is concerned, must be very considerable ; again, each animal may differ from its fellow as to the amount required ; further, it is impossible to premise what damaging influence the chemical combination of the drugs may exert upon the tissues of the animal.

The toxic property of the adrenalin is evidenced by the production of acute cardiac and respiratory disorders and death after a single injection. That this is not specific to adrenalin has been shewn by Fischer, Bennecke, and several others, who have found that similar effects can be obtained

by the use of entirely different drugs, with which they have succeeded in obtaining similar arterial degenerations. How far the prolonged toxic action of the drugs may *per se* conduce to the production of these changes is not yet understood.

Ziegler, Erb, and Lissauer are the chief supporters of the hypothesis that the injection of adrenalin produces disturbances of the vasa vasorum, such as cramp and contraction which lead to malnutrition and anaemic necrosis of the vessel-wall in the immediate area supplied by these vessels. This has been denied by several observers on the ground that changes in these vessels have not yet been described. It is possible that they have been overlooked; Ziegler, however, alludes to changes in the vasa vasorum around the necrotic foci. Huchard and his pupils have insisted upon the important part which these vessels play in the production of human arteriosclerosis.

From the results of his compression experiments, Harvey states that he can produce changes in the aorta of rabbits exactly similar to those induced by adrenalin injections. He concludes that the arteriosclerosis produced in rabbits by the injection of various substances into the circulation was due to the increased blood-pressure caused by these substances. He considers that there is no valid evidence in support of the hypothesis of a specific toxaemia caused by the drugs, nor of local anaemia resulting from occlusion of the vasa vasorum. It is noteworthy that the rabbit is so far the only animal which has yielded positive results, Fischer having conducted experiments on dogs and Erb on monkeys with negative results. It can only be concluded that the rabbit's aorta is especially susceptible to sudden internal stretching such as would be produced by mechanical compression of its lower end, or by adrenalin causing a resistance to the outflow from the aorta accompanied by a great rise of blood-pressure. Strain thus produced on the muscular elements of the aorta and occurring a sufficient number of times causes then a necrobiosis of the muscle-fibres followed later by a deposition of lime salts in the middle coat. It may be asked, what has caused this morbid condition in the muscle-fibres? Is it the effect of the mechanical stretching on the muscle-fibres or vasomotor nerves supplying them *alone* (trauma), or is it this factor in conjunction with others; for example, (*a*) the action of a poison produced in the system or introduced into the system by the experimenter; or (*b*) the interference with the nutritive exchanges by occlusion of the circulation in the vasa vasorum or lymphatic channels? Harvey states that, by successive compressions of the aorta, he can produce the same arteriosclerotic changes as are obtained by successive injections of adrenalin, and therefore concludes that adrenalin does not cause the sclerosis by its toxic effect on the muscular fibres. But it appears to me that he has only shewn that internal stress alone is able to produce arterial degeneration, and that because he does not find any changes in the vasa vasorum, he is not justified in excluding the possibility of a failure of nutrition caused by distension of the aorta obliterating the lymph-irrigation channels in the middle coat.

Gouget, who has made a special study of arteriosclerosis, thus describes the lesions produced by injection of adrenalin. " They occupy especially the aorta in the form of whitish gauffered or parchment-like plaques with thinning of the wall, or of chondroid or calcified plaques. In places there may be small cupuliform aneurysmal dilatations. Histological examination reveals degeneration and rupture of the elastic and muscular fibres followed by calcification and thickening, more or less marked, of the intima. These lesions are not always limited to the aorta ; they *may* be observed in other large arteries (carotids and iliacs) ; and even certain visceral arteries, such as the mesenteric, renal, and pulmonary, may shew sclerosis. The heart is often more or less hypertrophied." Stürli has determined these lesions with methylamino-acetopyrocatechin or adrenalin produced synthetically, and, contrary to the experience of others (Klotz, and Roger and Gouget), has obtained sclerosis as well as hypertrophy of the heart with pyrocatechin.

As a result of the experimental production of arteriosclerosis by adrenalin, attention has naturally been directed to the condition of the suprarenals in persons found after death to be the subjects of atheroma. Roger and Gouget described considerable hypertrophy of the suprarenals in a case of experimental aortic sclerosis due to lead intoxication, and Bernard and Bigart have since found histological signs of capsular proliferation in animals poisoned by lead. Since then Josué and Bernard, Kolisko, Manicatide and Jianu, Widal and Boidin have found these same modifications of the suprarenals in the subjects of atheroma ; Sabrazès and Husnot have observed them in 386 necropsies on old people with atheroma. In 70 successive necropsies at Claybury Asylum the suprarenals were carefully weighed, tested chemically and physiologically, and microscopically examined by Prof. Halliburton and myself. We were unable to associate the marked differences of results obtained with any form of mental disease, nor can I, after careful examination of the notes referring to the kidneys, heart and vessels, in any way associate differences in the weight and condition of these glands with arteriosclerosis or atheroma. There was more often atrophy than hypertrophy, especially in old people with vascular disease. Attention has also been drawn to the relative frequency of suprarenal adenomas in interstitial nephritis (Pilliet, Letulle, Oppenheim, Aubertin and Ambard, Vaquez, Lemaire, Froin and Revet, Parkes Weber). Boinet has remarked that suprarenal hyperplasia of the atheromatous is more pronounced in cases of concomitant interstitial nephritis. Moreover, Cesari and Parrisset have observed in the bovine species several cases of aortic atheroma with atheroma of the vena cava strictly limited to the openings of the suprarenal veins. Brooks and Kaplan describe focal necrosis in the arteries of a woman who in the course of three years received 2000 intramuscular injections of adrenalin (10 to 120 minims of 1 in 1000 solution) for asthma.

In the light of these observations certain authorities have come to consider arteriosclerosis, or at least atheroma, as a result of the exaggerated

function of these glands due itself to the action of different physical, nervous, toxic, or infectious causes that have been described. Many objections, however, can be raised to the view that arteriosclerosis is caused by hypertension of suprarenal origin. Many authorities, especially in Germany, dispute the identity of the arterial lesions produced by adrenalin with atheroma as it occurs in the human subject. Gouget, however, states that the proliferation of the endarterium, the hyaline degeneration, and the calcification that adrenalin determines are the fundamental lesions of atheroma ; further, he has in this way produced a sclerosis of the arterioles which presents no difference from ordinary arteriosclerosis ; and lastly, the lesions which adrenalin produces are absolutely similar to those which certain microbial toxins produce. In this connexion it is of interest to note the experiments of D' Amato, which, if confirmed by subsequent workers, are of great importance. He found that if dogs were fed for a long period of time with the products of putrid flesh an atheroma of the aorta is produced " which macroscopically reminds one of human atheromatosis ; histologically the lesions consist of inflammatory and degenerative areas of the wall of the aorta affecting especially the adventitia and media (hyperaemia, haemorrhages, small-celled infiltration, hyaline necrosis, destruction of muscular and elastic fibres, calcareous deposits, and hyperplasia of the intima)." He found that repeated subcutaneous injections of urate of sodium into rabbits produced necrosis of the muscular and elastic fibres of the aorta, pulmonary artery, inferior vena cava, and heart-muscle. He also obtained changes with ergot and sclerotic acid. But he remarks that in all these experimentally produced scleroses in animals there is not complete agreement with the changes met with in human atheroma. His researches, however, support the conclusion that poisonous substances continually entering the circulation damage the vessel-wall not merely by the effect of high pressure and strain but also by direct toxic influence on the muscular tissue, otherwise how can the changes in the heart-muscle, pulmonary artery, and vena cava be explained ?

Returning, however, to the question of suprarenal hyperplasia in atheroma, is there a hyperplasia of the active vaso-constricting portion of the gland—the medullary substance ? According to my experience there is more often a destruction.

How far these arterial lesions produced experimentally in animals can be applied to human pathology is as yet undetermined. They apparently shew that high blood-pressure plays an important part in the production of arterial sclerosis, not only when the strain is continuous but also when there are sudden strong accessions of hypertension followed by hypo-tension, conditions submitting the arterial walls to a kind of gymnastics. Likewise, arteriosclerosis affecting a number of vessels must lead to an in-crease of arterial pressure which, reacting on the heart, causes hypertrophy ; the two are progressively reciprocal, and although the cardiac hypertrophy is a necessary physiological compensation to overcome the increasing peri-pheral resistance, yet the high arterial pressure which it engenders leads

to an increasing strain upon the walls of the aorta and arterio-capillary system generally. The arterial lesion may then be both the cause and effect of the hypertension. But on the other hand, certain cases of arteriosclerosis, notably of the diffuse form, may be explained by the direct action of the poison. From the histological point of view the experimental lesions are considered to approximate most closely with the medial type of human arteriosclerosis, especially with that, described by Mönckeberg, which affects mainly the peripheral vessels and is associated
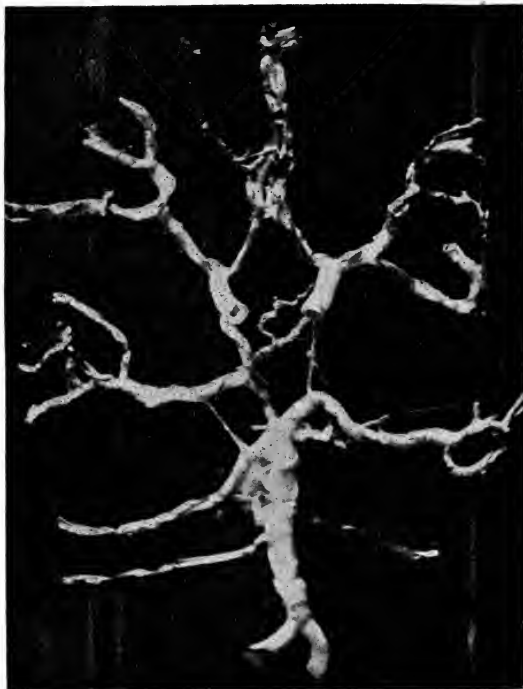
Fig. 79.—Extreme atheroma of the vessels forming the circle of Willis and their principal branches; from a man aged fifty-four suffering from dementia.

with the deposition of calcium salts in the middle coat. Considered from the clinical aspect, sufficient evidence has not yet been adduced to prove that the degenerative vascular changes produced in rabbits by the experimental methods so far described are identical in nature and origin with those found in man.

Symptomatology.—The clinical history varies in every case according to the organ which suffers most and suffers earliest. If it be admitted that in many instances the disease may and does start in defective metabolism and altered quality of the blood, and that this is antecedent to the production of the organic changes in the vessels, and an important

factor in it, then it must also be admitted that there is a stage in which the disease, if recognised, may be prevented or delayed by prophylactic measures.   Mahomed shewed that before the appearance of albumin in the urine in scarlatinal nephritis there is a rise in blood-pressure (pre-albuminuric stage).   Huchard, Traube, and other authors consider that there is a prodromal curable stage of arteriosclerosis ; a stage of toxaemia causing spasm of the arterio-capillary system, increased peripheral resistance, increased functional activity of the heart, and increased pressure in the arteries, but not necessarily changes in the vessel-walls.   The toxaemia may produce headache, drowsiness, morning fatigue, and inaptitude for work, coldness of the extremities, noises in the ears, migrainous or neuralgic attacks, which, together with the high-pressure pulse and accentuated second sound of the heart occurring in a man of middle age who lives well, should always suggest premonitory symptoms of arteriosclerosis.   Dr. Haig asserts that in these cases there is excess of uric acid in the urine.   It is probable that the toxic agents are many and various, and arise from defective nitrogenous metabolism ; possibly, as suggested by Bouchard, they may consist of ptomaines and leucomaines absorbed from the alimentary canal and imperfectly dealt with by the liver.

Under the name *senile plethora* Sir Clifford Allbutt has drawn attention to such irregular and indefinite perturbations of health occurring in persons on the farther side of middle life, the nature of which is indicated by persistent elevations of arterial blood-pressure.   This " hyperpiesis," as he names it, may persist for years, especially if untreated, and may never be associated with renal disease, though both maladies may arise, no doubt, from the same or like causes.   Sir Clifford Allbutt has watched many individual cases of this kind over many years, years of more or less persistent ailment, insomnia, cerebral confusion, despondency, and nervousness, but not necessarily of danger to life.   Most of these cases, he tells us, are remediable by deobstruent means.   And, although in all the condition tends to recurrence, yet in the less inveterate each recurrence is less obstinate to treatment, and recovery may be anticipated.

When there is sclerosis of the arteries the disease must necessarily be progressive and cumulative ; as the vascular degeneration progresses, the nutrition of the body generally and certain organs in particular suffer in their order ; not only do the muscular and elastic tissues of the arteries undergo degeneration, while fibrous tissue takes their place, but the parenchyma of organs and tissues likewise atrophy, and are replaced by fibrous overgrowth.   Thus the process of decay extends in all directions ; and to the symptoms of arterial degeneration are added those of failing or perverted bodily nutrition, with the special symptoms attaching to impaired function of the several organs engaged.   Yet, as pointed out by Sir William Gull, certain types of the disease may be constructed.   In one case cerebral symptoms, in another cardiac, in another renal symptoms predominate ; or again, bronchitis and emphysema may first bring the patient to the physician and lead to the recognition of the general character of the disease.

Not only are the functional disturbances most variable, but also the physical signs obtained by examination of the heart and of the arteries accessible to our investigation present marked differences from one case to another. They may even be almost completely absent when the arteriosclerosis is localised in certain organs, *e.g.* the brain.

We shall now consider the physical signs and functional disturbances of arteriosclerosis.

· *External Appearance.*—The subject of arteriosclerosis frequently presents the appearance of being prematurely aged, and there may be a well-marked arcus senilis. He is often anaemic, sallow, and emaciated; the skin is dry and hangs loosely, lacking the warmth of a good circulation, and often presenting a greyish-yellow or earthy appearance. Sometimes, however, there is nothing characteristic in the general bodily condition; the patient may be pale, flabby, stout, or even obese, and if of a gouty diathesis he may have a high-coloured complexion. It is remarkable how rapidly this disease may progress. Prof. Osler, indeed, says: "I have known the peripheral arteries to grow old and stiffen in a couple of years."

Physical Signs.—In most cases of arteriosclerosis we find a combination of the signs of thickened arteries, high-pressure pulse, hypertrophy of the heart and frequently indications of renal sclerosis, as evidenced by polyuria and nocturnal micturition of a low specific gravity urine which may contain a trace of albumin and a few hyaline casts. The increased pulse tension and the signs of cardiac hypertrophy may be looked upon first as compensatory, and are consistent with fairly good health, provided that complications and accidents do not arise; but as experiment and clinical observation shew, persistent hypertension leads to a progressive stiffening and loss of elasticity of the arteries, and sooner or later serious symptoms arise, according to the vascular area which is most affected. It must, however, be remembered that arteriosclerosis is not always associated with cardiac hypertrophy and high tension of the pulse. The blood-pressure may be normal, or even below normal, and whereas in the former case of high tension, arteries, especially in the brain, are liable to rupture and cause serious symptoms or death from apoplexy, a low-pressure pulse and failing circulation, combined with disease of the arterial walls, may be associated with thrombosis and cerebral softening.

Cardiovascular Symptoms.—The usual signs of cardiac hypertrophy are generally present, although all of them may not be demonstrable. They are more marked as a rule in cases in which there is much renal sclerosis and aortitis. In advanced cases the apex-beat may be an inch or more outside the nipple, and there is an extension of the cardiac dulness to the left on percussion. The impulse is heaving and forcible. These signs, however, are frequently masked by hypertrophous emphysema. On auscultation the first sound may be heard exaggerated in intensity, and sometimes there is a "bruit de galop." The second sound at the base and over the aortic cartilage is usually accentuated and often clear and ringing. According to Friedmann it may be heard over the back in a

line proceeding from the angle of the scapula to the spinous process of the 7th dorsal vertebra. The accentuation of the second sound indicates aortic hypertension, but as we have said, arteriosclerosis may occur when the tension is normal or subnormal, consequently in these cases the second sound is not accentuated. It is not uncommon to find the second sound replaced by a murmur when dilatation of the ascending aorta or damage to the cusps has led to insufficiency of the aortic valve. It has been pointed out that the cusps of the mitral valves may be the seat of sclerosis, consequently a mitral murmur may be present.

Frank and Curschmann have asserted that there is an exaggeration of the normal deviation of the apex of the heart towards the left axilla when the patient is made to lie on the left side. They consider this a sign of elongation and dilatation of the aorta caused by the morbid process. It is, however, of doubtful value, for what the aorta may gain in length by the sclerotic process it would lose by its failure in elasticity. It is said that in a dilated aorta an increase of the breadth of aortic dulness of the ascending aorta can be demonstrated by percussion, which would shew an extension of the dulness beyond the right border of the sternum. It is doubtful, however, whether this could be determined in many cases; x-rays are much more valuable for demonstrating aortic dilatation. Sometimes a painful sensation may be experienced by pressure of the finger into the left second intercostal space; pressure on the abdominal aorta is said to cause pain in certain cases of aortitis. A valuable clinical indication of dilated aorta is the presence of palpable pulsation in the suprasternal notch, owing to dilatation of the arch, and the right subclavian artery, which normally lies behind the clavicle, may be raised above it and be felt.

Peripheral Arteries.—On inspection, especially in these subjects, the superficial arteries, such as the temporals, brachials, femorals, and radials, are visible and tortuous. They may be rigid, and in advanced cases the calcified arteries feel like a pipe-stem, the trachea of a bird, or beaded like a rosary. The radial, from its situation, offers especial facilities for the investigation of the arterial wall and pulse. Owing to its rigidity the artery can be rolled under the finger; the pulse may be of normal frequency, increased in frequency, or slow, even very slow. It is sometimes full and compressible, more often hard and incompressible. It may not be equally felt on the two sides. A sphygmogram shews (1) a sudden percussion-stroke, fairly high, generally straight and vertical, not infrequently jerky or slightly oblique. (2) In place of the usual acute summit to the percussion-wave, it is rounded, or there is a long horizontal plateau. This can be explained by the fact that the artery, having lost its elasticity, is readily distended, but does not react immediately to the force of distension. It is probably also due to the resistance to the outflow from the arterial system. (3) The artery contracts slowly, consequently there is a prolonged oblique line of descent; the dicrotic wave, which depends on the elastic recoil of the aorta and great vessels, is in a great measure obliterated. Arterial hypertension is an early and a warn-

ing symptom, and according to Potain and von Basch it may constitute the only appreciable sign pointing to sclerosis of the arterioles, which eventually leads to sclerosis of the arteries. This hypertension may be measured by the sphygmomanometer, of which there are several forms, and in spite of much difference of opinion in regard to the constancy of hypertension in arteriosclerosis, it is undoubtedly a valuable diagnostic sign, provided that arteriosclerosis is not excluded because there is *not* hypertension ; for it is only necessary to remember that hypertension cannot exist where there is cardiac insufficiency. Many authorities consider that the pressure would remain normal in the great majority of cases, were not renal disease so common an association.

The condition of the small arteries can also be studied by examination of the fundus oculi. They appear thickened, and shew greyish-white streaks; veins passing over these thickened arteries appear kinked. According to Raehlmann, these alterations of the retinal arteries can be observed in quite one-half of the cases of arteriosclerosis, and Thoma and other authorities recommend the use of the ophthalmoscope as a valuable means of early diagnosis.

Functional Disturbances.—Among the most important of the functional disturbances are transitory attacks of vaso-constriction. They constitute crises of hypertension, and have been described by Pal under the name of *vascular crises*. These will be referred to in describing the disturbances occurring as a result of aortitis, arteritis of the limbs, and of visceral arteriosclerosis. Thoracic aortitis when diffuse may be manifested by a sensation of weight in the region of the sternum, at certain moments exaggerated into an anginal attack, palpitations, and dyspnoea, provoked or increased by physical stress, and crises of pseudo-asthma ; these symptoms, however, are often due to associated disease of the heart and other organs.

Pseudo-gastralgic attacks, consisting of deep-seated pain of variable character below the umbilicus, occurring in crises of a few minutes to one hour or more, often repeated, and accompanied sometimes by vomiting, and often by distension, have been associated with abdominal aortitis. The attacks are not related to the taking of food except it be indigestible or excessive in quantity, but they occur as the result of violent emotions, sexual excess, and physical stress.

When the arteries of the limbs are affected the patient may complain of formication and disagreeable pains or sensations in the limbs. Pain in the calves, the soles of the feet, around the nails, cramps, coldness with pallor of the extremities, up to intermittent claudication, form together a sign of especial diagnostic importance. The horse, as a result of the narrowing of the lumen of the abdominal aorta, or of a large artery of one of the hind limbs, may exhibit signs of intermittent claudication. The animal while at rest, or even when trotting at a moderate pace, shews no signs, but the blood-supply is insufficient to permit it to continue any rapid trot, and it soon goes lame, drags the limb, and sometimes falls, without being able to get up again for some time. The limb

is found to be cold and without appreciable arterial pulsation. François, and especially Charcot, then Sabourin, shewed that exactly the same complications may arise in man, and more recently Goldflam and Erb have described this intermittent claudication. Although particularly frequent in diabetes, intermittent claudication is not in any way especially related to this disease, but to an associated chronic arteritis. While at rest the subject of intermittent claudication does not experience any inconvenience, and, at first, walking can be accomplished without any symptoms, but in a few minutes to half an hour one or both the lower limbs become the seat of a sensation of painful feebleness. Sometimes it is a painful feeling of heaviness, of cold, or of a burning, localised sometimes in the toes, sometimes in the soles of the feet, the instep, the calves, or the thighs. If the patient continues to walk, the pain becomes almost insufferable, and he is obliged to stop or to slow the pace ; he takes short steps and drags the leg, and finally a painful cramp may seize the limb and compel inactivity. On examination the limb is found to be cold, pale, and bloodless ; sometimes, however, it has a blue marbled appearance. Sensibility is more or less blunted. The tendon reflexes may be normal, sometimes they are exaggerated. The arterial pulsations in the posterior tibial and dorsalis pedis may be imperceptible or completely abolished. After some minutes' rest, all the symptoms disappear, and the patient is able to walk again, but only to be shortly arrested by the same symptoms. With each attack that occurs, the interval between them lessens in time, and the necessity for rest increases. Sensory disturbances may even come on at night while the patient is at rest in bed. Although it is usually the lower limbs that are affected, the upper limbs are not exempt.

Intermittent claudication is not only a sign of arteriosclerosis, but it is a forerunner of gangrene, often designated *senile gangrene*. When signs of claudication occur it is seldom that more than a few years elapse before gangrene ensues, the exciting causes being injury or cold, often accelerated by some constitutional disease, such as diabetes, gout, and heart-disease with failing circulation. When gangrene is supervening the pains are severe, burning, lancinating and tearing, exaggerated by the least movement. The skin is cold and almost insensible ; at first it is pale, and then scattered dusky patches of cyanosis appear, and finally the whole of the toes and the foot become black. Such was the course of events in the case shewn in Fig. 2, Plate II., in which gangrene of both feet occurred as a result of obliterative arteritis. Occasionally, as in this case, the nose may become gangrenous. I have seen the tips of the ears and the upper limbs affected, and it is said that the penis may suffer likewise.

Of all the organs and structures of the economy the *nervous system*, by virtue of its manifold and important functions, offers the most varied complex functional disturbances in arteriosclerosis. The whole cerebral nervous system, or portions of it, may suffer from partial or complete ischaemia, and according to the localisation of the arterial change and

the extent and severity of the morbid condition will groups of symptoms arise. In discussing the symptomatology of syphilitic arteritis allusion has been made to a local or widespread arterial change, and I am convinced, from long experience and investigation, both macroscopic and microscopic, that syphilis is the underlying factor in a large proportion of the cases of cerebral arteriosclerosis arising between forty-five and fifty-five in persons of both sexes who have acquired syphilis in earlier life. Cases may shew some arteries with sclerosis indistinguishable from arteriosclerosis, and other arteries with an endarteritis indistinguishable from a recent syphilitic arteritis. The sclerotic change may be universal in the brain and spinal cord, affecting not only the larger arteries, but also the arterioles and capillaries, and even the veins. The vessels, although thickened, are, owing to the degeneration of the muscular coat, brittle and liable to the formation of small miliary aneurysms and rupture. Besides, scattered about in the cortex, small haemorrhages may be found in the perivascular sheaths and the nervous substance of the basal ganglia, medulla oblongata, and spinal cord. In three well-marked examples of arterio-capillary fibrosis in the subjects of chronic lead poisoning I found hyaline thickening of the arterioles, capillaries, and veins with scattered multiple miliary haemorrhages (*vide* Figs. 2, 3, 4, Plate III.). Again, patches of softening, varying in size from gross lesions to those only recognisable by a hand lens or the microscope, may occur. It can therefore be readily understood that the whole brain or portions of the brain may, owing to the diminution of the vascular area, loss of elasticity, and therefore of adaptability of the arteries, suffer with an insufficiency of blood-supply, and various disturbances of function may arise which may be transitory or permanent, according to the extent of the disease and the possibility of re-establishment of the circulation. Among the cerebral troubles due to circulatory disturbances may be mentioned heaviness and oppression, headache, irritability of temper, migraine, apathy, drowsiness and inability to concentrate the attention without a sense of effort and weariness, defective memory, sometimes transitory attacks of amnesia, aphasia, speech embarrassment, and hemiparesis. Disturbances of sleep are common ; not that there is insomnia, but the sleep is uneasy and less refreshing than formerly. Then the patient is unusually susceptible to the action of alcohol and tobacco, which excite and aggravate many of the cerebral troubles. In persons of insane temperament attacks of maniacal excitement or depression are especially liable to arise, accompanied not infrequently with hallucinations and delusions, so that it is not unusual, especially if the patient suffers with slight apoplectic or epileptiform seizures, followed by a transitory hemiplegia and a slight or marked progressive dementia, to look upon the case as one of general paralysis. In quite a number of these cases admitted to asylums there is a history of the patient having, owing to worry, distress, and mental depression, taken to the use of stimulants in quantities which would be moderate to a normal individual. In others there is a history of prolonged abuse of alcohol. As a rule the delusions from which these patients suffer relate to persecution rather than grandeur

and exaltation, and the dementia is not nearly so pronounced as in general paralysis ; moreover, paralysis when it occurs is usually coarse. Not infrequently there are signs of pseudo-bulbar or true bulbar paralysis with spasmodic laughing and crying. Sometimes there is progressive muscular paralysis with fibrillar twitchings. In certain cases of bulbar ischaemia attacks of Cheyne-Stokes breathing occur at intervals, or they may be habitual for months, especially during sleep. It must be remembered, however, that this symptom may be due, at any rate partially, to cardiac or renal insufficiency. Again, vertigo is a common symptom of cerebral arterial sclerosis ; it occurs especially when a sudden change is made from the recumbent to the upright position. In its slight early form it may not amount to more than a lack of the feeling of stable equilibrium, often increased by apprehension. The vertigo is not infrequently associated with *tinnitus*, and it is necessary to eliminate the possibility of the symptoms being due to Menière's disease. A permanent slow pulse, with attacks of giddiness, syncopal, epileptiform or apoplectiform seizures, a syndrome which was long considered to be especially diagnostic of sclerosis of the bulbar arteries, is now considered more probably to point to affection of the auriculo-ventricular bundle of His (Stokes-Adams disease, *vide* p. 130). Sometimes instead of bradycardia there may be paroxysmal tachycardia, with or without arrhythmia (*cf.* p. 553). Vertigo of labyrinthine origin and the syndrome of Menière's disease may occur as the result of arteriosclerosis, or the vertigo may be the result of sclerosis of the cerebellar arteries.

The Spinal Cord. —Certain French neurologists, notably Déjerine, Sollier, and Grasset, have described a syndrome due to intermittent contraction of the arteries of the spinal cord, and the group of symptoms closely resembles that in intermittent spasm due to arteriosclerosis of the lower limbs above described, the same motor and sensory troubles, sometimes unilateral, being set up by walking and disappearing when at rest. But according to Déjerine the pulsation persists in the arteries of the feet, vasomotor disturbances are absent, the deep reflexes, already exaggerated during rest, are still more so during the attack, and the extensor reflex of the toes is present ; lastly, bladder symptoms in the form of frequent desire to pass water, as well as genital symptoms, are constant. This claudication often ends in chronic spasmodic paraplegia. The arteritis which gives origin to it is almost always syphilitic. Besides this syndrome, Grasset considers that there is an intermittent spasm affecting the posterior columns and giving rise to symptoms resembling tabes, and consisting in a feeling of constriction of the thorax or abdomen, simulating an anginal or gastric crisis, besides transitory anaesthesia and paraesthesia. Grasset even goes farther, and assumes that the paroxysms of lightning pains and visceral crises may be due to intermittent spasm of the spinal cord.

Whatever may be the explanation of angina pectoris due to organic disease, it is almost exclusively met with in the subjects of arteriosclerosis, and especially in cases of aortic disease.

Coronary Arteriosclerosis. —Occlusion of the trunk of a coronary artery is sometimes the only lesion to be found in cases of sudden death, even

in the absence of all evidence of previous attacks of angina. Coronary arteriosclerosis is often associated with myocardial degeneration, but it may directly give rise to a patchy fibrosis owing to thrombotic occlusion of a large branch causing necrosis of the muscle and fibrous substitution. Sudden death in the so-called fibroid heart may really owe its causation to coronary disease.

Renal arteriosclerosis is indicated by the passage of larger quantities than normal of pale urine of low specific gravity, which may or may not contain albumin and hyaline casts. The patient usually has to rise several times during the night to pass water. It is associated with the signs of cardiac hypertrophy and high-tension pulse. The relation of this condition to cerebral haemorrhage has been already dealt with, but such patients are liable to attacks of *epistaxis*, and I have several times seen cases of fatal apoplexy which was preceded by several attacks of bleeding from the nose. An early symptom may be dyspeptic trouble, and various authors have associated different gastro-intestinal disturbances with arteriosclerosis.

In conclusion, it is desirable to point out that headache, weariness, irritability of temper, defects of memory, mental depression, morbid apprehension, lack of power of attention, and exhaustion following mental or physical exertion, may be due to neurasthenia, but when occurring between forty-five and fifty-five years of age they may be the initial symptoms of arteriosclerosis.

Diagnosis and Prognosis.—One of the principal difficulties in diagnosis arises from the fact that a number of disturbances of functions met with in arteriosclerosis may occur as a result of ischaemia due to spasmodic vasomotor constriction, occurring in neurasthenia and hysteria ; *e.g.* intermittent spasm, cold hands and feet, pseudo-asthmatic conditions, slow pulse, rapid pulse, arrhythmia, vertigo, noises in the ears, headache, fainting attacks and fits. It must be remembered, however, that the neuroses in which these conditions are met with, for example, hysteria and neurasthenia, may occur in individuals who are at the same time the subjects of arteriosclerosis. Although physical signs of arteriosclerosis are usually observable, they are not determinable when affecting only the viscera, for example, the brain. Still a careful investigation of the family history, the past personal history, and the signs and symptoms presented by the patient will generally enable a diagnosis to be made with a fair degree of certainty. The prognosis depends very much upon the stage of the disease, for in the early period of high arterial pressure very much may be done, by hygienic measures, to prevent and arrest the disease. It also depends upon the localisation of the disease ; thus, when there is reason to suspect affection of the coronary arteries the prognosis is especially grave. Again, although renal sclerosis is generally slow and insidious in origin, grave complications are very likely to supervene and the patient may succumb to uraemia, intercurrent maladies, or cardiac insufficiency. If there have been any signs pointing to cerebral arteriosclerosis, the prognosis must be bad, for it is probable that all the cerebral

vessels are more or less affected, and sooner or later some grave complication, such as thrombosis or haemorrhage, will occur. The Wassermann serum reaction might prove useful as an aid to diagnosis in relation to treatment in some cases of arteriosclerosis.

Treatment.—All conditions which may interfere with the nutrition of the body must be avoided. Mental and bodily strain and excessive business activity should be forbidden. Tepid baths, friction, massage, and rational daily exercise are to be enjoined. Excess of meat, especially of red meat, is to be avoided. Fish dinners once or twice a week, or even a milk diet, may be recommended in some cases. If there be a gouty tendency, or the disease be of the renal type, this is especially necessary ; as is also the avoidance of beer and certain wines, such as sweet wines, champagnes, and burgundies. Beef-tea, meat extracts, and essences, so frequently employed as "supporting measures," should be sparely used or avoided. Certain mineral waters are very useful, for example Carlsbad, and a course of treatment at one of the many watering-places is very often beneficial—not merely by the drinking of the waters, but by the regular mode of life, diet, and exercise. Each patient must be treated, however, according to the nature and relative prominence of the symptoms. Many people suffering from arteriosclerosis have lived a regular life and have habitually eaten and drunk in moderation, and it often happens that we are unable to make much improvement in their diet ; for a palate which has not been vitiated by indulgence in excessive eating and drinking is a good guide to a suitable dietary. If the patient enjoys and thoroughly chews his meals of simple well-cooked food, taken at regular hours and moderate in quantity, the normal processes of digestion will take place and gastro-intestinal fermentative processes will be as efficiently avoided as by limiting the patient to any particular kinds of food or restricted (vegetarian) diets. The temperament and idiosyncrasy of the individual must be studied in prescribing a diet ; thus, milk, by virtue of its being a perfect food, introduces into the body a minimum of toxic substances, diminishes bacterial growth and intestinal fermentation, and acts as a natural diuretic. Moreover, when taken in small quantities at a time, it lowers arterial pressure. For all these reasons, therefore, it is indicated as a staple article of diet, yet experience shews that in some cases it cannot be tolerated at all, and in others milk has to be supplemented with other articles of food, such as fish, eggs, and light farinaceous foodstuffs. There can be no doubt as to the value of milk as a constant source of nutrition in all cases of arteriosclerosis with renal insufficiency. If the signs of renal insufficiency disappear, a return to a moderate meat diet is often desirable. It is essential that there should be a regular daily evacuation of the bowels. If the patient cannot abstain from alcohol and tobacco, it is desirable that he should restrict himself to a small quantity of whisky, well diluted with mineral water, at lunch and dinner. He should be advised not to carry smoking materials, not to inhale the smoke, avoid foul pipes and strong cigars ; and to limit his indulgence to a cigarette, a mild cigar, or a pipe after meals.

Clothing — Climate.—Persons suffering with arteriosclerosis are the subjects of deficient circulation and heat-production. They should therefore be warmly clad, wear woollen underclothes, and avoid a cold and damp climate. Life in the open air in a warm climate by the sea is indicated, and a resort should be selected which is sheltered from cold winds and where the temperature is fairly constant. Arteriosclerotic patients, especially if they present any signs of cardiac insufficiency, should avoid high altitudes and health resorts with an elevation of 2500 feet; even altitudes less than this may be found undesirable for residence, as the patient may complain of insomnia or dyspnoea.

Various authorities have recommended hydrotherapy in arteriosclerosis, on the ground that by acting upon the skin nutrition is stimulated. There can be no doubt that cold baths increase arterial tension and warm baths the converse. As a rule the subjects of arteriosclerosis should avoid cold baths, hot-air and vapour baths; but tepid baths, simple or alkaline and not prolonged, may be recommended, followed by friction or massage in certain cases.

Carbonic acid gaseous baths have been recommended, and some authorities proclaim the favourable results in angina pectoris obtained by the use of the baths at Nauheim. This treatment has been much discussed; according to certain authorities the work of the heart is diminished by dilating the peripheral vessels, according to others the result is a general rise of arterial pressure. Byrom Bramwell and Huchard are of opinion that these baths only cure the anginal cases of neuropathic origin; in cases of organic angina pectoris, of marked cardiac insufficiency, and of renal sclerosis, they are contra-indicated, as they may lead to serious complications.

Therapeutic Treatment.—The iodine compounds and notably the iodides are the most useful therapeutic agents for arteriosclerosis. It is generally assumed that the iodides lower arterial pressure. No drug and no treatment will restore elasticity to the arteries already stiffened by calcareous deposit, but it is probable that the iodides can prevent or delay the extension of the sclerosing process. It is supposed that a combination of potassium and sodium iodides is useful, for it is stated by Gouget that the former has a more powerful resolving action and the latter a more marked hypotensive action. Small doses can be given, 3 to 5 grains simultaneously, or alternately, except in cases in which syphilitic aortitis or arteritis is suspected, then much larger doses are indicated. In certain individuals coryza is produced even with small doses. Other preparations of iodine may be used when the iodides appear inactive or are not tolerated. Tincture of iodine in doses of a few drops in a claret-glass of water before meals has been recommended, also the many albuminoid and fatty compounds of iodine recently introduced have been employed. Among the former may be mentioned iodone, iodalose, and tiodine, and among the latter iodipin, which can be used in the form of subcutaneous injection; but with all these organic compounds the elimination of the iodine is slower than with the iodides. Some authorities have used

thyroiodin on the assumption that thyroidectomy not only increases the viscosity of the blood, but also, according to the experiments of Rosenblatt and v. Eiselberg, leads to the production of patches of arteriosclerosis. To whatever iodine compound a preference is given, to obtain results it is necessary to commence the treatment in the early stages and to continue it for a long period with intervals of cessation ; thus, the drug may be administered for three weeks in every month. The employment of the iodine preparations is justified empirically rather than by experiments on animals. They are contra-indicated where there is renal disease with albuminuria and in cases of cardiac failure. Liq. arsenicalis in 3 to 5 minim doses may be given with the iodide or in the intervals when the iodide is not administered ; its use is indicated in cases presenting anaemia or signs of neurasthenia.

Amyl nitrite, trinitrin, or erythrol tetranitrate should be administered for the relief of symptoms arising from paroxysmal attacks of vasomotor spasm, *e.g.* angina pectoris. Where an immediate relaxation of the arterial system is required inhalation of amyl nitrite is most valuable, as its action is most certain and rapid, but it is also the most transitory. Trinitrin and nitrite of sodium have a more lasting hypotensive action, but they take five to ten minutes before the full effect is produced. The action of the former is said to persist for two to three hours, and the latter for four to five hours, but the effect produced varies somewhat in different individuals. When cardiac failure supervenes, and there is incipient regurgitation, we must resort to cardiac tonics, such as digitalis, strophanthus, or spartein ; or strychnine and caffeine may be used, especially if oedema of the lungs and dropsy set in. Sansom recommended the combination of the cardiac tonic with trinitrin or iodides. Ten-minim doses of tincture of digitalis may be given with 1-drop doses of the solution (1 to 100) of nitroglycerin. Venesection has been employed with success in some cases of cardiac insufficiency with dyspnoea and lividity. It is very desirable to explain to the patient, in the less hopeful cases, that he is suffering from a disease the symptoms of which can only be alleviated, and that alleviation depends almost entirely upon intelligent assistance on his own part in following out implicitly the rules of the physician. The prognosis and treatment of the numerous morbid conditions under which arteriosclerosis may be manifested, for example, renal disease, lead poisoning, pulmonary fibrosis and emphysema, cardiac degeneration, aneurysm, cerebral haemorrhage, migraine, and psychical disturbances, are discussed in other portions of these volumes, and for further information the reader is referred to the special articles upon these subjects.

<div style="text-align: right">F. W. MOTT.</div>

REFERENCES

1. ADAMI. " On the Relationship between Inflammation and Sundry Forms of Fibrosis," The Middleton-Goldsmith Lecture, 1896.—2. ALLBUTT, Sir CLIFFORD. " Senile Plethora," *Trans. Hunterian Soc.*, 1895-96.—3. VON BASCH. " Ueber latente Arterio-Sclerose, etc.," *Wiener med. Presse*, Nos. 20, 23, 27, 30, 1893, and " Die Herzkrankheiten bei Arteriosklerose," Berlin, 1901.—4. BERHEIMER. " Ueber

schweren Veränderung bei hochgrädige Sclerose der Gehirn-Arterien," *Graefe's Arch.*, xxxvii.—5. BOLLINGER. *Pathological Anatomy*, vol. i. p. 28.—6. BREGMAN. " Ein Beitrag zur Angio-Sclerose," *Virchows Jahrb.*, 1891.—7. BROADBENT, Sir WM. *The Pulse.*—7a. BUNTING, C. H. "Formation of True Bone with Cellular (Red) Marrow in a Sclerotic Aorta," *Journ. Exper. Med.*, New York, 1906, viii. 365.— 8. CHARCOT. *Compt. rend. Soc. biol.*, Paris, 1858; *Progrès méd.*, Paris, 1887, and *Bull. méd.*, Paris, 1891, v. 1105.—9. COATS and AULD. "Endarteritis Deformans and Aneurysm," *Journ. Path. and Bacteriol.*, Edin. and London, 1897, iv. 78.—10. COUNCILMAN. *Trans. Assoc. Amer. Phys.*, 1891, vi. 179—11. CROCQ, *fils.* "Quelques mots sur l'artério-sclérose," *Gaz. hebd. de méd.*, 1892, xxix. 523. —12. "Discussion on the Relation of Renal Disease to Disturbances of the general Circulation, and to Alterations in the Heart and Blood-Vessels," *Intern. Med. Congress*, London, 1881, i. 374.—13. DÉJERINE. *Rev. neurol.*, Paris, 1906, xiv. 341.—14. DICKINSON, H. "Morbid Effects of Alcohol," *Med.-Chir. Trans.*, 1873, lvi. 34 ; and Baillie Lectures. "On the Cardio-vascular Changes of Renal Disease," etc., *Lancet*, 1895, ii.—15. EPPINGER. *Pathogenesis, Histogenesis u. Aetiologie der Aneurysmen*, 1887.—16. ERB. *Deutsche Ztschr. f. Nervenheilk.*, 1898, and *Münch. med. Wchnschr.*, 1904.—16A. FRÄNKEL und MUCH. *München. med. Wchnschr.*, 1908, lv.—17. GIBSON, G. A. "Diseases of the Heart and Aorta," 1898.—18. GOLDFLAM. *Deutsche med. Wchnschr.*, 1895, and *Neurol. Centralbl.*, 1901.—19. GOUGET. "L'artério-sclérose et son traitement," 1907.—20. GRASSET. *Rev. neurol.*, Paris, 1906, xiv. 433.—21. GRASSET et RAUZIER. *Vertige cardio-vasculaire artériel.* —22. GULL and SUTTON. "Arterio-capillary Fibrosis," *Med.-Chir. Trans.*, 1872, lv. 273.—23. HAMPELN. "Ueber Syphilis u. das Aorten-Aneurysma," *Berl. klin. Wchnschr.*, 1894, xxxi. 1000, 1021, 1067.—24. HEGERSTEDT und NEMSER. *Krankhafte Veränderung u. Verschliess.*—25. HELMSTEDTEN. " Du mode de formation des anévrysmes spontanées," *Virch. Jahrb.*, Strassburg, 1873.—26. HOFFMANN. "Ueber Aneurysma der basilar Arterien," etc., *Virch. Jahrb.*, 1894.—27. HOLLIS. "Atheroma," *Journ. Path. and Bacteriol.*, Edin. and London, 1896, iii.—28. HUCHARD. *Mal. du cœur.*—29. JACOBSOHN. "Ueber die schwere Form der Arterio-Sclerose im central Nerven-System," *Virch. Jahrb.*, 1894.—30. JOHNSON, Sir GEORGE. "Relation of Cardio-vascular Changes to Bright's Disease : a Criticism of Arterio-capillary Fibrosis," *Med.-Chir. Trans.*, 1873, lvi. 276.—31. KÖSTER. "Ueber die Entstehung der spontanen Aneurisme u. die chronische Mesarteritis," *Berl. klin. Wchnschr.*, 1875, xii. 323.—32. LAHN. "Ueber Vascularisation der Media u. Intima bei Endarteritis chronica," *Verh. d. Kongr. f. inn. Med.*, Berlin, 1891.—33. LANCEREAUX. *Dictionnaire encyclopédique médicale.*—34. *Idem.* "L'endartérite ou artério-sclérose généralisée," *Arch. gén. de méd.*, Jan.-Nov. 1893.—35. LANGHANS. (Anatomy of the Arteries), *Virchows Arch.*, 1881, xxxvi.—36. LITTEN. "Ueber circumscripte gitterförmige Endarteritis," *Deutsche med. Wchnschr.*, 1889, xv. 145.—37. MACLEAN. *Brit. Med. Journ.*, 1876, i. 283.—38. M'CRORIE, D. "Atheromatous Disease of Arteries," *Glasgow Med. Journ.*, 1892, xxxviii. 110.—39. MEIGS. "Chronic Endarteritis and its Clinical and Pathological Effects," *Med. Rec.*, N.Y., Aug. 24, 1889.—40. DE MUSSY, G. "Étude clinique sur les indurations des artères," *Arch. gén. de méd.*, 1872.—41. PEKELHARING. "Ueber endothel. Wucherung in Arterien," *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, xviii.—42. PRONG. "Stenosis arteriae coronariae cordis," *Virch. Jahrb.*, 1893. —43. PUPPE. *Untersuchungen über der Aneurysma der Brust-Aorta.*—44. RAEHLMANN. xiiie Congrès de Neurol. Allem., Fribourg, 1888.—45. ROKITANSKY. *Lehrbuch der Pathologie.*—46. ROMBERG. "Ueber Sclerose der Lungenarterien," *Deutsch. Arch. f. klin. Med.*, 1891-92.—47. ROGET, GOUGET, et BOINET. "Maladies des artères et de l'aorte," Paris, 1907.—48. ROY. "On the Elasticity of Arteries," *Journ. Physiol.*, 1881, iii.—49. SACHS. "Ueber Phlebo-Sclerose," *Virch. Jahrb.*, 1888.—50. SANSOM. "Diseases of the Blood-Vessels," *Twentieth Century Practice of Medicine*, iv.— 51. SAUNDBY. "Hypertrophy of the Vascular System in Granular Degeneration of the Kidney," *Edin. Med. Journ.*, Oct. 1896.—52. SAVILL, T. D. "Arterial Hypermyotrophy," *Brit. Med. Journ.*, 1897, i. 188.—53. SHAW, BATTY. "Goulstonian Lectures," *Lancet*, 1906, i.—54. STRAUBE. "Ueber die Bedeutung der atheromatösen Arterien-Krankh.," Würzburg, *Virch. Jahrb.*, 1893.—55. THAYER, W. S. "Cardiac and Vascular Complications and Sequels of Typhoid Fever," *Johns Hopkins Hosp. Bull.*, 1904, xv. 323.—56. THOMA. *Pathological Anatomy*, trans. by Alex. Bruce.—57. *Idem.* "Ueber die Abhängigkeit der Bindegewebe," etc., *Virch. Arch.*, 1888, xciii.— 58. *Idem.* "Arterial Elasticity," *Lancet*, 1896, ii.—59. THOMA und KAEFER. "Ueber

die Elasticität gesunden u. kranken Arterien," *Virchows Arch.*, 1889, cxvi. 1.—60. TRAUBE. "Entstehung der Arterio-Sclerose," *Berl. klin. Wchnschr.*, 1891, Nos. 29, 31, 32.—61. TSCHIGAJEW. "The Importance of Muscular Work in the Production of Arteriosclerosis," *Virch. Jahrb.*, 1895. — 62. VIRCHOW. *Cellular Pathology.* — 63. WELCH. *Med.-Chir. Trans.*, 1876, lix. 59 ; *Lancet*, 1875, ii.—64. WILKS and MOXON. *Pathological Anatomy.* **Experimental Arteriosclerosis :** 65. AUBERTIN et AMBARD. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1904, xxi. 175.—66. BAYLARD et ALBARÈDE. *Compt. rend. Soc. biol.*, Paris, 1904, lvii. 640.—67. BENNECKE. *Virchows Arch.*, 1908, cxci. 208.—68. BROOKS and KAPLAN. *Arch. Int. Med.*, Chicago, 1908, i. 329.—69. D'AMATO. *Virchows Arch.*, 1908, cxcii.—70. DIXON. *Proc. Roy. Soc. Med.*, London, 1908, *Therap. and Pharm. Sect.*, 33.—71. ERB, W. (junior). *Arch. exper. Path. und Pharmakol.*, Leipzig, 1905, liii. 173.—72. FISCHER. *Deutsche med. Wchnschr.*, Leipzig, 1905, xxxi.—73. GILBERT et LION. *Compt. rend. Soc. biol.*, Paris, 1889, i. 583 ; and *Arch. de méd. expér. et d'anat. path.*, Paris, 1904, xvi. 73.—74. GOUGET. *L'Artériosclérose et son traitement*, 1907. — 75. HARVEY. *Journ. Exper. Med.*, N.Y., 1906, viii.; *Journ. Med. Res.*, Boston, 1907, xvii.; and "Dissertation on Experimental Arteriosclerosis," *Proc. Roy. Soc.*, 1908.—76. HUCHARD. *Maladies du cœur et de l'aorte.*—77. JOSUÉ. *Presse méd.*, Paris, 1903, xi. 798 ; and 1904, xii. 281.—78. KLOTZ, OSCAR. *Journ. Exper. Med.*, N.Y., 1905, vii., and 1906, viii.—79. LISSAUER. *Berl. klin. Wchnschr.*, 1905, xlii. 675.—80. LOEB and GITHENS. *Amer. Journ. med. Sc.*, Phila., 1905, cxxx. 658.—81. MÖNCKEBERG. *Virchows Arch.*, 1903, clxxi. 141. — 82. MOTT and HALLIBURTON. *Archives of Neurology*, 1907, iii.—83. PEARCE. *Journ. Exper. Med.*, N.Y., 1908, x. 735.—84. PEARCE and STANTON. *Ibid.* 1906, viii. 74.—85. RICKETT. *Journ. Path. et Bacteriol.*, Cambridge, 1908, xii. 15.—86. ROGER, GOUGET, et BOINET. *Maladies des artères et de l'aorte*, 1907.—87. STÜRLI. *Gesellsch. f. inn. Med. und Kinderheilk.*, Wien, 1905.—88. SUMIKAVA. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1903, xxxiv. 242.—89. ZIEGLER, K. *Ibid.* 1905, xxxviii. 229.

F. W. M.

ANEURYSM

By Prof. W. OSLER, M.D., F.R.S.

Definition — History— Classification — Etiology — Pathological Anatomy— Dilatation-Aneurysm—Dissecting Aneurysm—Saccular Aneurysm of the Aortic Arch—Aneurysm of the Descending Thoracic Aorta—Aneurysm of the Abdominal Aorta—Arterio-venous Aneurysm—Diagnosis—Prognosis—Treatment.

Definition.—A tumour containing fluid or solid blood in direct communication with the cavity of the heart, the surface of a valve, or the lumen of an artery.[1]

History.—There is no mention of the disease in Hippocrates. Galen was well acquainted with it, and he described two forms, one from dilatation, in which the tumour was deeply seated, and when pressed upon communicated to the fingers a noise (so that he must have recognised the thrill) ; the other, which arose from wounding of a vessel, was rounded and felt more superficial. He also gave an account of the aneurysm following venesection at the bend of the elbow, and cured one

---

[1] This definition embraces a majority of the conditions to which the term aneurysm is given, but it cannot be said to include simple dilatation of the aorta or of its branches, and the abnormal communication between two vessels.

due to this accident by the application of a sponge and bandages. That the Graeco-Roman profession knew external aneurysm thoroughly is shewn by the brilliant operation of Antyllos (*circa* 55-118 A.D.), which is still recognised in surgical textbooks. With Galen this author recognised two kinds of aneurysm—one from local enlargement of a vessel, the other following an injury, and speaks of the thrill as the distinguishing feature of the latter. In his operation the sac was enclosed between ligatures, and then laid open and emptied. That the modern practice of extirpation of the sac must have been carried out by some of his contemporaries is evident from a criticism which he makes upon this procedure. Practically nothing was added to our knowledge of the subject until the recognition of internal aneurysm by Fernelius, a distinguished French physician of the sixteenth century, who remarked, "that aneurysm likewise happens sometimes in the internal arteries, especially under the breast, about the spleen and mesentery, where the venous pulsation is often observed." In 1555 the great anatomist, Vesalius, was called to Augsburg to see one Leonhard Wolser suffering with severe pains. He found in the region of the back a pulsating tumour, which he diagnosed as an aneurysm and said it was incurable. Two years later the patient died, and an aneurysm of the aorta was found eroding the spine above the diaphragm. The sac had perforated the chest and projected beneath the skin. The patient had had severe haemoptysis. In the letter which Vesalius writes to Gassner, thanking him for sending him an account of the necropsy, he remarks on the frequency of aneurysm and on the varied character of the contents of the sac. He also mentions the case of a woman with an aneurysm in the abdomen, so that to Vesalius belongs the credit of having first described both abdominal and thoracic aneurysms. The first case is in many respects a remarkable one historically; not only was the diagnosis made by Vesalius, but we possess Gassner's very full notes of the necropsy describing the hard, fleshy concretions in the aneurysm; and the comment of Vesalius in reply shews that in his wide clinical and anatomical experience he had become very familiar with the disease. From this time on scattered references occurred in the literature, of which an excellent account is given by Freind in his *History of Physic*. Next to Vesalius, perhaps the most important sixteenth-century writer on the subject was Ambroise Paré, who recognised aneurysm by anastomosis, rupture, erosion, and injury. He was the first to suggest the relation of aneurysm to syphilis, and he describes the noise or blowing sound associated with the tumour, and the frequency of thrombosis in the sac and the occasional calcification. In the eighteenth century a number of important works on the subject appeared. In 1728 there was published the great monograph of Lancisi, the distinguished Roman physician, who recognised the importance of syphilis in the disease, and even spoke of a "venereal aneurysm." Morgagni's famous work, *De Sedibus et Causis Morborum*, 1761, gives an excellent impression of the very full knowledge which the men of that time had of the disease. An account of the symptoms and morbid anatomy of thoracic aneurysm

could almost be compiled from Morgagni's description alone, and we owe to him the knowledge of the Valsalva method of treatment. As already mentioned, Galen had recognised two forms of aneurysm—one in which the vessel dilated spontaneously, and the other which followed injury. Throughout the eighteenth and early part of the nineteenth century the forms and classification of aneurysm were much discussed. Lancisi, adopting the Galenic view, divided aneurysm into true and false, corresponding to our division of spontaneous and traumatic. William Hunter not only made an important contribution to the subject of arteriovenous aneurysm, but he gave us the division into *true*, in which all the coats were dilated, *spurious*, in which one or more of the coats were ruptured and the other or others dilated, and a *mixed* aneurysm, in which the coats were dilated, and subsequently, by a rupture, a true was converted into a spurious aneurysm. A great discussion took place about the mode of origin and classification. Scarpa, whose famous monograph appeared in 1804, disregarding all these divisions, insisted that whatever its situation or its form an aneurysm did not arise by dilatation, but by rupture or corrosion of the internal lining of the muscular coats. He laid special stress upon the importance of the media in maintaining the strength of the vessel. Scarpa did not regard the uniform dilatation of the vessel as aneurysmal, holding that form only to be aneurysm which arises "at some point of the parietes of the arteries from the rupture of their proper coats." The exclusiveness of the views of Scarpa were criticised by Hodgson (1815), who believed that loss of the natural elasticity of the vessel-wall might lead to general dilatation, which he also regarded as an aneurysm. In his *Anatomie Pathologique* Cruveilhier gave an accurate account of the structure of the aneurysm, and in opposition to Scarpa held that the dilatations of all three coats existed at first, and that as the aneurysm grew the inner and middle coats were stretched and there were secondary sacculations. In the great monograph of Rokitansky (1850) spontaneous aneurysm was held to arise either through inflammation of the external wall, through tears or splits of both inner and middle coats, but most commonly by disease of the coats themselves, whether resulting in a diffuse cylindrical dilatation or in the formation of a sac.

The modern views of the origin of aneurysm date from the studies of Helmstedter (1873) and Köster (1875), who shewed that the primary and important change is in the elastic and muscular fibres of the middle coat of the vessel. Since then there have been many researches upon aneurysm, the two most important studies being Eppinger's and Thoma's. Both emphasised the changes in the media as the primary event, Eppinger regarding this change as rupture, Thoma holding that various disturbances of nutrition led to atrophy. The latter brought his views on aneurysm into accord with his studies on arteriosclerosis, believing that the compensatory thickening of the intima could even obliterate a small aneurysm. His article upon the dilatation-aneurysm is the most important that has appeared of late years. Of special interest is the confirmation of the old views of Paré and Morgagni of the syphilitic origin of the majority of

cases of aneurysm of the aorta. The relation of the mes-aortitis to syphilis has been demonstrated by Köster, Heller, Chiari, Benda, and others. The introduction of the *x*-rays has given us an important help in the diagnosis of internal aneurysm. For clinical reference the most important monographs are those of Crisp (1846), Broca (1856), and Sibson's papers in his collected works ; the recent pathological literature is admirably given in Lubarsch and Ostertag's *Ergebnisse* (1904).

Classification.—Numerous classifications have been made of aneurysms of the larger vessels, based on their external forms, the structures of the wall of the sac, or on the etiology. For practical purposes the following may be adopted :—

I. *True Aneurysm* (aneurysma verum or aneurysma spontaneum), in which one or more of the coats of the vessel form the wall of the tumour: (*a*) Dilatation-Aneurysm. (1) Limited to a certain portion of a vessel, fusiform, cylindroid; (2) Extending over a whole artery and its branches —cirsoid aneurysm. (*b*) Circumscribed saccular aneurysm in which there is a localised distension of two or more of the coats, or a dilatation of a limited area of the wall after destruction of the intima and part of the media. This is the common form of aneurysm of the aorta. (*c*) Dissecting aneurysm with splitting of the media, and occasionally with the formation of a new tube lined with intimal endothelium. II. *False Aneurysm* following a wound or the rupture of an artery, or of a true aneurysm, causing a diffuse or circumscribed haematoma. III. *Arterio-venous Aneurysm*, either with direct communication between an artery and vein, aneurysmal varix, or with the intervention of a sac, varicose aneurysm. IV. *Special forms* such as the parasitic, the erosion, the traction, the mycotic.

Etiology.—There are two chief factors, weakening of the coats of the aorta by disease, and strain, as expressed in sudden or prolonged increase of the intra-aortic blood-pressure. " In the normal condition, notwithstanding the variations in this stress from moment to moment, and its maintenance up to the point of physiological efficiency during a long lifetime, the balance existing between the elastic resistance of the vascular walls (chiefly due to the middle coat) and the forces tending to expand it is wonderfully well preserved ; and persons may attain an advanced age in whom neither the heart nor any of the larger arteries appear to have suffered in any appreciable degree. This, if duly considered from the purely physical point of view, is nothing less than wonderful ; and the wonder of it will surely increase when we remember how difficult— nay, how impossible—it would be to construct an artificial machine of elastic and distensible materials, which would not only resist indefinitely a constant mean internal pressure acting upon it through the contained liquids, but also a sudden impulse and variable increase of that pressure repeated periodically at the rate of over 100,000 times a day, or, say, 40,000,000 times a year, unceasingly, for all the seventy years of an average healthy human life " (Gairdner).

Disposing Causes—Age.—Hospital statistics and the Registrar-General's

reports shew that aneurysm of the aorta is most common between the ages of forty and fifty. Of the 1101 deaths from aneurysm in males in England and Wales in 1906, 549 occurred between the thirtieth and forty-fifth years. In Crisp's well-known figures dealing with 555 cases of aneurysm in different regions, the largest number, 198 cases, occurred between the ages of thirty and forty. In the young, and in the very old, aneurysm of the aorta is rare, though it may occur at any age. Le Boutillier has collected 80 cases of aneurysm in persons under twenty years of age, 14 cases being under twelve years of age. Only 18 of these were of the thoracic aorta, and 5 of the abdominal. The cases may have all the features of thoracic aneurysm in the adult. Congenital aneurysm is very rare ; one of the abdominal aorta was large enough to obstruct labour (Phénomenow). In the aged, thoracic aneurysm is more frequent than the figures indicate. It is very often latent, and multiple sacs are not infrequently met with.

Sex.—All statistics shew a marked predominance of males in aneurysm of the aorta, according to Lebert, in the proportion of 10 to 3. Both the factors mentioned above prevail in males ; in females strain does not play an important part. In 1906, 882 males and 219 females died of aneurysm in England and Wales. The age-incidence in females is very much higher than in males.

Race and Locality.—Statistics indicate that the disease is much more common in Great Britain than on the Continent. In Vienna among 19,300 necropsies, there were 230 of aneurysm, whereas at Guy's Hospital among 18,678 there were 325 cases. It is stated that aneurysm is more frequent in England than in France, but I do not know of any comparative statistics to justify this statement. The truth is that neither race nor locality are important factors in comparison with the prevalence of syphilis among the hard-working members of the community. In the negroes of the Southern States of America aneurysm is more common than among the whites. At the Johns Hopkins Hospital, Baltimore, of 345 admissions to the medical wards for aneurysm, 132 were in coloured and 213 in white patients, a ratio of 1 to 1·61, whilst the proportion of white to coloured, among the total admissions to the hospital, is 5 to 1.

Prof. Leonard Rogers has very kindly analysed for me the post-mortem records of the Calcutta Medical College for the last thirty-five years. There were only 30 aneurysms in 5900 subjects—0·5 per cent. The Europeans, who formed only 7 per cent, had 0·22 of cases as against 0·28 per cent of the Hindu. Prof. Rogers states that syphilis and arterial disease are common among the natives, and he attributes the comparative scarcity of aneurysm to the low blood-pressure.

Occupation.—Persons who use their muscles to excess, particularly those whose occupation necessitates sudden strain, are particularly liable to aneurysm of the aorta. Sir Clifford Allbutt in 1871 called attention particularly to sudden strain as " not only the cause, but the commonest cause of aortic aneurysm." Soldiers, sailors, draymen, iron- and steel-

workers, and dock-workers are particularly prone to the disease. The great frequency of aneurysm in the British Army demonstrated years ago by Myers and by Welch still continues. The figures for 1907 give 8 deaths from aortic aneurysm in a total strength of 118,521 for the home contingent. In Germany, 1904-5, there were only 4 cases of aneurysm in a strength of 555,777 ; in Italy in 1903, there were 6 cases in a strength of 206,468. In the British Navy in 1907 there were 24 cases among 108,740 men. At the Naval Hospital, Haslar, aneurysm is very common ; 47 cases were admitted in seven years.

Determining Causes.—These may be placed in three groups : those which weaken directly the coats of the vessel ; strain or internal trauma, leading to a break in the enfeebled coats ; and certain special causes. I. The acute infections are the most important single cause of arterial degeneration ; in scarlet fever, measles, diphtheria, enteric fever, small-pox, foci of degeneration are common in the aorta, but so far as aneurysm is concerned they are not very important. In many instances the intima alone is involved, but, as W. S. Thayer has pointed out in connexion with enteric fever, and Wissal in connexion with the acute infections of children, the changes may be in the media. There is only one infection of any moment with which aneurysm is connected, namely, *syphilis*. This was recognised, as we have already mentioned, by Paré. Both Lancisi and Morgagni realised the great influence of venereal disease, and in his wonderful section on aneurysm the latter gives case after case with a history of syphilis. In 1876, Francis H. Welch of the British Army called attention to the great frequency of syphilis among the subjects of aneurysm in the British Army—66 per cent. These figures have been amply confirmed, and among the percentages given may be mentioned the following :—Malmsten, 80 ; Heller, 85 ; Hampeln, 82 ; Etienne, 69 to 70 ; Pansini, 65. As has proved the case with locomotor ataxia, the more closely individual cases are investigated the higher the percentage is shewn to be infected. Indeed, in men under 40 it may be said to be by far the most important single cause.

There is now a consensus of opinion regarding the existence of a syphilitic aortitis with very definite characters. Macroscopically it is limited in extent, involving the root of the vessel or a band an inch or two in width of the arch or of the descending aorta just above the diaphragm, or there is a patch at the orifice of the coeliac axis. The intima does not present the usual appearance of the atheromatous change, but there are shallow depressions of a bluish tint, short transverse and longitudinal puckerings, sometimes with a stellate arrangement, or the intima is everywhere scarred with depressions and linear sulci. In most cases the diagnosis may be made at a glance, but there may be much more disease than indicated by inspection, and under a smooth and normal-looking intima there may be widespread mes-aortitis in an early stage. Microscopically there are found (1) foci of small-celled infiltration in the adventitia and media, sometimes so large that they look like miliary gummas, and were so described as far back as 1877 by Laveran and by Heiberg. (2)

Necrosis, fracture, separation, and disappearance of elastic and muscular fibres of the media, usually in patches, but often widely spread throughout the aorta. (3) The spirochaetae may be found in the lesions. The following features suggest syphilis in a given case :—The age—under forty a large majority of cases are luetic ; sudden death due to perforation in the small aneurysm of the arch ; multiple sacs, particularly the small cup-shaped form ; the presence of syphilitic lesions of bones, eyes, liver, testicles, etc. ; the onset with angina pectoris and aortic insufficiency ; parasyphilitic manifestations as tabes and general paralysis; or the husband may have tabes and the wife aneurysm, or, as reported by Jaccoud, both husband and wife may have aneurysm ; and, lastly, the beneficial effects of antisyphilitic remedies.

In any of the infections a patch of mes-aortitis may lead to a localised weakening of the wall and aneurysm, and in this way pneumonia, erysipelas, and influenza may be mentioned as possible causes. French writers, particularly Lancereaux, lay great stress upon malaria as a cause, but recent studies have not borne out this view. In Baltimore, where aneurysm is so common among the negroes, we were never able to trace any direct connexion with this disease. In another way the acute infections may be associated with the formation of aneurysm. The endocarditis of rheumatic fever, pneumonia, or of septicaemia may extend directly from the aortic valves to the intima of the vessel with the production of an extensive aortitis and aneurysmal dilatation. Quite as frequently, I think, as in the remarkable case I have reported, the process is embolic, and over foci of mes-aortitis the intima splits with the production of an aneurysm. Rheumatic fever as a cause of aneurysm has been specially studied by French writers, and the literature is fully given by Feytaud. The cases are in young subjects who have had repeated attacks.

The other great factor in weakening of the coats of the vessel is the intoxications :—the exogenous poisons, such as alcohol, lead, and tobacco, and the endogenous poisons, the results of perverted metabolism in gout, diabetes, chronic Bright's disease, etc. These lead to widespread degeneration of the arteries, but they are in themselves rare determining causes of aneurysm. The relation of atheroma of the aorta to aneurysm has been much discussed. It has long been recognised that the age-incidence of atheroma and of aneurysm of the aorta is not the same. A large number of cases of aneurysm occur in aortas that are not extensively diseased. On the other hand, the most extreme endarteritis deformans may exist without aneurysm. Arterial degeneration does not necessarily weaken the vessel, as when the intima is chiefly involved it may be, as Thoma points out, protective and defensive. The danger lies in the localised areas of degeneration in the elastic and muscular elements of the media, not in the widespread pan-aortitic changes or what is called atheroma (*vide* also p. 599).

II. The second important factor in the production of aneurysm is high blood-pressure—*internal strain*—particularly that which is associated with

sudden and violent muscular effort. Aneurysm occurs most frequently at the active periods of life, and in men who use their muscles very vigorously for short periods of time. A permanent high pressure may lead to degeneration of the coats, but what is much more important is that in a severe exertion, as in lifting, jumping, straining at stool, or in the act of parturition, a sudden heightened tension (an internal trauma) picks out a weak spot and the intima cracks over an area of mes-aortitis. The experimental production of aneurysm illustrates very clearly these two methods of the formation of aneurysm. Following the introduction of adrenalin, there is widespread aortic degeneration with areas of calcification of the media. In certain regions of the aorta local bulgings are found in which the intima is pushed directly into an area of weakened media and externa; in others, as so well figured by Fischer, over local areas of degeneration of the media the normal-looking intima splits, and behind this is a small saccular dilatation—the beginning of an aneurysm. In man the latter method is the more common ; the former may occur in the aged in any vessel extensively diseased.

III. *Occasional Causes.*—(*a*) Embolism.—The emboli may consist of valve vegetations, bits of thrombi, or calcified fragments from the valves. The cases are most commonly met with in infective endocarditis. The aneurysms are often multiple, and may be in the peripheral or mesenteric vessels. They are most frequent in the smaller arteries, and they rarely attain a very large size. The septic embolus may infect the arterial wall, causing acute inflammation of all the coats and the formation of a circumferential aneurysm. This is the common event in infective endocarditis. In the larger vessels the embolic aneurysm is due to foci of softening of the media in consequence of infection through the vasa vasorum. The intima over these small areas ruptures, and in this way small and large saccular aneurysms are formed. As many as four or five of such sacs may be present in the aortic arch, as in a remarkable case which I described in 1888. The embolic aneurysm is not always mycotic. A small calcified fragment may lacerate the wall. In an interesting case of this kind at the Radcliffe Infirmary, Oxford, the patient, an old examination subject, whose musical aortic diastolic murmur had been well known for some years, had a sudden severe pain in the calf of one leg, which became swollen, hot, and painful. When admitted, there was so much swelling and pain that it was not possible to say what was the nature of the trouble, but as these subsided pulsation became visible and he recovered in a few weeks with a well-marked aneurysm of the posterior tibial artery. A point of special interest was the disappearance of the musical quality of the diastolic murmur, which makes it probable that a calcified spike of vegetation had been whipped off as the embolus.

(*b*) External injury has been recognised as an occasional cause of aneurysm since the time of Vesalius. A blow on the chest, a sudden fall, or the jar of any accident may cause rupture of the aorta with the formation of a dissecting aneurysm, or in the course of a week or a month

the clinical features of aneurysm may be present. Usually the cases present the symptoms of ordinary saccular aneurysm of the aorta, but in one of my cases, reported by M'Crae, there was dilatation of the aorta. Trauma is a very common event in aneurysm of the abdominal aorta. The essential feature is a split of the intima and inner portion of the media; the wall may rupture completely, a dissecting aneurysm may follow with healing or with rupture, or the split may be small in extent and behind it the weakened media and adventitia yield with the gradual formation of a saccular aneurysm.
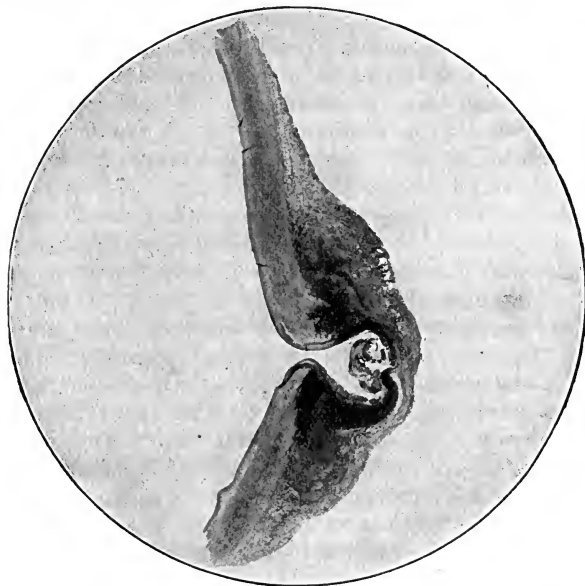
Fig. 80.—Tuberculous aneurysm of the aorta. The section is through part of the thoracic aorta. From a case of intensely acute miliary tuberculosis in which there was an old focus of tuberculosis in the apex of one lung, and this by haemic infection produced tuberculosis of the adventitia of the aorta. This extended until the media was so weakened that dilatation occurred. There were numbers of tubercle bacilli in the loose coagulum at the bottom of the aneurysm, and also in the caseous material around it. × 8. (Councilman.)

(c) Among the rarer causes of aneurysm may be mentioned external erosion. A bullet may lodge near the wall of an artery or abraid it, or the wall may be weakened by an adjacent tuberculous focus or by a small abscess.

(d) Hypoplasia of the aorta may be associated with aneurysm. Lee Dickinson described two cases in young adults with very small aortae without arterial sclerosis. One case presented three aneurysms.

Pathological Anatomy.—Two different groups of cases of aneurysm of the aorta come to necropsy. In hospital practice we see the large tumours in patients who have had well-marked symptoms. Only a limited number die suddenly in the wards without the condition having been

recognised. The second group come under the notice of the medical jurist, and it is only the coroner-physician in a large city who appreciates the extraordinary importance of aneurysm as a cause of sudden death. These are the small tumours above the aortic ring, the syphilitic aneurysms in young men, the splits and fissures of the intima, and the dissecting aneurysms.

Considering how commonly the tumour perforates the sternum, rupture with death from external bleeding is very rare. Not uncommonly the area of skin at the point of maximum protrusion becomes necrotic, or the fibrinous laminae may be exposed. After death, a large external tumour may diminish greatly in size. In opening the thorax in a case of suspected aneurysm it is important to make a very careful dissection of the relations of the great vessels in situ. Rupture takes place frequently into the pericardium, in which case the sac is distended and may contain a pint or more of blood. In a few rare instances the fatal result does not follow at once, and there may be firm and laminated thrombi. Rupture into the pleura is also very common, in which case the volume of blood is large, amounting to three or more pounds, the lung is compressed, and there is a large solid clot at the posterior part of the pleura with the clear liquor sanguinis above. When perforation has taken place into the trachea or a bronchus, blood is usually brought up and some may pass into the bronchi. In a few instances blood is not brought up, but passes into the stomach; I dissected one case of this kind in which death occurred without any external bleeding.

Situation of the Aneurysm of the Aorta.—The arch is most commonly affected, and the ascending portion more frequently than the transverse; the descending thoracic aorta being the part least often affected. The ratio of implication of the abdominal and thoracic was about 1 in 6 in the Guy's Hospital series. In the collected statistics of Crisp, Lebert, and Myers the ascending aorta was involved in 159, the transverse arch in 113, the descending thoracic aorta in 49, and the abdominal aorta in 83.

Number.—As a rule, only a single aneurysm is present, but it is not uncommon to find two or three. Cases are recorded of a dozen or more in the course of the vessel. The mycotic aneurysms are usually multiple. In the ascending arch there may be four or five small cup-shaped tumours. Some individuals are peculiarly subject to aneurysm of different vessels; the late Dr. Thomas King Chambers had first an aneurysm of the left popliteal artery, then of the right vessel, and finally of both carotids.

The *size* of the aneurysm of the aorta ranges from a cup-shaped sac the size of the tip of the little finger, to a huge tumour as big as an adult's head, the contents of which may weigh five or six pounds. The largest sacs are connected with the terminal portion of the arch and the descending thoracic aorta. Growing in these situations, the tumour may occupy a large part of the thorax, or it may perforate the chest-wall and form a huge subcutaneous tumour, as shewn in Fig. 81.

Form.—There are two great types, one in which the lumen of the

aorta is dilated, the other in which the limited section of the wall gives way ·with the formation of a sac. The dilatation-aneurysm may be cylindrical, in which case there is uniform enlargement of the tube, either in a limited section or, as it sometimes happens, of the entire aorta. More frequently it is a localised enlargement of the arch alone, either cylindrical or fusiform. Sometimes the arch forms a huge flabby sac, or there may

FIG. 81.—Aneurysm of the thoracic aorta perforating the chest-wall.

be a very definite spindle, or even the sections of two spindles. Some of the most typical fusiform aneurysms are of the abdominal aorta. The localised aneurysmal tumour may be saccular, communicating with the lumen of the aorta by a narrow neck, saucer- or cup-shaped, crater-like, tent-shaped, or sphenoid, or multilocular when on a large sac a series of secondary tumours arise. Clinically, the dilatation and the saccular aneurysm are very different. Sometimes they are combined.

Structure.—The large fusiform or cylindrical aneurysms of the aorta

present a rough atheromatous intima, often with calcified plates, foci of atheromatous softening, and here and there localised bulgings or secondary sacs, with flakes of adherent thrombi. The walls of the dilated vessels are thin. There may be very little adhesion to adjacent structures. In other cases parts of the fusiform dilatation may be occupied by firm thrombi.

In the localised saccular aneurysm, whether cup-shaped, crateriform, or tent-like, the intima of the aorta usually terminates at or close to the margin of the sac. In the small cup-shaped aneurysms just above the aortic ring the walls are thin, even translucent, and there is frequently a spot of perforation into the pericardium. Only in small sacs is the lining composed of a thickened intima; in the larger ones it is made up of remnants of the media and a thickened adventitia. Then comes a stage in the growth of large sacs when part of the wall is no longer made up of arterial coat, but is in direct connexion with the adjacent tissues, lung, pericardium, bone, mediastinal tissues, or skin.

Evolution of an Aneurysm.—Three factors are concerned, first, the necrosis and fracture of the elastic and muscular elements of the media, permitting a split or tear of the intima or its gradual yielding over the weakened area; secondly, a constant blood-pressure in the aorta, heightened by many causes ; and, thirdly, remarkable reparative processes, namely, new growth of connective tissue and a strengthening of the wall of the sac by the deposition in sheets of fibrin. Once started it becomes a struggle between the blood-pressure, which tends gradually to stretch the weakened spot, and the reparative processes which strengthen it. In some very acute cases there is little or no attempt at repair ; but in a majority of aneurysms of the aorta the reparative processes are among their most distinctive anatomical features. Two structures are at work in repairing the break—the connective tissues of the wall and of the adjoining structures, and the blood, from which tough fibrinous mats are laid down. In small aneurysms, in which the connective-tissue repair is seen to perfection, it is an intimal affair. As Thoma has pointed out, a local bulging on the wall of such an artery as the ophthalmic may be completely obliterated by new growth from the intima, so that the inner surface of the artery and of the sac are on a level. Even in small sacs of the aorta this process may be effective (see Fig. 5, Pl. I., *Virchows Arch.*, Bd. cv.), but it is much more common in the smaller vessels. This power of repair is present in every aneurysm in which the intima remains ; but as a rule the growth of the sac is far too rapid for the reparative endarteritis to be of much service. In the ordinary aneurysm of the aorta the active proliferation of the tissues of the adventitia, the thickening of the mediastinal, pericardial, pleural, and other structures, and the passive resistance offered by neighbouring parts, are the three great factors in resisting the gradual enlargement by the incessant strain of from 60 to 80 charges per minute of a powerful pump into its interior. The second important element in the repair of an aneurysm is thrombosis— the deposition from the blood of laminated fibrin. It is not the ordinary

clotting, the red clot, but it is by the formation of white thrombi, the active clotting of Broca, that the fibrin is deposited layer by layer, until a sac the size of an orange may be completely filled with from 30 to 60 layers, which may be peeled out like the coats of an onion. We know nothing of the circumstances in which this process occurs, except that it is seen most often in sacs with narrow necks, but there may be no trace of it in aneurysms that look most favourable for the process. It may occur in the spindle or cylindrical forms. The process may be traced on the wall which shews a greyish-white deposit of platelets ribbed like sand on the sea-shore, or arranged in a network. The lamination
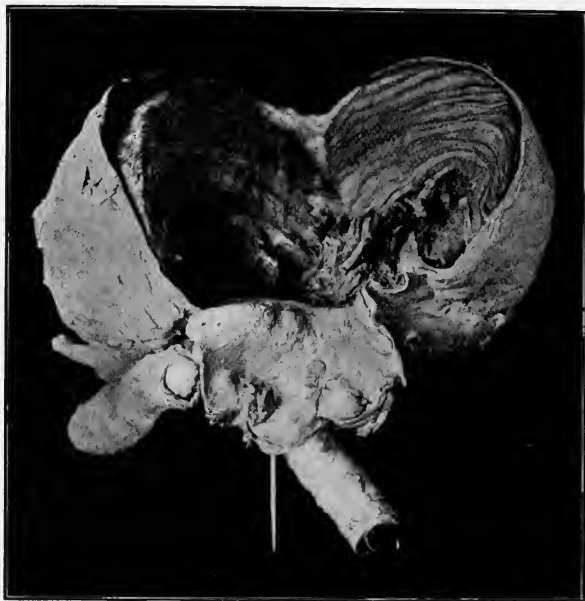
Fig. 82.—Healed aneurysm of the arch of the aorta. The specimen is in the museum of M'Gill University, Montreal.

is in some way connected with the deposition of the blood-plates, with which, as we know, the thrombosis is associated. Leucocytes and red corpuscles take part in the process, and at first the layers of fibrin are reddish-brown, but in very old sacs the colouring matter disappears and the laminae become greyish-white. In long-standing cases lime salts may be deposited, and the whole becomes a firm calcified mass. In the natural process of healing the sac may be obliterated, a cup-shaped depression remaining at the mouth, but the surface of the thrombus may be as smooth and hard as the palm of the hand. Healed in this way the sac shrinks to a firm solid mass, which may remain unaltered for twenty years or more. On cross-section, as shewn at Fig. 82, the concentric laminae are seen. Organisation does not take place, and even

at the periphery, where the thrombus is embraced by a firm fibrous capsule, vessels do not pass into the laminae. In the fusiform aneurysm, as shewn beautifully in Hodgson's figure, the thrombus appears to be canalised; but this represents in reality the original lumen of the vessel.

Effects of Compression.—In its growth an aneurysm destroys all structures which resist its advance. Soft structures, as the vessels, the oesophagus, and the lung, may be pushed aside, but the incessant pounding hour by hour, day by day, gradually destroys the hardest tissues. Passing anteriorly the sac erodes the sternum, the cartilages, and the ribs, and may force a breach in the wall of the chest sufficiently large to admit two fists. By the growth of the aneurysm backwards the bones of the spine may be eroded even to the canal, and not uncommonly the half-destroyed bodies of from three to six or even more vertebrae may be found free in the sac. As noted by Morgagni, the intervertebral discs, which are yielding structures, are not destroyed at the same rate as the bone, and may remain more or less intact, while the bodies are deeply eroded. The cord may be compressed, and the dura mater has been exposed for 4 or 5 inches in the sac. In rare cases, the spinal changes are so extensive and deep that a curvature follows. In the upward growth of an aneurysm of the arch the clavicle may be dislocated, and its sternal half destroyed. A solid organ may completely disappear; in one of my cases the left kidney had been absorbed. Many hypotheses have been advanced to explain this destruction of solid bodies by the aneurysm—a "corrosive ichor," from the blood, mechanical erosion, and, in the case of bone, a rarefying osteitis. The mechanical effect of the intermittent pounding is the important factor, and the structures are worn away as the often-falling drop erodes the hardest stone.

DILATATION-ANEURYSM.—Diffuse dilatation-aneurysm of the arteries is met with under two conditions. In one there is a passive dilatation of the aorta or of its main branches, due to disease of the walls; this may be local or involve the aorta in its entire course. In the other condition, which is seen most frequently in the smaller branches, there is an active dilatation, due to growth and enlargement of the vessel.

Dilatation-Aneurysm of the Aorta.—Morgagni drew a clear distinction between the aneurysm which occupied the whole circumference of the vessel and the tumour which affected one side only. Scarpa, who recognised dilatation of the whole aorta, did not regard it as aneurysm. Joseph Hodgson, in 1815, distinguished the condition clearly from ordinary aneurysm, and spoke of it as a "preternatural, permanent enlargement of the cavity of an artery." He described it as specially affecting the arch, and observed that the symptoms suggested organic disease of the heart rather than aneurysm. He knew of its association with insufficiency of the valves, and it is this combination to which the French gave the term *Maladie de Hodgson.* In his well-known study on aneurysm Thoma gives by far the best recent account, and he describes and figures a number of forms. The following are among the most important

varieties :—(1) Diffuse dilatation of the entire aorta. Occasionally the entire tube is uniformly dilated, measuring 7, 8, or even 9 cm. in circumference. Nearly every museum contains one or two illustrations of this, the specimens having usually come from very old persons with extensive endarteritis deformans. Cruveilhier gives a good figure of this condition in his atlas. (2) Localised dilatation. This is much more common in the arch, which may be uniformly affected, sometimes cylindrically, in others spindle-shaped. The disease may be confined to the thoracic or to the abdominal portion of the aorta. Thoma figures a whole series of these dilatations, particularly the varieties of fusiform aneurysm. Some of these are multiple, and not uncommonly a sac is engrafted on a spindle-shaped aneurysm. In connexion with the upper part of the thoracic aorta, Thoma describes a special form, the tent-shaped or sphenoid, which he regards as the result of extra tension at the point where the upper intercostal arteries are given off. Thrombi may be present, and occasionally form firm laminae ; a fusiform aneurysm of the descending thoracic or of the abdominal aorta may be almost completely occupied by firm laminae. The vessels given off from the arch and the iliacs are not infrequently involved in the dilatation. With dilatation of the arch the aortic ring may be dilated, and the valves themselves are often sclerotic and shortened.

Etiology.— The essential factors are really those of chronic aortitis leading to loss of elasticity and with gradual dilatation. In T. M'Crae's series of 35 cases at the Johns Hopkins Hospital the ages were as follows : thirty to forty years, 8 ; forty-one to fifty years, 12 ; fifty-one to sixty years, 11 ; sixty-one to seventy years, 3 ; seventy-one to eighty years, 1. These figures from the wards of an active general hospital scarcely give the true incidence, as the condition is common in old people and is met with much more frequently in almshouses and asylums. Of the patients under forty years 4 had a definite history of syphilis. Males are much more frequently the subjects of the condition—31 to 4 females in M'Crae's series.

At the Johns Hopkins Hospital our attention was early called to the great frequency of arterial disease in the coloured patients, and in M'Crae's series of dilatation of the aorta 14 of the 35 were negroes. Syphilis is an important factor, and there was a definite history in 10 cases and a strong probability in 7 others. A large proportion of the patients had done heavy muscular work. In 1, the dilatation seemed to have followed a severe strain and exposure in a shipwreck.

Symptoms.—As Hodgson remarked, the condition is very frequently mistaken for heart disease. Pressure features are not so often met with as in the saccular aneurysm. On the other hand, the symptoms of aortic insufficiency, myocarditis, and angina pectoris are common. Now that the *x*-rays have given such important aid in the diagnosis the condition will be more frequently recognised. Thoma remarks in connexion with his interesting series of cases that in scarcely one had the condition been recognised in the wards. There are several groups of cases : (1)

The latent in which the dilatation is discovered in persons who have not had any indications of cardiovascular trouble. In the post-mortem work of infirmaries and almshouses it is not very uncommon to find extensive dilatation of the arch or, indeed, of the entire aorta in individuals who have not presented any special symptoms. An old woman, in the Philadelphia Hospital, whose calcified peripheral arteries edified the students of successive ward-classes, had otherwise a very normal circulation ; her heart's action was slow, and it was frequently a matter of comment that it should be so normal. After her death from an intercurrent malady we were all greatly surprised to find that her thoracic aorta was very markedly and uniformly dilated, and in a state of the most advanced endarteritis deformans. (2) The clinical picture may be that of angina pectoris. This is most commonly seen in the young syphilitics with aortitis and slight dilatation of the arch. Sometimes it is in the senile form that the attacks may recur at intervals for years without any other sign of heart disease. (3) A group in which the clinical features are those of aortic insufficiency, or the symptoms are directly associated with progressive failure of the heart-muscle. This was the group which Hodgson referred to as having the clinical features of disease of the heart. Lastly, in a few cases there are the symptoms and physical signs of aneurysm. Pain was a prominent feature in 6 cases. In the 35 cases analysed by M'Crae, dyspnoea was the most constant symptom, and was almost always associated with cardiac insufficiency, which also explained other features such as oedema of the feet and cough.

Pressure Effects.—In striking contrast to the saccular aneurysm these are not well marked. Fulness of the veins of the neck and arms was noted in 12 cases. The radial pulses were unequal in 2. In a few cases of great dilatation the trachea is flattened, but paroxysmal dyspnoea is rare. Tracheal tugging was noted in three instances. Inequality of the pupils was present in 4 cases, and paralysis of the left vocal cord in 2. Dysphagia was present in 1 case. Erosion of the bones is rare ; it may occur in connexion with the saccular aneurysm engrafted upon the dilated arch.

Physical Signs. — Inspection gives the most important indications. Visible pulsation in the episternal notch is present in a majority of the cases, and it may extend above the right sterno-clavicular articulation. Barié thinks the throbbing in this situation, due to elevation of the right subclavian, is one of the best indications of dilatation of the arch. Sometimes there is a prominent tumour filling the sternal notch. Pulsation in the first and second right interspaces is not nearly so common. It was present in 6 cases in the series. A diffuse pulsation of the manubrium is present in about half the cases, but in old persons with rigid chest-walls there may be an extreme degree of dilatation without any visible pulsation. Careful inspection in a good light is very necessary for the detection of the diffuse impulse over the manubrium. In no case of the series was pulsation visible in the back. By palpation the forcible throbbing of the dilated arch may be felt in the sternal notch. A rough systolic thrill is

sometimes felt over the manubrium, and occasionally, when the valves are insufficient, a diastolic thrill. A sharp diastolic shock may be felt over the aortic area. In a majority of cases, 29 out of the 35, percussion reveals dulness over the manubrium, which may extend into the interspaces on either side. On auscultation the most characteristic sign is a bell-like second sound of a very clanging and metallic, sometimes an amphoric, quality. Another important auscultatory feature is the transmission upwards, when present, of the diastolic murmur, which may sometimes be heard loudly over the manubrium, and even propagated into the vessels of the neck. The blood-pressure of 20 of M'Crae's 26 cases in which it was taken was below 140 mm. In two patients there was an average of 80 mm. The highest pressure was 260 mm.

Examination by *x*-rays.—In skilled hands the diagnosis is readily made with the fluoroscope, as the dilated aorta casts a very definite shadow extending high in the thorax, much larger than the normal aorta and shewing very little difference in extent during systole and diastole.

The *diagnosis* is not often made, unless a constant outlook is kept for the condition. The important points are : (1) The diffuse pulsation, for the detection of which a good light is always necessary, and sometimes trained eyes. (2) The clanging metallic quality of the second sound. (3) When present, the widespread diffusion of the diastolic murmur upwards; and (4) lastly, and most important, the fluoroscopic examination.

Active Dilatation-Aneurysm.—Diffuse Arterial Ectasia.—Cirsoid or Racemose Aneurysm.—In this remarkable form the arteries enlarge actively by a new growth of vascular tissue. The blood-vessels retain their embryonic power of growth throughout life, and when a main vessel of a limb is tied, the collateral circulation is re-established by an active growth and enlargement of the arteries. The enlargement of arteries in splenomegaly, in the pregnant uterus, and in many other conditions illustrates the extraordinary plasticity of these structures. Spontaneous enlargement with dilatation is chiefly seen in the smaller branches, and is known by the names already mentioned. Vessels of the fourth and fifth dimensions are most frequently affected. In many cases the process is confined to the arteries ; in other instances the veins are dilated, and even the capillaries may be implicated, forming a diffuse angioma. The vessels most often attacked are those of the head and the hands, but the blood-vessels of the feet or any group in the body may be involved. Occasionally those of the internal organs are affected.

Etiologically there are three groups of cases. In the first, the process begins in a small birth-mark or a tiny angioma, particularly in those about the ears or the forehead. In such the angiomatous structure is preserved, the skin is involved, and while the arteries progressively increase in size and throb forcibly, the veins and capillaries also enlarge. In the second group, aneurysmal dilatation follows directly upon an injury, such as a blow upon the head, a slap on the face, a slight burn ; and, lastly, in a remarkable group the condition follows an acute infection. In a case reported by Bazy, a man, aged nineteen, had, during convalescence

from an attack of enteric fever, an induration on the palmer surface of one hand. In a few months dilatation of the arteries of this hand began, which progressively increased, and within a couple of years the radial artery was as large as the brachial. In a patient of Reverdin's, aged thirty-one, an illness resembling enteric fever was followed by a swelling over the left eyelid. It gradually increased and formed a pulsating tumour of the temporal region. Reverdin operated and removed a bunch of convoluted branches of the temporal artery. In 1903 a patient, convalescent from enteric fever, under my care at the Johns Hopkins Hospital, presented a very remarkable bruit high up in the interscapular region, and on both sides of the spine a group of greatly enlarged, tortuous, throbbing arteries not involving the skin. Two other bunches, not quite so large, were present in the subcutaneous tissues of the abdomen. He was aware of their presence, but he thought they had enlarged since the fever.

Symptoms.—When small the cirsoid aneurysm causes no trouble. The patient just referred to had no unpleasant sensations. The large throbbing angiomas, which invade the skin, cause great disfigurement, and in the hands or feet considerable disability. In those starting from a small birth-mark or a naevus, the skin is swollen, of a bluish tint, the dilated arteries are readily seen, and if the whole skin is not involved there may be numerous telangiectases. With the large angiomas on the side of the head there may be exophthalmos, and the process may implicate the skull and the dura mater. One of the most extraordinary features of the cirsoid aneurysm is the rapidity of its growth, and in this respect it may resemble a neoplasm. Over the dilated vessels there is pulsation, and usually a thrill. With the stethoscope, if the arteries alone are dilated, there is a loud systolic murmur. When the veins and capillaries are extensively implicated there is a continuous whirring murmur with systolic intensification. Another remarkable feature is the occasional spontaneous disappearance, of which a number of cases are on record. In several the diminution has followed an acute erysipelatous inflammation. A striking instance is reported by Fernell : A man, aged twenty years, had a large pulsating tumour above the right clavicle, which had lasted many years, and which involved all the branches of the thyroid axis except the inferior thyroid ; the transversalis coli and suprascapular arteries were easily felt, greatly enlarged, and tortuous. During an attack of measles, in which the temperature rose to 106·5° F., the tumour looked very red and angry, and pulsated very strongly, as if about to rupture. A compress was applied and veratrum viride, ergot, and iron were given. After the attack the tumour began to subside, gradually the pulsation and thrill disappeared, and it shrank to a mass of hard connective tissue which could be rolled about.

DISSECTING ANEURYSM.—A majority of aneurysms of the aorta begin with a split or crack of the intima over a spot of mes-aortitis. Recently experimental work has shewn the important bearing of these fissure. When a fracture has once started, five events may follow :—The

aorta may rupture in all its coats ; an ordinary aneurysm may form at the site of the split ; the fracture, though large and even circumferential, may heal completely ; the blood may extend between the coats of the aorta separating them for many inches or in the entire length of the vessel ; and, lastly, a dissecting aneurysm thus formed may heal perfectly.

I. **Rupture of the Aorta.**—There are two groups of cases : (*a*) The traumatic, in which the accident follows a blow on the chest or back, or a fall ; and (*b*) the spontaneous, in which, during rest or during a sudden effort, the vessel ruptures. Four-fifths of the cases come in this class. Prolonged straining efforts, as at stool, during confinement, or in lifting, emotional excitement, laughing, crying, the strain of coitus, are among the exciting causes. It occurred in a healthy boy of thirteen after prolonged muscular exertion. Men are more liable to it than women in the proportion of 70 to 24, according to Maurice Martius. It is more common after the thirtieth year of life. The usual seat of the rupture is the first part of the arch, 89 cases ; there were 18 of the transverse portion, and only 5 of the abdominal aorta. In a large proportion of the cases rupture takes place into the pericardium.

The vessel may be almost torn away from the heart ; the intima usually shews a very sharp-edged break, which may be from a few millimetres in extent to the entire circumference. Some little separation of the coats is usual before the blood bursts through the adventitia, so that the external opening is not always opposite the break in the intima. The vessel itself is almost always diseased, though little may be apparent, as there may be extensive mes-aortitis with a smooth intima.

Symptoms.—Death may take place instantly (26 cases) ; in others it follows in the course of a few hours, but in about half the cases there are two very characteristic stages, one corresponding to the rupture of the inner coats, the other to the external and fatal break. In one of the first and one of the best reported cases in literature, by Linn, a woman, aged twenty-nine, while in labour, started up in bed with an agonising pain in the heart, and said she was dying, and became cold and pulseless. Linn thought the heart had ruptured. After delivery she revived and improved gradually until the fourteenth day, when she again had an agonising pain in the chest, and died in a few minutes. The aorta had ruptured into the pericardium. The interval from the internal rupture, which seems always to be accompanied with great pain and collapse, may be from six or eight hours to fifteen or sixteen days. The cases are of great medico-legal interest as rupture may follow a very slight blow or fall, or occur in a scuffle during a quarrel.

II. **Rupture of the Inner Coats with the Formation of a Saccular Aneurysm.**—Weakness of the media due to necrosis and fracture of the muscular and elastic fibres is the essential factor in ordinary spontaneous aneurysm. A split of the intima may occur over this weakened spot and the blood, instead of separating the coats, may cause a bulging or aneurysmal dilatation. This may be well seen in experimental aortitis in

the rabbit. The little fissures in a perfectly smooth intima lead into a pocket or pouch. In the embolic mes-aortitis, which may follow endocarditis, I have seen in the arch five splits of the intima, each one leading into a small aneurysm. A woman, aged thirty-five, who died suddenly, presented in the thoracic aorta a linear fissure 1·5 cm. in extent, which led into a sac the size of a small apple ; this had ruptured into the oesophagus. This aspect of the subject will be more fully considered under ordinary aneurysm (p. 650).

III. **Fracture of the Inner Coats with Healing.**—Rokitansky first pointed out that splits and tears of the intima might heal completely ; he reported 5 cases. The condition is one of the most remarkable in the whole range of vascular pathology. A man, aged sixty, was

FIG. 83.—Completely healed split of the intima surrounding the entire aorta just above the valve. Recent split with dissecting aneurysm, which burst into the pericardium. Daland's case.

admitted dyspnoeic and dropsical, with pain in the chest and a very feeble heart ; after death there were found above the aortic valves splits of the intima with separation of the middle coat, but the edges were smooth and rounded, and where the middle coat was exposed it had a cicatricial appearance. Von Recklinghausen, Zahn, von Schroetter, and others describe and figure the condition. Reproduced here is the drawing made from Daland's case, the heart of which I dissected with him. Three years before death the man had an attack of severe pain in the chest and unconsciousness, from which he gradually recovered. Death occurred suddenly from rupture into the pericardium. In the entire circumference of the aorta the intima was split as if cut with a razor. From this a dissecting aneurysm had separated the coats of the ascending aorta, and broken into the pericardium. But the remarkable feature was the scarred and cicatricial first inch above the valves, where

on a former occasion, probably in the attack three years before, the intima had ruptured and exposed the media which, with the edges of the intima itself, is smooth and fibrous.

IV. **Dissecting Aneurysm.**—Extensive separation of the coats of the aorta beginning at the site of the split is rare. There were only 2 cases in sixteen years at the Johns Hopkins Hospital, but in medico-legal work it is comparatively common. The late J. B. S. Jackson, of Boston, U.S.A., made a large collection in the Warren Pathological Museum, where I was able to look over twenty specimens. One of the earliest cases described was that of George II., who died of rupture of the right ventricle, and in whose aorta there was also a transverse fissure through which the blood had passed under the external coat forming an ecchymosis. Nicholls, who described the case in the *Philosophical Transactions*, made a number of experiments to determine the strength of the aorta, and the conditions under which rupture could occur. Laennec gave an excellent account of a case, and Pennock, of Philadelphia, who reported a remarkable case, determined experimentally that the situation of the aneurysm was between the layers of the middle coat. Peacock wrote two admirable monographs, from which our statistical information on the subject dates. The primary split, most frequently in the arch from 2 to 3 centimetres above the valves, is in the form of a transverse or vertical clean-cut incision, as if made with a razor. The intima about it may be smooth or it may be atheromatous; sometimes the splitting of the coats takes place at the edge of an atheromatous ulcer. The extent may vary from a few millimetres to a long tear of 4 or 5 centimetres extending round the entire circumference of the vessel, or running obliquely along the inner surface of the arch. There may be two or more tears near each other. The extent of the dissection is variable. If it reaches the adventitia rupture is certain to take place, for it is only the structures of the middle coat that can resist for any time the pressure of the blood. The blood may pass for an inch or more between the layers of the media, and a clot, an ecchymosis as Nicholls calls it, raises the intima. In other cases the blood passes for 3 or 4 inches or more, separating the coats, and then may burst externally or into the lumen of the vessel. In other cases the dissection is most extensive, reaching from the ascending arch to the bifurcation, and even passing down the iliacs and femorals to the vessels of the legs. Upwards the dissection may reach to the branches of the carotids and the brachials. In rare instances, as in one described by Rokitansky, the dissection involved the aorta from a little above the ring, and nearly all its main branches in the head, arms, and legs. In the circumference of the aorta a small section only may be involved, or the coats may be so separated that a double tube is formed, joined here and there by bridles of the media and by the arterial branches. Curiously enough it is these very extensive dissections that appear to heal, as will be considered in the next section. In nearly all cases there is an orifice of exit, through which the dissecting aneurysm communicates with the interior of the

aorta, or there may be two or three. The state of the cavity depends upon the length of time it has lasted. If the patient has only lived a few days, there will be fresh clots adherent to the rough walls, but at the end of a few months the thrombi are firm and may be laminated. In a few cases complete healing takes place.

The symptoms are those already described under rupture (p. 638), a sudden sharp pain in the chest with great shock and collapse, in which death may take place ; or recovery may follow for a week or ten days, and sudden death occurs from rupture. There may be agonising pain along the course of all the arteries extending into the limbs.

V. **Healed Dissecting Aneurysm.**—When there is a great rent of the intima, and the coats of the vessel are separated from the valves to the bifurcation and the branches torn off, it is impossible to conceive a lesion of greater severity ; and yet, so marvellous are nature's capacities, complete healing may follow, even to the extent of an internal lining for the new tube. So perfect may be this *restitutio ad integrum* that not only by the older observers, but every now and again at the present day the condition is described as a congenital anomaly—a double aorta. Shekelton, a Dublin surgeon, first reported a case of this kind which he thought was possibly an anomaly, but in a second case he clearly understood the condition. Pennock, of Philadelphia, who, in 1838, reported a remarkable case, appreciated the mode of formation, and made a number of experiments to shew that it was in the middle coat alone that dissecting aneurysm was possible. So extraordinarily natural did the outer tube appear to be that Hope, in his well-known book on the heart, favoured the view that it was a congenital anomaly. The best recent accounts are given by Boström and by Adami. The latter has collected 39 recorded cases, among which women and men were about equally affected. In a majority of the cases there was no advanced disease of the aorta itself. The site of the primary rupture was in the ascending aorta in 13 cases, below the origin of the left subclavian in 12, at the lower end of the thoracic aorta in 5, in the abdominal aorta and in the iliac in one each. As shewn in the figure in Boström's paper, the outer tube may extend the entire length of the aorta, occupying a variable extent of the circumference. The branches may take their origin from the outer tube, the lining of which is smooth, like a normal intima, and Rindfleisch shewed that the intima was in reality reformed. Atheromatous changes may occur in the new tube. The duration extends over many years. The late James E. Graham reported a case in which a soldier, who had been discharged from the army for aneurysm after the Crimean War, died thirty years later. When a student in Toronto, I often saw this man with Dr. Richardson, and knowing my interest Dr. Graham sent the aorta to me for dissection. There was a healed aneurysm at the terminal portion of the arch, and from the margin of this sac to the iliacs the aorta formed a double tube, exactly like the one depicted by Boström. As the patient was discharged from the army for aneurysm, it is probable that this had lasted for more than thirty years.

SACCULAR ANEURYSM OF THE AORTA.—A great majority of the cases in medical practice affect the aorta, and are of the type known as saccular. As the arch is most commonly involved, we shall describe first the aneurysms of this part, and then those of the descending thoracic and abdominal portions.

I. **Aneurysm of the Arch.**—General Features.—For purposes of description this part of the vessel may be divided into the sinuses of Valsalva, the ascending, and the transverse portions.

Aneurysm of a sinus of Valsalva is a common and most important variety, with special features. One, two, or all three sinuses may be involved. The tumours are small and cup-shaped, and rarely attain a size sufficient to give physical signs. The coronary arteries may be given off from the sac, or one or other of these vessels may be dilated. The aortic ring is apt to be involved, and one or more of the valves may be rendered incompetent. Perforation is common, usually into the pericardium, more rarely into the superior vena cava, the pulmonary artery, or one of the auricles. In some cases the aneurysm appears to be given off directly from the aortic ring, and involves as much of the ventricle as of the sinus. Whilst a few of the cases are in association with ordinary atheromatous changes, this special form is most frequently met with in acute syphilitic aortitis. The special features may be thus summarised : (1) It is very often latent, sudden death occurring before any symptoms have appeared ; (2) it is a medico-legal aneurysm, met with in connexion with coroners' cases ; (3) angina pectoris is not infrequent ; (4) aortic insufficiency is often associated with it ; and (5) in the majority of cases characteristic syphilitic changes are present in the aorta.

Aneurysm of the Ascending Arch.—The convexity of the vessel as it passes up is the common point of origin of the ordinary saccular aneurysm. The tumour grows to the right and anteriorly, " pointing " in the 2nd or 3rd right interspace, and as it increases pushes aside the lung. Some of the largest sacs are in this situation. Perforation occurs into the pericardium, the superior vena cava, the right bronchus, the pleura, and rarely externally. As the sac enlarges it may erode the sternum and the 2nd and 3rd ribs and cartilages, and appear as a large smooth external tumour. Less often it passes back and erodes the spine.

Aneurysm of the Transverse Arch.—A very common situation is at the orifice of the innominate, or this vessel and the arch may be involved together. The sac may originate from any part of the circumference of the vessels, most often from the posterior or postero-inferior. Owing to the very small space between the spine and the sternum, aneurysm in the situation has not much room for growth, so that pressure-symptoms appear early—pressure on the windpipe producing cough and dyspnoea, on the veins causing congestion of the face and arms, on the recurrent laryngeal nerve causing hoarseness, and on the oesophagus causing dysphagia ; a small tumour may give all the symptoms of aneurysm without a single

physical sign. As the result of its growth in a forward direction the sternum is eroded, and a large tumour may appear externally ; when it grows backwards the spine may be eroded, and sometimes the sac reaches a very large size and involves the bodies of three or four vertebrae. Tumours growing from the concavity and terminal portion of the arch implicate the recurrent laryngeal nerve at an early period, and there may be only tracheo-laryngeal symptoms.

In the terminal portion of the arch occurs the rare *traction* aneurysm, described by Thoma, which arises at the point of insertion of the ductus arteriosus. Usually funnel-shaped and small and not of much clinical importance, it is met with in young persons, and particularly in cases of kypho-skoliosis with displacement of the thoracic viscera.

Symptoms. — Many cases are latent. The records of coroners' inquests prove how frequently sudden death in apparently healthy individuals is due to rupture, more particularly in robust persons with syphilitic aortitis and the small cup-shaped aneurysms just above the valves. An aneurysm, however, may attain quite a large size without causing any symptoms. Indeed, it may perforate the chest-wall and project as a tumour of considerable size. An instance of this sort I saw at the University Hospital, Philadelphia, in a very intelligent working man, who assured us that he had had discomfort for a few days only before he noticed a prominent bulging tumour which had eroded the 2nd rib on the right side. As Sir William Broadbent pointed out, there is a certain antagonism between the symptoms and physical signs, and he suggested a useful division into aneurysms with symptoms and aneurysms with physical signs. Usually, both are combined, but it is not uncommon to see cases with every symptom without a physical sign or, at any rate, with scarcely a physical sign ; and, on the other hand, a large bulging tumour may present all the possible signs in a patient who is quite free from cough, pain, and shortness of breath. The symptoms arise from two causes—the aortitis of the root of the vessel, and from pressure. Early in the disease the aortitis may be associated with severe pain, often recurring in attacks of angina pectoris. The sudden splitting of the intima over a patch of mes-aortitis may cause severe and agonising pain. It is interesting to note in how many cases the patients have at the onset attacks of pain of the greatest severity, which gradually disappear as the tumour grows larger and the signs of aneurysm become manifest. I have reported a series of such cases, and have recently had under observation a man, aged forty-nine, who for two years had attacks of agonising pain in the chest, which had completely incapacitated him. For months he was unable to assume the erect posture. For five years now he has had a large aneurysmal sac to the right of the sternum. The attacks of pain ceased suddenly and have not returned. As a rule the symptoms of aneurysm are those of tumour, and their intensity depends on the situation, the rapidity of growth, and the size of the tumour. Certain functional disturbances are associated with its growth and may be the earliest symptoms complained of—palpitation of the

heart, unpleasant forcible throbbing, and pronounced vertigo or actual fainting. Almost all of the symptoms may be referred to compression of adjacent parts in the growth of the tumour, and these may be considered in detail. From the situation of the aorta in the narrow space, measuring a few centimetres only, between the spine and the posterior wall of the sternum, tumours growing either directly backwards or forwards are very apt to cause compression of adjacent parts, even before they reach a large size. A small sac from the posterior part of the transverse arch may cause the most intense symptoms without any physical signs. On the other hand, the tumour growing from the ascending portion may pass to the right and go into the pleura, pushing aside the lung, or forward through the chest-wall, and this situation may contain quite large tumours which have caused very few symptoms. From the terminal portion of the arch just beyond the left subclavian, the sac may grow into the pleura compressing the lung, or into the lung itself.

Pain is one of the earliest and most distressing results of compression. As already mentioned, this may be associated with aortitis. When due to compression it is one of the most constant and enduring features of the disease. When one considers the richness of the nerve plexuses in the neighbourhood of the heart and the close relations of large nerve-trunks to the aorta, it is indeed surprising that so many patients with aneurysm of the arch have comparatively little pain. The character of the pain varies a good deal in different cases. There may be (1), as already mentioned, anginal attacks occurring very early and before any symptoms are present, but occasionally they are met with throughout the course of the disease, either with all the well-known features of paroxysmal angina or with modifications. (2) Neuralgic pains, shooting up the neck, cervico-occipital, down the arm, or in the course of the intercostal nerves. The former are very common and may be associated with numbness and tingling of the fingers. The patient may complain of ordinary cervico-brachial neuralgia. The attacks may vary greatly in intensity and in some cases may disappear for weeks, whilst in other instances they form a most persistent and distressing feature. (3) In deep-seated aneurysm associated with erosion of the bone and compression of the nerve-roots near the spine, the pain may be of an intense boring character without intermission and often requiring large doses of morphine for its relief. It is surprising how little pain may be caused in the process of erosion of the costal cartilages and the sternum. The most severe and persistent pain is met with in aneurysm of the descending thoracic and abdominal aorta; it may be of a characteristic girdle form or it may take the form of an ilio-lumbar neuralgia. In connexion with the formation of a dissecting aneurysm, the rupture of the intima is associated with intense pain usually in the region of the heart, and with symptoms of profound collapse which may pass off in the course of a few hours. It is a good practical rule, as laid down by Gairdner, that " whenever a case of obstinate or frequently recurring pain, such as

might, constructively, be due to pressure upon nerves or upon solid parts, and such as is not fairly in accordance with some disease known to exist in the organs of the thorax or abdomen, the suspicion, at least, of an aneurysm ought in all cases to arise." Drs. James Mackenzie and Head have called attention to the sensitiveness of the skin areas on the chest and arm in connexion with thoracic aneurysm. The skin may be sensitive to touch in the region of the nipple along the left sternal border and the left side of the neck over the sterno-mastoid muscle.

Certain special nerves may be compressed or irritated with the production of remarkable symptoms. Hiccup may be caused by irritation of the *phrenic nerve* and in rare cases paralysis of the diaphragm has followed its destructive compression. The *pneumogastric nerve* may be stretched and even destroyed on the sac without causing any special symptoms. In some cases nausea and vomiting and dyspeptic troubles generally have been attributed to irritation of this nerve and its branches. The pulmonary affections attributed to implication of this nerve are most frequently due to compression of the trachea or bronchi.

Compression of the Sympathetic.—Macdonnell, senior, of Montreal, in 1850 described a case in which a malignant tumour pressed on the sympathetic, and thus caused contraction of the pupil and ptosis on the same side. Walshe in 1853 noted contraction of the pupil in connexion with aneurysm; Gairdner in 1854 called special attention to it, and in 1858 John Ogle exhaustively discussed the whole question. The common features attributed to compression of the nerve are contraction or dilatation of the pupil on the affected side, and certain thermic and secretory phenomena. Pressure on the sympathetic cord itself, a rare event in aneurysm of the aorta, may be associated with all these characteristic symptoms. Dilatation of the pupil on the affected side occurs when there is simple irritation, permanent contraction when there is complete paralysis; a slight drooping of the eyelid, with contraction of the eyeball itself, increase of heat and redness of the ear and flushing with sweating of one side of the face may be present. In a few instances the sweating is very profuse; I once saw it extend to the side of the chest and to the right arm, and the skin of the hand was like that of a washerwoman.

The *oculo-pupillary features* of aortic aneurysm are of special interest. Thought formerly to be due always to implication of the sympathetic, they are in reality of varied origin. (1) In rare cases as just mentioned the cord of the sympathetic is involved. (2) The anisocoria or inequality of the pupil present in a very considerable number of cases has been shewn by Drs. Cecil Wall and Ainley Walker to be due to local vascular conditions. In twenty-six consecutive cases of thoracic aneurysm with inequality of the pupils a definite relationship existed between the state of the pupils and the arteries. As is well known, low blood-pressure is associated with large pupils, contracted pupils with a high pressure; and these authors found that in aneurysm there was a relation between the state of the pupils and of the arteries—where the temporals or

radials were small the pupil was large. Compression or obstruction of the carotid in the neck is associated with enlargement of the pupil on the same side. In one case of aneurysm at the root of the neck in which the pupils were equal, deligation of the right common carotid was followed by enlargement of the right pupil, and an operation is reported on the carotid artery in which the same sequence followed. This is, to my mind, by far the most satisfactory explanation as yet given of this common feature in aneurysm. (3) Inequality of the pupils, myosis, and absence of the reaction to light in aneurysm may be parasyphilitic manifestations, and be associated with tabes. This combination of aneurysm, absent knee-jerks, lightning pains, and oculo-pupillary phenomena has been called in France, Babinski's syndrome.

Pressure on the Recurrent Laryngeal Nerve.—Aneurysm of the transverse portion of the arch is particularly likely to press on the left recurrent. The large saccular aneurysm of the ascending aorta may pass far enough over to implicate the right recurrent. In the former the nerve may be compressed by very small tumours. In the rare cases in which both nerves are involved there may be two aneurysms. It is still an open question whether irritation of the recurrent can cause attacks of spasm of the larynx with dyspnoea and aphonia, and in some cases loss of consciousness, since it has not been proved that the recurrent laryngeal nerve in man contains centripetal fibres, and indeed it is more likely that when these symptoms do occur, they are due to pressure upon the pneumogastric nerve. These differences were well pointed out many years ago by the late Sir George Johnson.

Compression of the recurrent laryngeal nerve causes unilateral paralysis, which is on the left side in two-thirds of the cases. As Sir Felix Semon has pointed out in cases of organic progressive paralysis affecting the roots or trunks of the motor nerves of the larynx, the abductor fibres of the recurrent laryngeal always [1] suffer first. He has discussed the whole question in Vol. IV. Part II. p. 260, and it is therefore sufficient to state here that, if the abductor fibres of the recurrent are gradually disabled by the increasing pressure of an aneurysm, paralytic contracture of the adductors in conformity with general neurological laws gradually supervenes, and forces the affected vocal cord into the middle line. When it is fixed in this position there is no dyspnoea, and the voice at first remains perfectly normal. This is of great importance from the point of view of diagnosis, since it is only by methodical laryngoscopic examination in cases of suspected aneurysm of the aorta that the laryngeal complication can be detected when in its early stages. Later on, when the pressure further increases, first paralysis of the internal tensor (the internal thyro-arytaenoid muscle) is superadded, and then the paralysed vocal cord, although still standing *in* the middle line, appears somewhat excavated ; eventually the adductors too become paralysed, and the cord recedes into what is called the " cadaveric " position. In these stages alteration of the character of the voice is always present.

[1] Only one exception to this law has hitherto been recorded.

As Walshe puts it, " The speaking voice may be husky, muffled, cracked, and hoarse, or simply weakened or tremulous and variable in pitch or actually lowered in register." The epithet "cracked " is often applied to it, and sometimes the voice may be reduced to a whisper. With bilateral paralysis, which is certainly very rare in aneurysm and is not often complete, there is more or less stridor during exertion.

Associated with laryngeal paralysis there may be paroxysmal dyspnoea with stridor and retraction of the intercostal spaces and epigastrium. This is the most distressing of all the features of aneurysm. It is due to irritation of the vagus, for Sir G. Johnson shewed that bilateral spasm may result from irritation of one vagus only, an observation which has been confirmed by experiment (Horsley and Semon). In bilateral abductor paralysis, which is very rare in aneurysm of the aorta, the dyspnoea and stridor are not paroxysmal but continuous, although they may be aggravated from time to time either by spasm or direct compression of the trachea and bronchi. In a few cases a double stenosis may exist, namely, there may be double abductor paralysis in the larynx and direct compression of the trachea or bronchi, both symptoms being due to an aneurysm. In such cases the performance of tracheotomy will not, of course, relieve the patient if the compression-stenosis is considerable, unless in addition a Koenig's cannula or a soft tube is successfully passed through the compressed part of the trachea. These cases of double stenosis, however, are more frequent in carcinoma of the oesophagus than in aneurysm of the aorta. Gerhardt states that one of the most important points is that, if the obstruction is in the larynx, this organ makes violent respiratory excursions, whilst in tracheal stenosis the larynx remains still ; " in spasmodic and stridulous breathing movement of less than one centimetre is a certain sign of tracheal or tracheo - bronchial stenosis."

Compression of the Wind-pipe and Bronchi.—Often the very first symptoms are associated with irritation of the trachea, causing cough, more particularly when the sac is near the bifurcation. It may be a dry, irritative cough, occurring chiefly on exertion and coming on in paroxysms. The characteristic brassy cough is due to compression of the air-tubes and not to the laryngeal paralysis. When there is tracheitis there is much secretion which may be blood-stained. There may be paroxysms of intense severity associated with dyspnoea and stridor. An extreme degree of compression of the wind-pipe may exist with very little dyspnoea if the patient be at rest, but the slightest effort or an attempt to assume the erect posture at once brings on a cough with a noisy stridor. When a small sac projecting from the posterior wall of the transverse arch grows directly backwards upon the bifurcation of the trachea, there may be orthopnoea with paroxysms of suffocative dyspnoea and stridor without a single physical sign pointing to the nature of the compression. The pulsations of an aneurysm compressing the trachea can be seen by means of the bronchoscope (*vide* Vol. IV. Part II. p. 310).

Very remarkable symptoms may be caused by slow compression of a

main bronchus or of its principal branches ; the respiratory murmur may be absent or greatly diminished on one side. When the process is slow, the lung or one lobe may become atelectatic and gradually fibrotic, and sometimes this is associated with recurring haemorrhage into the bronchi.

Figure 84 from the first edition of this work shews this lobular condensation. But much more frequently the aneurysm causes retention of the secretion and intense bronchiectasis with expectoration of large quantities of mucus and pus. There may be irregular fever with sweating and emaciation, so that the patient may be sent to hospital for pulmonary tuberculosis. In the Montreal General Hospital I saw several cases of this sort with George Ross, who used to speak of them as "aneurysmal phthisis." Dilatation of the bronchi may follow, but more commonly the lung becomes consolidated and the bronchi filled with inspissated pus and there may be small abscesses and cavity formation. One lobe or the entire lung may be in this state.

FIG. 84.—Surface of the left lung shewing lobular condensation from old and recent haemorrhage into the left bronchus. The differences of colour and of prominence of the lobules are due to differences in date of the haemorrhagic condensation ; the older haemorrhage being nearly decolorised, and in part absorbed, or converted into puriform matter which is seen at some points to be forming small abscesses below the surface. (Gairdner.)

Compression of the Lung.—By the growth of the sac the lung may be pushed aside and slightly compressed, or in other instances it may form part of the wall of the sac. Very large tumours may lead to extensive compression of the lung, the upper lobe of which may be atelectatic and fibroid. When the sac grows directly into the lung, more important symptoms result. Even a small sac, growing from the convexity of the arch into the right upper lobe or from the terminal portion of the arch upwards into the left upper lobe, may early become adherent to the lung and grow directly into its tissue. Haemoptysis almost invariably follows, and may be early and even fatal before there are any features suggesting aneurysm. In other cases the sac may grow into the lung and form a large tumour completely filled with laminated clots, as in a remarkable specimen in the McGill Museum shewing a large portion of the left lung occupied by an aneurysm which is completely obliterated by firm thrombi.

Compression of the Oesophagus.—In a majority of cases aneurysm of the aorta does not involve the gullet. Dysphagia may result from spasm in

connexion with pressure on the recurrent laryngeal. Small sacs growing directly backwards may compress it before there are any suggestive physical signs. The dysphagia is rarely extreme, but it may be sufficient to prevent the patient from swallowing solid food, and it may come on in paroxysms. The condition may be mistaken for simple stricture or for cancer. When ulceration or sphacelus occurs, swallowing may be very painful and the dysphagia increases rapidly. Gairdner states, on the authority of Sir Spencer Wells, "that one of the most consummate surgeons of the last generation had been so unfortunate when exploring a case of dysphagia as to plunge an oesophageal sound into the sac of an aneurysm lying close to and compressing the oesophagus, with the result that the patient died in the consulting-room." Janowski describes a similar case. Compression may be followed by necrosis of the wall and ulceration with pain in swallowing. Perforation may take place from a small sac and the patient may bleed to death into the stomach, or the blood may be vomited. As in the trachea, the perforation is not always fatal and the orifice may be closed by thrombi. The pulsations of the aneurysmal sac have been registered by means of an oesophageal tambour, but the procedure is not without risk. An abdominal aneurysm or one of the lower end of the thoracic aorta may compress the cardia and cause great dilatation of the gullet, with regurgitation of food and great emaciation.

Compression of Blood-vessels.—The pressure may be on either cava, on the innominate, or on one of the subclavian veins. In the common aneurysm of the arch congestion of the veins of the neck and head, and sometimes of one or other of the arms, is a frequent symptom. One side of the face and neck may be more engorged than the other. In view of the situation of the superior cava the rarity of complete compression and obliteration of its vessel is remarkable. Narrowing is common enough with engorgement of the vessels of the upper part of the body. Obliteration due to aneurysm was present in only 4 of my 29 collected cases of complete obliteration of the superior cava. In these circumstances the collateral circulation is carried on through a number of channels : (1) When the obliteration is above the point of entrance of the vena azygos major, a large amount of the blood from the arms and trunk finds its way into this vein through communications of the intercostals with the internal mammary veins. (2) Over the surface of the chest the plexus of mammary veins enlarges and the swollen subcutaneous tissues of the entire front of the chest become occupied by a system of greatly distended veins. These may be seen in and beneath the skin as tortuous channels the size of the finger and converging to two or three large vessels which unite with the epigastric veins. On the front of the abdomen are seen large convoluted vessels which empty below into the femoral veins. In some cases the venous plexuses are entirely subcutaneous. In others the veins of the skin itself are dilated and give the general surface a purplish-red hue. So distended may the superficial mammary veins become that in the large sinuses thrombi form which

may ultimately calcify, forming vein-stones.   (3) Extensive communications exist between the deep cervical and the vertebral veins with the intercostals and the whole network of veins along the front of the spine. These communicate freely with the branches of the vena azygos major, or when the orifice of that vein is obliterated numerous channels are established between the lumbar vessels and the tributaries of the inferior vena cava.   Perforation into the superior cava will be considered with the arterio-venous aneurysm (*vide* p. 667).   Compression of the inferior vena cava is not common in aneurysm, so that we do not often see signs of congestion of the lower extremities and the formation of an extensive collateral circulation.   Compression of the vena azygos major may cause oedema of the chest-wall or effusion into the right pleura.   Symptoms due to compression of the *thoracic duct* are rare, but there may be great enlargement of the abdominal lymphatic vessels with varices and lacunae, as noted by Morgagni in one of his cases.   Compression of the pulmonary artery is not uncommon, but is rarely associated with any symptoms.   It is stated that gangrene of the lung has been caused by it ; perforation of the vessel will be considered on p. 667.

Compression of the *spinal cord* may follow erosion of the bodies of the vertebrae.   The cases are rare.   I have not seen one, but several are on record ; this event is one of the causes of painful paraplegia.

Symptoms due to Rupture.—Rupture of thoracic aneurysm into the air-passages is very common and causes *haemoptysis*, which is by far the most frequent form of external haemorrhage.   The bleeding may come from erosion of the trachea or of a bronchus, or directly from the lung tissue.   The common situations are just above the bifurcation and in the left bronchus.   In a few cases the sputa are bloody for weeks or months, as the result of a granular tracheitis.   As a rule, the haemorrhage is profuse and rapidly fatal.   In other instances small bleedings occur for weeks or months, especially when the trachea is eroded and the laminae of the sac have blocked the orifice.   There may be two or more orifices, or the trachea and one bronchus may be perforated.   Recovery, however, may follow a profuse and almost fatal haemorrhage.   A patient whom I saw with Dr. Fussell, of Philadelphia, lived for four years after his first very severe haemorrhage.   The famous surgeon Liston, in July 1847, had a feeling of constriction at the top of the wind-pipe and slight difficulty in swallowing ; this was followed by a profuse haemorrhage of from 30 to 40 ounces, from the effect of which he nearly died.   It is interesting that Liston himself suspected aneurysm, but neither Watson nor Forbes could find anything in the chest.   The symptoms were greatly relieved by the haemorrhage and he was able to return to work, but in October the symptoms returned and on December 6th he died in a paroxysm of dyspnoea.   An aneurysm was found compressing the trachea. T. W. Clarke has reported a case in which sixteen haemorrhages occurred between July the 23rd and September the 15th, the amount of blood ranging from a few ounces to 36, a total of 14 pints in seven and a half weeks.   Here the bleeding came from the small bronchi of the right

lung into which the sac projected. Gairdner reports a remarkable instance in which a man, aged forty at the time of his death, had been the subject of aneurysm for about ten years. Five years before his death he had a severe haemoptysis, and then occasionally he had rusty-coloured sputum and sometimes a more copious haemorrhage, in one of which he died.

The deep-seated sac growing into the middle of the left lung or into the upper lobe may be associated with cough and haemoptysis, and simulate tuberculosis. As the sac grows it compresses the main bronchus, leading to retention of secretion and fever. Gairdner's comment on this may be here quoted : " In cases of aneurysmal haemoptysis (to which the majority of observations refer) it has been established (*Clin. Med.* p. 520) that haemorrhages short of a directly fatal result may take place for weeks or for months in the form of (1) a frothy bronchitic sputum streaked with blood ; (2) a rusty sputum very like that of pneumonia, but usually more abundant, more frothy, and less viscid ; (3) a deeply-dyed purple or brownish-purple sputum, like the so-called ' prune-juice ' expectoration, closely resembling that of pulmonary haemorrhagic condensation from valvular disease of the heart ; (4) any of the preceding, alternating with small discharges of pure, unmixed, but generally imperfectly coagulated blood. When such haemorrhagic sputa approximate to the characters observed in pulmonary condensations, it is legitimate to infer that such condensations exist ; and it is probably quite impossible, in some cases at least, to distinguish the aneurysmal from other forms of such condensations ; but it is desirable, none the less, to have it clearly in view that such changes may be due to an aneurysm pressing on a main bronchus. The pulmonary alterations thence arising may vary almost indefinitely from recent condensations, with partial lobular collapse of the lung (and giving the impression as if blood had been pumped backwards into it through the bronchus), and infiltrations of older standing, ' nodulated and dense, some violet-coloured, others of a sandstone-gray tint.' In other cases, also, softening and ulceration of the tissues of the lung take place, leading up to a true bronchial and aneurysmal phthisis, of course non-tuberculous."

Oesophagus and Stomach.—When the sac perforates the oesophagus, or in abdominal aneurysm the stomach, blood is brought up in large quantities. This is much less common than haemoptysis. Perforation of the gullet occurred in only 9 out of 226 of Crisp's collected cases, and in 40 of Boinet's series of 195 cases of rupture. Weeks or months before perforation occurs pressure on the oesophagus may lead to erosion, and bleeding may follow from the granulations, or the laminae may be exposed and the sac weeps. The usual situation for the rupture is where the arch crosses the gullet. The wall may be much stretched, and shew small superficial ulcers ; in other cases there are two or three perforations. Gangrene may occur, with perforation of the lung or pleura, and a case is on record in which the aneurysm perforated a carcinoma of the gullet.

Rupture through the skin is a comparatively rare event in aneurysm of

the aorta, occurring in only 3 or 4 per cent of the cases. It is not uncommon to have necrosis of the skin, and even exposure of the laminae of fibrin, and the sac may weep for months, and yet the patient may die of internal haemorrhage or some complication. William Hunter reported the case of a man with an aneurysm perforating to the right of the sternum, in whom the sac bled for weeks at intervals from an orifice plugged by a coagulum which protruded and retracted with the systole and diastole of the heart. A sudden cough burst out the plug, and "the blood gushed out with such violence as to dash against the curtain and wall, and he died not only without speaking, but without a sigh or groan." The most common site of external perforation is just to the right of the sternum. In the process of external rupture the skin becomes reddened and gradually necrosed, sometimes with the formation of a small sub-cutaneous abscess. In rare instances a cutaneous perforation has cica-trised and the patient has lived for several years. Sometimes the rupture takes place into the subcutaneous tissues, with the formation of a huge haematoma.

Internal Rupture.—Into the *pericardium* the small rapidly-growing sacs of the ascending aorta are very liable to rupture. The frequency with which this occurs is well-known by those engaged in medico-legal work, and is very much higher than indicated in the statistics of Crisp and of Sibson. There may be a small pin-point perforation or a tear of consider-able extent. The amount of blood in the pericardial sac varies from 200 or 300 cubic centimetres to as much as 1000 c.c. Pericardial adhesions may retard the outflow of blood. As a rule, death occurs suddenly, but there are instances on record in which the patient has lived for hours, or even days. Rupture into the *pleura*, most frequently into the left, is a common mode of termination. There are three situations in which it is met with, one in front on either side as an aneurysm of the arch increases, another behind in the aneurysm of the descending aorta, and a third when the abdominal sac ruptures through the diaphragm.

It may be preceded for some days by a pain in the chest or by signs of pleurisy. The amount of blood lost is usually very large, amounting to three or four pounds, and the patient dies rapidly. Recovery may take place, and cases with recurring pleural haemorrhages are on record (Stokes). The blood may be encysted between the pleura and the lung; it may leak slowly through a small orifice, and form a pleural haematoma. Rupture into the *mediastinal tissues* is also common, but it is not necessarily fatal. Small ruptures may occur with the effusion of a moderate quantity of blood, which gradually becomes absorbed. In other instances a diffuse aneurysm is formed, and the blood may pass up the neck, forming a large tumour. Death is not often directly due to rupture into the mediastinum. Rupture into the *retroperitoneal tissues* is a very common event in aneurysm of the abdominal aorta. There may be repeated small haemorrhages, associated with an increase of the pain in the back. The blood may pass down in front of the psoas muscle and form a tumour in the groin. In other instances large diffuse sacs are formed. Rupture into the *spinal*

canal was described by Laennec, and a few cases have been recorded. It usually follows signs of compression. Death takes place within a few hours.

Rupture into the *great vessels* and into the heart will be referred to in the section on arterio-venous aneurysm (p. 667).

The relative frequency of the different regions of rupture are given in the statistics of the *Bulletin de la Société anatomique* of Paris from 1826 to 1906 by Boinet. Of 349 aneurysms, 195 terminating in rupture, the more important situations were : into the left pleura 36, the right 10 ; the pericardium 29, the left lung 13, the left bronchus 15, the trachea 17, the oesophagus 40, spinal canal 3, and externally through the skin 9.

Physical Signs. — *Inspection.* — Patients with aneurysm are usually vigorous-looking subjects, young or middle-aged, often with an appearance of such good health that a suspicion of aneurysm, or at any rate of cardiovascular disease, is aroused when such men are seen in a hospital ward. Marked suffusion of the face, with dusky infiltrated conjunctivae, may be present when the sac compresses the veins near the heart. Occasionally the vessels on one side of the face are much distended. Inequality of the pupils is common, and may at once attract attention. The conditions under which it occurs have already been referred to. The head may jog with each cardiac impulse, particularly when the aortic valves are incompetent. Inspection of the neck may reveal great engorgement on one or both sides, and occasionally there is enormous distension of the right jugular sinus. The carotid may pulsate forcibly on one side, whilst none may be seen on the other. The pulsation of the aneurysmal tumour may itself be seen above the sternum. Visible tracheal tugging, with systolic retraction of the entire box of the larynx, may be seen. In thin subjects lateral deviation of the trachea may be obvious. When the sac reaches the thoracic wall, the diagnosis may, as a rule, be made " at sight." Good light, good eyes, and a certain method of routine are required for successful examination. The patient should be stripped and seated on a stool facing the light, or with a good side light on the chest, and it must be remembered that inspection from the front does not always bring out all that can be seen ; a slight heaving impulse of the sternum or moderate pulsation to the right, not seen when looked at directly, may be very evident when looked at from the side with a good light falling on the chest. Aneurysmal pulsation is most common (1) to the right of the sternum in the 2nd and 3rd interspaces, (2) on the manubrium, (3) to the left in the 2nd and 3rd interspaces, (4) above the sternal notch, and (5) at the back in the left interscapular region, or in the infrascapular area. It is well to make the inspection in a routine manner, and not to omit the back after carefully examining the front of the chest. A network of distended veins is not uncommon on the skin of the upper part of the chest, and it may extend over the shoulders and be continuous with the large vessels in the neck. Very great enlargement of the mammary veins is not so often seen in aneurysm as in tumour, but in a few cases with compression of the superior vena cava the whole of the

front of the chest is occupied by a large plexus of vessels communicating with the epigastric veins. When looking for pulsation it is well to bear in mind the different types of it as seen in the chest. First, there is a general shock, such as occurs in violent beating of the hypertrophied heart or of an aneurysm. In great enlargement of the heart, particularly when associated with aortic insufficiency, or in acute fever, or in anaemia, there may be remarkable throbbing of the whole front of the chest due to the excited action of the heart. Even without organic disease of the valves, as in Graves' disease, neurasthenia, and severe anaemia, this diffuse throbbing, particularly when associated with marked pulsation of the subclavians, may lead to the diagnosis of aneurysm. The shock may be of such intensity as to jar the entire body, and the pulse may be counted by the movements of the patient's head, or the bed may receive a distinct jarring shock. Secondly, limited to one side of the chest, to the right mammary or subclavian regions, or to the lower axillary region, there may be a diffuse impulse which differs from the general thoracic shock. It is seen in its characteristic form in pulsating pleural effusion, gaseous or liquid, and I saw a case in St. George's Hospital in which a suppurating hydatid cyst in the anterior mediastinum pulsated in this manner. It is remarkable that a limited area of pulsation on one side of the chest may occur without any obvious cause; I have seen chronic mediastinitis accompanied with deceptive pulsation simulating aneurysm. The patient, aged fifty-nine, had increasing dyspnoea, cough, pain in the chest, a cracked voice, and in the 2nd left interspace extending towards the axilla was a diffuse impulse, very definite when the breath was held. The fluoroscope shewed an indefinite shadow to the left of the sternum. There was also slight tracheal tugging, and naturally enough the diagnosis of aneurysm was made; at the necropsy there was a condition of posterior chronic mediastinitis. Sailer has reported a case in a Russian Jew, aged twenty-six, with a normal but not very vigorously beating heart, and with marked throbbing of the abdominal aorta. There was slight, definite, visible systolic pulsation of the whole right side of the thorax, perceptible also on palpation. This type of throbbing may be most deceptive in cases of severe anaemia, and occasionally it is singularly localised, as in the case reported by A. R. Edwards; over the lower left chest there was a diffuse pulsation, extending horizontally from the angle of the left scapula into Traube's space and the epigastrium. "The pulsation was vigorous and distinctly expansile to both the eye and hand." A systolic bruit was heard over it, and the case was regarded as one of aneurysm of the thoracic aorta, but the necropsy shewed a moderate arteriosclerosis of the aorta. Lafleur has reported a very similar case with pulsation in the same region without aneurysm. Thirdly, the pulsation of aneurysm, which may be of two kinds. In the deep-seated tumour, which does not come in contact with the chest-wall, a shock may be communicated similar to that which is seen and felt in the chest in cases of hypertrophy of the heart. The true aneurysmal impulse is only seen when the sac reaches the chest-wall. When localised, the visible

expansile character is readily appreciated. It is usually single, systolic in time, occasionally undulatory or even double. It may be well to state the regions of the chest in which visible impulses may be seen, which must not be mistaken for aneurysm. (1) The throbbing of the conus arteriosus in the 2nd left interspace—very common in young persons and in thin chests, and seen particularly well during expiration. (2) Pulsation of the heart in the 2nd, 3rd, and 4th left interspaces, extending as far out as the nipple line in cases of fibrosis and retraction, from any cause, of the upper lobe of the left lung. (3) Cardiac pulsation in the 2nd, 3rd, and 4th right interspaces in connexion with similar conditions of the right apex. (4) Effusion on either side of the chest may so dislocate the heart that there is a marked impulse at or outside the nipple-line on either side. (5) Throbbing subclavians seen in the outer half of the infraclavicular regions, usually bilateral; this is met with in thin-chested persons, in neurasthenia, in early tuberculosis, and in anaemia. Sometimes it is unilateral, and when accompanied with a thrill and a murmur it may form a mimic or phantom aneurysm. (6) In the back part of the chest visible pulsation is nearly always aneurysmal ; but occasionally, in Broadbent's sign, the tugging may be so limited and localised in one interspace that it simulates pulsation, but palpation easily corrects this.

Inspection of the arms and hands may give valuable information. The radials and brachials may shew visible pulsation, particularly when aortic insufficiency coexists. Swelling of one or both upper extremities may be present when a sac, springing from the ascending aorta, has compressed the superior cava. More commonly, the arm on one side is congested and swollen. Pallor and sweating may be seen in one hand and arm as a result of pressure on the sympathetic. Clubbing of the finger-ends I have seen twice—once on the right side, once on the left. It seems to be associated with peripheral stasis (*vide* Vol. III. p. 66).

Palpation. — Over an aneurysmal sac which has reached the surface there may be felt: (1) The true aneurysmal impulse. To appreciate its character it must be remembered that it is synchronous with the cardiac impulse, and to learn to recognise it, palpation of an actively beating apex should be carefully practised. The remarkable vigour and intensity, the impossibility of resisting it, the proximity to the fingers, and the definite expansile quality, are its important features. Of course, these features can only be recognised when the aneurysm reaches the surface, but even when the sac itself cannot be palpated there may be communicated to the chest-wall a forcible heave, which is entirely different in sensation from the ordinary shock. In the deep-seated tumour beneath the manubrium this may sometimes be appreciated best by bimanual palpation—one hand upon the spine and the other forcibly compressing the sternum. The communicated shock or jar, which is felt over the chest in a case of hypertrophied heart or a throbbing aorta, is diffuse, without localisation, without any punctuate, heaving quality, and without that sense of forcible expansion directly

beneath the fingers which is so characteristic of the cardiac and of the aneurysmal beating. (2) Over the aneurysmal sac near the heart may be felt a shock either of a thudding first sound, or, what is much more common, of the sharp flap of the second sound ; sometimes the shock of both. The second is of great diagnostic importance, and at times may be felt, by the slightest application of the finger to the sac, as a short snapping shock, and coincident with it a diastolic impulse may be felt. (3) A marked vibratory thrill may be felt, usually systolic in character, much more rarely diastolic, and not often double. It is not a special feature of aneurysm of the thoracic aorta, and is absent in a great majority of cases. It is relatively more common in aneurysm of the abdominal aorta. A diastolic thrill is exceedingly rare. Forcible compression of the sac should not be attempted on account of the danger of detaching portions of clot, an accident which has been followed by hemiplegia.

Tracheal Tugging.—When adherent to the wind-pipe, the pulsations of the sac may cause visible depression of the box of the larynx, or when the fingers are pressed upon it a downward tug or jar may be felt. In some cases the finger placed upon the box of the larynx appreciates this tracheal tug. In other instances to bring it out the procedure first described by Surgeon-Major Oliver should be carried out : Place the patient in the erect position, and direct him to close his mouth and elevate his chin to almost the full extent ; then grasp the cricoid cartilage between the finger and thumb, and use steady and gentle upward pressure on it, when, if dilatation or aneurysm exist, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand." It is present in a large proportion in all cases of aneurysm of the transverse portion of the arch, and the very great value of the sign is not diminished by its very occasional presence in tumours other than aneurysms.

Palpation of the Arteries.—Changes in the sac or pressure by it upon the large vessels may lead to retardation, feebleness, or obliteration of the pulse in the peripheral arteries. The carotid pulse on one side may be feeble or obliterated. Inequality of the radials is more common. The right is more frequently smaller than the left, and it may occur without any alteration in the carotid pulse on the same side. This may be due to thrombosis within the sac, whereby the orifice of the innominate is narrowed, or the large sac may compress the subclavian. Occasionally the radial pulse may be smaller on the side opposite to that on which the sac is prominent. A feeble pulsation in the left radial, when there is a projecting sac from the ascending aorta on the right side, may be caused by a small secondary aneurysm, or it may be due to atheromatous narrowing of the orifice of the left subclavian. Harvey appears to have been the first to notice this change of pulse in aneurysm, for he describes in Chapter III. of the *De Motu Cordis* the following case :—" A certain person was affected with a large pulsating tumour on the right side of the neck, called an aneurysm, just at that part where the artery descends into the axilla, produced by an erosion of the artery itself, and daily increas-

ing in size ; this tumour was visibly distended as it received the charge of blood brought to it by the artery with each stroke of the heart ; the connexion of parts was obvious when the body of the patient came to be opened after his death. The pulse in the corresponding arm was small in consequence of the greater portion of the blood being diverted into the tumour and so intercepted." The retardation of the radial pulse on one side may be perceptible to the finger. The difference in the character of the pulse in the two radials is very well shewn by the sphygmograph. The tracing of the pulse-wave on the one side may be greatly diminished in amplitude. Simultaneous tracings of the two radials may shew retardation of the pulse on one side by as much as $\frac{5}{100}$ of a second. The capillary pulse is seen when aortic insufficiency coexists.

A very large aneurysm of the transverse arch may cause great feebleness of the pulse in the arteries of the head and extremities. In one instance, in a case of very large aneurysm of the thoracic aorta, no pulse was felt in the abdominal aorta or in the femorals. Gairdner remarks that an aneurysm low down in the course of the aorta has been practically cured by one higher up, the force and impulse of the blood-stream having been so much checked by the latter as to promote firm coagulation and entire cessation of the pulsation originally present in the former.

The arteries in aneurysm are, as a rule, sclerotic, but in a good many young subjects the syphilitic arteritis, upon which the aneurysm depends, is entirely limited to the aorta.

Percussion.—In very small sacs there may be no changes, but when the tumour reaches the thoracic wall the percussion-note is altered, the situation depending upon the point of contact of the aneurysm with the chest. Impairment of resonance, shading to dulness, is common to the right of the sternum, over the manubrium, in the left subclavian and mammary areas, or in the left interscapular region behind. Even when large, the deep-seated sac, entirely surrounded by lung, may cause very little change in the percussion-note. When the lung is compressed on either side various shades of tympanitic resonance may be brought out.

Auscultation.—There are no characteristic aneurysmal sounds or murmurs. The most constant abnormality heard over an aneurysmal sac is the intensification of the heart-sounds—the first dull and thudding, the second clear, ringing, and accentuated ; or the latter alone may be heard. This diastolic accentuation, when present, is a valuable diagnostic sign. In perhaps a majority of cases of aneurysm no murmur is audible, or at best a very soft systolic, which is propagated into the vessels of the neck. A to-and-fro murmur is present with insufficiency of the aortic valves. Occasionally a diastolic murmur is heard alone without incompetency of the valves. A continuous humming-top murmur, with systolic intensification, is diagnostic of a communication between the sac and one of the large vessels or one of the chambers of the heart.

Auscultation of the Lungs.—Pressure of the sac upon the lung itself, or more frequently compression of one bronchus, may lead to alteration of the pulmonary sounds. The breathing may be feeble over the whole of one lung, or of one lobe. In one case, with every pressure-symptom of tumour, including paroxysmal dyspnoea of a most aggravated character, the only physical sign was weakened breathing over the lower lobe of the left lung. A small aneurysm from the termination of the arch grew backwards and compressed the bronchus going to this part. Sonorous and sibilant rhonchi and harsh stridulous breath-sounds are common with compression of the trachea. Extreme compression of one bronchus, leading to bronchiectasis or destructive changes in the corresponding lung, may be associated with corresponding physical signs. Dr. David Drummond has called attention to the presence of a blowing sound heard in aneurysm when the stethoscope is placed over the trachea or sometimes at the open mouth. This phenomenon of so-called pulse-breath, when present, is very striking. A whiffing bruit may be heard by the patient himself, and it may be plainly audible when the ear is placed opposite the patient's mouth, or even at some distance from it (Packard).

The Blood-Pressure in Aneurysm.—The careful observations made at the Johns Hopkins Hospital, and by Dr. O. K. Williamson at the Middlesex Hospital, shew that whilst the arterial pressure is normal, or only slightly above the average, there is often a marked difference in the two brachial arteries. In examining 30 cases Dr. Williamson found in a majority a marked difference in the blood-pressure in the two arms, so that a difference of more than 20 mm. suggests aneurysm.

The State of the Heart.—As a rule, the heart is not enlarged Dilatation and hypertrophy occur in cases with aortic insufficiency and in such associated conditions as contracted kidney and widespread arteriosclerosis. Occasionally, without any obvious reason, the heart may be very large. In a man, aged forty, with a big saccular aneurysm of the descending aorta, the signs of hypertrophy were very prominent during life. At the necropsy there was no valvular disease ; the organ was greatly enlarged, particularly the left ventricle, which measured from the ring to the apex 12 cm., the walls being from 1·5 to 2 cm. in thickness. Large sacs of the arch dislocate the heart downwards and to the left, and, as is so well seen in the x-ray pictures, it assumes a more horizontal position. Occasionally a very large aneurysm growing downwards may gradually occupy the position of the heart, as in a remarkable case reported by Dr. S. Gee. A large sac of the descending aorta growing forward may flatten the heart and give a remarkably diffuse impulse on the front of the chest, or a double pulsation—the double jogging impulse of Hope.

II. **Aneurysm of the Descending Thoracic Aorta.**—No portion of the vessel is so free from aneurysm. The relative frequency is variously given ; in the combined statistics of Crisp, Lebert, and Myers the descending aorta was involved in 49 cases out of 404 ; of 64 cases of

aneurysm of the aorta among the first 2060 necropsies at the Johns Hopkins Hospital, this part was affected in 13 ; in 3 the sac occupied both the arch and the descending vessel, and in 1 the aneurysm sprang from the junction of the thoracic and the abdominal portions. Clinically, it is much the least frequently encountered, in fact it is less common in the wards than in the post-mortem room, owing to the latency of its course. Up to 1903, the first fourteen years of the work of the Johns Hopkins Hospital, only 15 cases were recognised, a number often exceeded in a single year by aneurysm of other parts of the aorta. The tumour may be small, and prove fatal by perforation into the oesophagus before causing any symptoms that attract attention. On the other hand, the slowly-growing sac may reach an enormous size, and, perforating the chest-wall, may form the largest aneurysmal sac met with. The relations of the vessel explain certain features of aneurysm of this part. It is apt to grow backwards and erode the bodies of the vertebrae ; of the 14 cases I reported, the spine was involved in 9. One or two bodies, or in some instances almost the entire spine, may form the posterior wall. One or two ribs may be destroyed close to the spine, or portions of four or five are eroded, and the sac perforates the chest-wall, forming a huge subcutaneous tumour. Connected with the erosion of the spine is the distressing symptom of pressure on the nerve-roots with pain of an agonising character. In a few instances the spinal canal is reached, and there is a pressure-paraplegia, or sudden death from rupture of the sac.

The pain may simulate angina, or there may be intercostal neuralgia with marked hyperaesthesia of the skin and points of tenderness at the angles of the ribs. Herpes zoster has been met with. It is remarkable how variable is the pain ; one case of my series, a robust healthy fireman, who came in with a huge pulsating tumour of the back, had very little pain, and yet the sac had eroded the 7th and 8th vertebrae, and destroyed large portions of the 6th, 7th, and 8th ribs. In other cases the pain is more severe and persistent than in any other form of aneurysm, and may require enormous doses of morphine ; one patient, for example, took as much as 38 grains in the day. In connexion with the erosion of the spine remarkable attitudes are assumed. One patient would sleep for hours bent double upon his knees with a couple of pillows under him ; another would rest for hours and even fall asleep leaning upon the window-sill ; and another would go to sleep in a chair, bent double, with his hands resting upon his insteps and the trunk on his thighs. From its close relation to the descending aorta the oesophagus is particularly liable to compression. Yet in my series difficulty of swallowing was not often complained of ; in fact it was present in only 2 cases, and in only 1 did perforation take place. In several of my cases the oesophagus at the necropsy appeared to be compressed by the sac, and yet the patient had not complained of dysphagia. Perforation of the gullet, with fatal haemorrhage, may occur without any previous symptoms. In a woman, aged thirty-five, who had always been very strong and healthy, death took

place suddenly in syncope. In the lower third of a healthy-looking thoracic aorta was a linear slit, 1·5 cm. in extent with clean-cut margins, directly opening into a small aneurysm, 5 by 5 cm., which had perforated the oesophagus. The sac may erode the oesophagus, and weeping of blood may occur for weeks before the final perforation. The vomiting of blood and melaena in aneurysm may not be due to erosion of the gullet. In one of my cases there was vomiting of blood on three occasions, which, of course, was attributed to erosion of the oesophagus, but the necropsy shewed that the bleeding came from an ulcer of the stomach.

Aneurysm of this part of the aorta is specially likely to cause pulmonary symptoms. The sac may grow upwards and forwards into the lung. In a specimen in the M'Gill University Museum the sac is seen to be embedded in the lung tissue. In Case 10 of my series the upper lobe of the left lung was almost entirely occupied by an aneurysm, which was not adherent to the trachea, oesophagus, or other structures. A large sac may compress the lower lobe or a large part of the lung, causing atelectasis with fibroid transformation. In these instances, as Stokes pointed out, there may be retraction of the left side of the chest. By the forward growth of the aneurysm the main bronchus, or the bronchus of one lobe, may be compressed, leading to bronchiectasis or extensive destruction of the lung, as already described under pulmonary symptoms. Rupture into the pleura is common in this form, and occurred in 3 cases of my series. Compression of the thoracic duct occurred in 2 cases of the same series.

Certain special symptoms of aneurysm in this situation may be referred to. Some of the cases are latent—3 in my series. A man, aged thirty-five, had a fracture of the lower jaw, which was wired in the out-patient department under ether. He recovered, and was able to dress himself partially, when he died with profuse haemoptysis. An aneurysm, 7 by 5 cm., projected from the beginning of the descending aorta and opened into the left bronchus. The latent aneurysm of the aged is not infrequently seen in this part of the aorta. It is from this vessel that very large sacs originate, which grow into the lung or the pleura and become consolidated, forming large tumours, the nature of which may be very difficult to recognise.

A man, aged seventy, had for more than fourteen years very anomalous thoracic symptoms—cough, attacks of haemoptysis, and husky voice connected with recurrent laryngeal paralysis. The left back from the spine of the scapula shewed impaired percussion and absence of breath-sounds, but neither pulsation nor bruit. There was slight retraction of that side of the chest, and the case was regarded by Dr. Palmer Howard as one of some obscure pulmonary trouble. At the necropsy there were two aneurysms of the descending aorta; one, the size of a large fist lined with very dense laminae of fibrin, had compressed the left lung, and flattened and almost occluded the left bronchus; the other sac sprang from the vessel just above the diaphragm. The most extraordinary case of this kind in the literature is reported by Sokolowski: "The patient, who held a prominent position in the German Government, had a

severe attack of dyspnoea in 1864. A physician, a friend, happened to come in during the attack, made a careful examination, and found, to his astonishment, impaired percussion over the upper right half of the thorax, with absence of the respiratory murmur, the heart's impulse being displaced a little down and to the left. From this time the patient had at irregular intervals severe attacks of dyspnoea of short duration, during which the radial pulse was very often impalpable. In 1869 the attacks became more numerous, and in March of that year he consulted Professor Oppolzer, who diagnosed an aortic aneurysm, and Professor Skoda, who diagnosed a mediastinal tumour. The patient improved during the summer and was worse in the winter. He was able to get about, although he had shortness of breath on exertion. In the winter of 1875-76, for the first time, he had bloody sputum. In the spring of the latter year he had a left-sided pleural exudate. In July 1876 he was in Brehmer's Institution, where he came under the observation of Dr. Sokolowski. He was then forty-three years old, well-built and well-nourished, but was extremely dyspnoeic and slightly cyanotic. The pulse was absent in the left radial, equal in the carotids. The apex-beat was in the seventh intercostal space in the axillary line. There was no heaving impulse in any part of the chest. Extensive absolute dulness was present over the greater part of the left half of the thorax. There was a loud tracheal rhonchus over the whole of the front of the thorax. Nothing was audible over the greater part of the area of dulness, and tactile fremitus was diminished. Behind there was a slight vesicular murmur to be heard. The dyspnoea increased, and he died July 29th, 1876. On opening the thorax the right lung extended to the middle line, but the whole of the rest of the visible field was occupied by a thick mass of connective tissue. The left lung was pushed up from behind, forcibly compressed, and airless. After some difficulty the heart was discovered at the left angle of the large mass, which was found to be a huge aneurysmal tumour of the entire thoracic aorta. The heart was not enlarged, and the valves were normal. The aneurysm lay between the sternum and the vertebral column and the ribs on the left side, filling the greater part of the left chest. The oesophagus and trachea lay in a groove in the back of the tumour. The aneurysmal sac began just 2 cm. above the orifice of the aorta. Its walls were of unequal thickness ; the posterior wall was covered with a dense coagulum as thick as the fist, and of the hardness of cartilage and much laminated ; the anterior wall was also covered with a dense, thick coagulum. As the cross-section of the sac shewed, the blood passed through the centre of the tumour in a very irregular sinuous channel. Evidently it was the enormous thickness of the laminae of fibrin that prevented the usual characteristic pulsation." It is specifically stated that there was no pulsation in the thorax and no murmur.

These two cases illustrate the chronicity and the great difficulty there may be in recognising the nature of certain cases of aneurysm in this situation. As a rule, the physical signs are fairly definite. In 11 cases of my series pulsation was visible. It may be diffuse, or there may be a definite circumscribed pulsating tumour. The left interscapular region is a favourite site. In large sacs the whole left interscapular region may pulsate. In 1 case there was well-marked pulsation in both interscapular regions. The sac may perforate the chest at the back and form a huge tumour. Anteriorly the pulsation may appear in the second,

third, and fourth left interspaces, or in the sixth, seventh, and eighth, when the sac pushes the heart aside. When the sac grows forward, just above the diaphragm, there may be marked pulsation in the epigastrium. With a large sac in this situation the pulsation may be very remarkable. In one of my cases the sac occupied the position of the heart, which was pushed far into the right chest, and there was an extraordinary width of impulse with an undulatory pulsation and a difference in time between the heart impulse and that of the aneurysm. The heart is usually pushed upwards and to the right, but in Sokolowski's case it was displaced downwards and to the left.

Complications of Thoracic Aneurysm.—The broncho-pulmonary features, due to pressure, have already been referred to. *Pneumonia* carries off a few cases. *Tuberculosis* is a not uncommon complication, as pointed out by Stokes. It has nothing to do with pneumogastric compression, but the local conditions favour the development of the tubercle bacilli. The lesions are often latent, and the cough and abundant expectoration are attributed to pressure on the trachea or bronchi. The early haemoptysis, when the sac grows into the lung, may lead to the diagnosis of pulmonary tuberculosis, and the same error is usually made in the cases with high fever, cough, sweating, and the septic state due to bronchiectasis and suppurative bronchopneumonia.

Pleural Effusion is a not uncommon event in aneurysm, and may be due to pressure on the veins, particularly the vena azygos, the associated cardiac conditions, or to an acute pleurisy. An effusion may completely mask the physical signs of aneurysm. The fluid may be blood-tinged. The terminal pleurisy, with exudate coming on abruptly with well-marked signs, or latent, is usually tuberculous. In a few cases the patient gets more and more feeble, wastes, becomes anaemic, and dies in what Stokes called aneurysmal cachexia. *Embolism* (of the cerebral arteries, of the aorta, or of a femoral) is an occasional cause of death. A few cases shew *mental symptoms* with suicidal tendency or a progressive melancholy.

III. ANEURYSM OF THE ABDOMINAL AORTA.—**Incidence.**—The ratio to aneurysm of the thoracic aorta is about 1 to 10. Sixteen cases occurred among 18,000 admissions to my wards at the Johns Hopkins Hospital. At Guy's Hospital between 1854 and 1900, in 18,678 necropsies there were 325 cases of aneurysm of the aorta, of which 54 were of the abdominal portion. Among 2200 necropsies at the Johns Hopkins Hospital there were 49 cases of aneurysm of the thoracic and 11 of the abdominal aorta. The incidence varies extraordinarily in different localities. Among 222 cases of aneurysm in 19,300 necropsies at Vienna there were only 3 of the abdominal aorta. I reported 16 cases in 1905, most of which had been in the wards of the Johns Hopkins

Hospital. J. H. Bryant's large figures indicate that this portion of the aorta is affected in about 16 per cent of the cases.

Sex.—Males are much more liable than females; of my 16 cases 2 only were in females, and of the 54 cases at Guy's Hospital 49 were in men.

Age.—As pointed out by Crisp years ago, the majority of the cases are under forty years of age. Of the Guy's cases there were under twenty years, 2; between twenty-one and thirty, 11; between thirty-one and forty, 23; between forty-one and fifty, 8; above fifty, 10. It is much more common in the labouring classes than in the well-to-do. Syphilis is an all-important factor; and strain and injury are perhaps more important than in aneurysm of the thoracic aorta. Lifting a heavy weight, a fall, a strain in recovering the balance, are common exciting causes, and not infrequently the patient mentions the exact date when the pain in the abdomen or the back began. Soldiers are particularly prone to it; 8 of the 49 males of the Guy's Hospital series had served in the army, and all had had syphilis.

Locality.—As Crisp's figures shew, a large majority are in the upper part, at or near the coeliac axis. Of the 54 Guy's Hospital cases 36 were in the neighbourhood of the coeliac axis, 11 at the orifice, 13 above the orifice, 12 below it, 5 at the orifice of the superior mesenteric artery, 3 at the orifice of one of the renal arteries.

Form.—The saccular is the most common, and may arise from a very small orifice. Dissecting aneurysm rarely starts in the abdominal aorta, but it may extend into it from the thoracic. Diffuse aneurysm is common owing to the frequency with which the saccular form ruptures into the adjacent tissues. Some of the largest blood-tumours known are produced in this way. The fusiform dilatation is rare, but a definite spindle is sometimes met with; in a remarkable specimen in the M'Gill University Museum the tumour is filled with beautifully laminated thrombi. The rarest variety is the arterio-venous, of which there were 2 out of the 54 in the Guy's Hospital series. The sac may arise from any aspect of the vessel, more frequently from the lateral or posterior. Erosion of the spine takes place in from 50 to 60 per cent of all cases.

Mode of Termination.—Rupture is relatively more frequent than in aneurysm of the thoracic aorta. In the Guy's Hospital series of 54 cases it occurred into the retroperitoneal tissues in 19, into the peritoneal cavity in 12, into the pleural cavity in 8, into the mediastinum in 2, into the pericardium in 1, and into the mesentery in 1. In some reported cases rupture has taken place into the stomach, the duodenum, the colon, the pelvis of the kidney, the gall-bladder, the bile-ducts, the urinary bladder, the inferior vena cava, and the spinal canal. Rupture into any of these cavities causes death very quickly. When the sac erodes the colon, stomach, or duodenum there may be weeping of blood and slight haemorrhages for weeks. The rupture into the retroperitoneal tissues, which is the most common, may lead to very remarkable changes, and when it occurs suddenly in a person in whom the condition has been

unsuspected, the clinical· picture may be that of the acute abdomen. I have known 4 cases operated upon, in 3 under the belief that appendicitis was present, and in one instance for an abscess. The diffuse aneurysm may gradually increase in size until a huge tumour is produced, as shewn in Fig. 85 ; and when the condition lasts for weeks or months and there is not any pulsation, the condition may be very difficult to diagnose. The pressure features of aneurysm of the abdominal aorta shew great variations. The tumour may compress the lower end of the oesophagus or the pyloric end of the stomach, and give rise to dilatation of the oesophagus or stomach. The ureter may be compressed, causing hydronephrosis ; and in one of my cases the left kidney was completely destroyed. Compression of the vena cava may cause great oedema of the lower part of the body. Local peritonitis over the sac is not uncommon.

Fig. 85.—Photograph of an abdominal aneurysm which lifted up the costal margin, and filled the whole of the left side of the abdomen.

Pressure on the nerves causes the most common and constant symptom —the pain.

Special Symptoms.—Pain is perhaps a more dominant feature in abdominal than in thoracic aneurysm. Occasionally there is no pain, but, as a rule, the sac growing forwards or backwards causes most severe pain. According to the situation of the sac it may be referred to the back or extend around the flanks. Very often in the early stages it shoots down the legs, or it may be referred to the hip, the sacro-iliac region, or to the lower part of the sternum. It may be continuous and of a gnawing, tearing character, or it may come on in crises of the most intense agony. The most intense pain is not always dependent on erosion of the spine, for it may be due entirely to stretching of the nerve-fibres over the sac. Nearly every one with large clinical experience can parallel the classical case reported by Beattie, which really first called attention to the importance of aneurysm of the abdominal aorta, and which is given fully by Stokes in his work on the heart.

Among other special features may be mentioned nausea and vomiting, which in two of my cases were so pronounced as to suggest serious disease of the stomach. Dysphagia may be caused by compression of the lower

end of the oesophagus, and, as already mentioned, pressure on the duodenum or pylorus may cause great dilatation of the stomach. Haematemesis is not always due to erosion, but may result from embolism of the gastric arteries or possibly from pressure on the vasa brevia. When the sac is high in the abdomen and projects upwards, cardiac embarrassment comes on early, and is manifested by palpitation, shortness of breath, girdle-pains, and sometimes by attacks simulating angina pectoris.

Plugging of the abdominal aorta may lead to gangrene of the legs. Embolism of the arteries of the legs is not common. Embolism of the branches of the abdominal aorta may occur,—of the gastric arteries giving rise to haematemesis, of the mesenteric causing sudden pain, abdominal distension, collapse, melaena, and death ; of the renal arteries, causing pain in the back and haematuria. Embolism is not very common ; it did not occur in any case in my series. Paraplegia, a rare symptom, may be caused by plugging of the abdominal aorta and anaemia of the cord or by erosion of the spine and direct compression of the cord. Paralysis of one leg may be caused by pressure on the nerves. In aneurysm of the abdominal aorta bronzing of the skin has been described by Pepper and by Jürgens. (For aneurysms of the hepatic artery, see Vol. IV. Part I. p. 153, and of the renal arteries Vol. IV. Part I. p. 650.)

ARTERIO-VENOUS ANEURYSM. — **Definition.** — A communication between an artery and a vein, with or without an intervening sac; the former is called a *varicose aneurysm,* the latter an *aneurysmal varix.*

This is the oldest known variety of aneurysm ; there can be no doubt that Galen recognised this form as a sequel of careless venesection, and that he cured a case of it. He had felt the thrill—the "noise" which is experienced when the hand was placed upon the sac. William Hunter gave the first complete modern description in 1757. An admirable account is given in Broca's monograph, which also deals with the older literature. Of the varieties of arterio-venous aneurysm some involve the external arteries and are usually traumatic ; others, of the internal vessels, are usually spontaneous. The one follows the simultaneous wounding of an artery and a vein by a sharp instrument or by a bullet ; in the other, rupture takes place from an artery into a vein, or erosion may occur from a vein into an artery.

Traumatic Arterio-Venous Aneurysm.—Formerly the common cause was venesection when an unskilful operator opened the artery and the vein at the bend of the elbow, nowadays stab wounds and bullet wounds furnish the largest number of cases. The femoral, brachial, axillary, and popliteal vessels are most often affected. It is stated that with the modern bullet the accident is more common, and this seems to be borne out by the statistics of the South African war and of the Japanese and Russian war recently published by Stevenson and by Siago.

The opening between the artery and the vein may be direct, the two vessels being in contact, but the arterial pressure is so much higher that

the veins are gradually dilated and the condition is known as an aneurysmal varix. In other cases, more commonly in the traumatic variety, there is a sac communicating directly with both vessels, and forming an ovoid or globular pulsating tumour. The three distinguishing features of arterio-venous aneurysm are :—(i.) The distension of the veins above and below the lesion. When the deeper vessels of a part are involved they may not be visible externally, but as a rule in arterio-venous aneurysm of the arm or leg the varicose veins are the largest ever seen, standing out as huge convoluted tubes forming great saccular lacunae. In the axillary and subclavian regions there may be great swelling without any visible varicosity. The circumference of the affected limb may be greatly increased, and in some recorded instances the limb has been elongated, and an extra growth of hair is not uncommon. (ii.) On palpation a thrill is felt of maximum intensity at the site of the lesion, but propagated in the course of the vessels, in some instances for a great distance. It is a continuous vibration, but is increased during systole, and is much more intense than in any other condition ; in fact the thrill itself is distinctive of arterio-venous aneurysm. The patient may be much inconvenienced by it ; one man could never sleep on his left side on account of the noise transmitted from the tumour in the axilla. In a case under my care the thrill could be felt to the toes, and as far as the swelling above Poupart's ligament. Pulsation is felt, and where a sac intervenes an expansile tumour. Sometimes there is only a diffuse impulse without special localisation. Pulsation may be better seen than felt in the larger veins and sinuses. For example, a large tumour in the right iliac fossa had scarcely any palpable impulse, but the throbbing with each systole could be seen at a distance. (iii.) On auscultation a bruit is heard with special characters ; it is very loud, in fact the loudest vascular murmur known ; it is continuous, with systolic intensification, as if composed of two elements, a deep roaring continuous sound, which is venous and resembles the *bruit de diable* in the neck, and a more sibilant higher-pitched intermittent murmur, which is caused by the blood passing through the opening in the artery.

The *symptoms* of external arterio-venous aneurysm depend entirely upon the situation. In the carotids and in the subclavian vessels there may be little or no inconvenience for years. I have reported the case of a man who at fifteen fell with the result that a lead-pencil pierced both artery and vein high in the axilla. An arterio-venous aneurysm formed with swelling in the axillary and subclavian regions. It caused him no inconvenience, and he became an athlete and rowed in races. I saw him ten years after the accident when the signs were well marked. He served in the South African war, and when last I heard of him, in 1901, twenty-three years after the accident, there was no change in the aneurysm. Rokitansky speaks of a man aged sixty-two who, thirty-three years before, received a bullet wound in the left shoulder, followed by an arterio-venous aneurysm. In the leg and in the forearm the progressive enlargement of the veins may

cause great disability, and in reality this is the special danger of the disease. After lasting for many years without change enlargement of the veins may take place rapidly. In the case mentioned by Rokitansky it was not until more than thirty years after the accident that the arm became blue and oedematous.

Internal Arterio-Venous Aneurysm.—Our knowledge of this form dates from the contribution of John Thurnam in 1840, whose paper almost exhausts the subject. The abnormal communication may be the result of a stab wound or of a bullet which penetrated the aorta and the vena cava, but it much more often follows the rupture of an aneurysm into the vena cava and the pulmonary artery. There are three situations in which it may occur :—

I. *The Aorta and Superior Vena Cava.*—In Thurnam's first case a man aged forty-two had a sudden pain in the chest with dyspnoea, and the upper part of his body became swollen and cyanotic. At the same time Thurnam discovered a loud "bruisement" like the vibration of a string to the right of the sternum. After death an aneurysm of the aorta was found to have perforated the superior vena cava. It is a comparatively rare lesion. I have only met with 2 cases. In 1890 Pepper and Griffith collected 28 cases from the literature, and a good many more have been reported since that date. The cases are usually in men, the subjects of aneurysm, often of the small unrecognised variety, but there have usually been pains in the chest, with cardiac distress. A most characteristic group of symptoms occurs with the perforation :—(1) There is a sudden onset with pain in the chest, dyspnoea, and signs of shock, small feeble pulse, and sweating. The shortness of breath increases, and becomes orthopnoea. (2) Within a few hours swelling begins of the face, neck, arms, and upper part of the body with cyanosis, which gradually deepens, so that the combination of swelling and lividity gives a terrible appearance to the patient. The veins of the neck, face, and arms become enlarged, and those of the upper half of the body, particularly the mammary, are greatly distended. At about the level of the diaphragm the swelling and cyanosis cease, so that there is an extraordinary contrast between the appearance of the upper and lower parts of the body. (3) Over the upper part of the sternum and in the right second and third interspaces may be felt a loud thrill with systolic intensification, and on auscultation a whirling murmur is heard of a continuous roaring quality, and intensified with each systole. (4) Evidence of aneurysm may be present. The cases are very characteristic, and with the exception of the sudden cyanosis of the upper part of the body which follows certain crushing accidents there is no other condition which could be mistaken for it.

II. *Aorta and Pulmonary Artery.*—This is a more frequent lesion, and has been studied particularly by Gairdner and Frederick Taylor, and recently Kappis, from Bäumler's clinic, has reviewed the literature of the subject. Usually the signs of aortic aneurysm have been present. The symptoms are practically those just spoken of in the previous section—a

sudden onset, cyanosis, orthopnoea, oedema of the upper part of the body (though this has not been so constant), and the characteristic physical signs of a communication between two vessels. When an aneurysm perforates one of the chambers of the heart the physical signs may be identical. An aneurysm growing into the lung may open branches of the pulmonary artery and a murmur of the same character may be heard.

III. *Abdominal Aorta and Inferior Vena Cava.*—This, the rarest form of arterio-venous aneurysm of the internal vessels, occasionally follows a bullet wound, but most of the cases have been in aneurysm. It occurred in one of J. H. Bryant's Guy's Hospital series. The symptoms are quite characteristic—sudden onset of swelling, with cyanosis of the legs and lower half of the body, the presence of a tumour in the abdomen, or the condition has come on after an accident, and the physical signs are those of a communication between an artery and a vein.

DIAGNOSIS OF ANEURYSM.—**General Remarks.**—In many instances the diagnosis is never made. The patient complains of ill-defined pains in the chest, and there is a fatal attack of angina, or death occurs from rupture. In a majority of all cases, when the sac reaches the thoracic wall, the diagnosis is easy. The small, deep-seated tumour growing upwards from the transverse arch or from the beginning of the ascending aorta may cause symptoms only, which are due to the pressure of the growth. Skoda, indeed, remarked that aneurysm of the thoracic aorta could not be recognised unless it reached the chest-wall, but with the *x*-rays even a small sac may now be seen, and there is rarely any difficulty in distinguishing between it and a solid tumour.

In doubtful cases there are generally circumstances which must be considered. A majority of all cases are in young or middle-aged males with a history of syphilis or hard work, or both combined. They are robust-looking and of a good colour, with what is spoken of in the hospital wards as the cardiovascular facies.

Special Conditions which simulate Aneurysm.—In two forms of valvular disease certain features suggest aneurysm. In *aortic insufficiency,* as pointed out originally by Corrigan, dynamic dilatation of the arch of the aorta and its branches may be so extreme that the diagnosis of aneurysm seems almost unavoidable. In the young, when the insufficiency has been rapidly produced, and when it is accompanied by high fever or anaemia, the throbbing to the right of the sternum may be so pronounced, or the pulsation above the right sterno-clavicular joint is so definite, that dilatation from organic disease is suspected. Unless the *x*-rays shew a well-defined saccular tumour, it is well to hesitate as to the diagnosis of aneurysm in the presence of aortic insufficiency. I have seen the pulsation three fingers' breadth to the right of the sternum, and there may be a definite tumour above the right sterno-clavicular articulation. Many cases of this sort have been recorded as aneurysm. A girl, aged seventeen years, after many attacks of rheumatic fever presented aortic insufficiency

and a very large, forcibly-beating heart. The diagnosis of aneurysm, made by two or three very skilful men, is not to be wondered at in the light of the following account given by H. A. Hare. "There was an egg-shaped protrusion in the suprasternal notch, very expansile and bulging with each systole of the heart, and the dilatation extended well up into the vessels." In this case, which I saw frequently, the tumour could be grasped very definitely, and was visible and palpable during the diastole. The necropsy which I performed shewed that the condition had been one of simple dynamic dilatation. The heart was enormously enlarged with an extreme degree of insufficiency of the aortic valves, and the arch of the aorta did not admit the index-finger, or the innominate artery the little finger. Many of the cases of so-called rheumatic aortitis and aneurysm in young persons are of this nature. The second condition which may simulate aneurysm is mitral insufficiency with enormous dilatation of the left auricle, stretching of the recurrent laryngeal nerve, and marked pulsation to the left of the sternum. The pulsation, often regarded as of the left auricle, is in reality due to the conus arteriosus. The paralysis of the left vocal cord is due to the stretching and atrophy of the left recurrent laryngeal nerve by the greatly dilated left auricle. The condition is sometimes very puzzling. Of this I have seen three instances, and a number of cases are on record. My first case, diagnosed by one of the most distinguished physicians in Europe as aneurysm, had mitral stenosis for years with paroxysmal dyspnoea, great cyanosis, paralysis of the left recurrent laryngeal nerve, and widespread pulsation in the third and fourth left interspaces. In a recent case the x-rays shewed very definitely that the widespread pulsation to the left of the sternum was not connected with the aorta.

Dynamic Dilatation.—The aorta is remarkably distensile, and there are conditions in which the walls lose their tonus, and during systole dilate and throb in a way to suggest aneurysm. This is seen in aortic insufficiency, of which I have already spoken, in neurasthenia and hysteria, in Graves' disease, and in anaemia. A very interesting case is reported by Dr. Byrom Bramwell: "So marked were the pulsation and dulness in the region of the heart as to lead Dr. W. Murray of Newcastle-on-Tyne to believe that an aneurysm of the ascending portion of the arch of the aorta was probably present." Within a few months these physical signs completely disappeared. It is more particularly in the abdominal aorta that this dynamic throbbing leads to error. The patients are neurotic, sometimes definitely hysterical, with the usual symptoms of nervous exhaustion and pains in different regions, often the subjects of mucous colitis, but the centre of all their unpleasant sensations is the throbbing in the abdomen, which may be severe enough to interfere with sleep or even with the taking of food. Morgagni recognised and gave a very good description of this condition. Allan Burns also refers to it, and quotes from Albers of Bremen a remarkable instance in which, associated with the throbbing, there was passage of dark blood in the stools. The association of small haemorrhages from the stomach and intestines has

been described by Dr. Phillips, but I have not met with any reported case more remarkable than that of Albers. The girl was excessively neurotic, had fainting fits, great palpitation in the abdomen, and an astonishing degree of violent pulsation. She passed blood from the bowels, and the diagnosis of aneurysm was made, but a Dr. Weinhalt, who was called in, said he doubted if the pulsations proceeded from aneurysm, as he had read of similar cases in Morgagni. A positive diagnosis in these cases is not always easy, and the throbbing may be so extreme that the diagnosis of an organic lesion of the vessel seems almost inevitable. This is the view taken by Potain, Teissier, and others who describe the condition as one of acute aortitis, of which, however, there is not any anatomical evidence. The points to be taken into consideration are : (1) that in moderate grades this throbbing is common in nervous women and in thin hypochondriacal men ; (2) the aorta is easily palpable and may be grasped with the fingers, sometimes feeling dilated and being very tender. With anaemia a thrill may be felt and a systolic murmur may be heard even without any pressure of the stethoscope ; (3) the subjective sensations may be most marked—abdominal distress amounting even to pain, nausea, and in rare instances the vomiting of small quantities of blood or the passage of blood in the stools. It is well to bear in mind that no pulsation however forcible, no thrill however intense, no bruit however loud, together or singly, justify the diagnosis of aneurysm of the abdominal aorta in the absence of a palpable expansile tumour. Another condition associated with dynamic dilatation of the aorta and great vessels is anaemia. In the abdominal aorta the throbbing may be extreme. Sometimes, too, in the thoracic aorta and its branches the pulsations become very forcible in traumatic anaemia, in aortic insufficiency with anaemia, particularly in infective endocarditis, and in cases of Addisonian anaemia. One instance may be quoted, as the pulsation was intense enough to jar the bed :—

A large stout man, aged forty-five, had for some months suffered from dyspepsia and pain in the abdomen. He had become very anaemic, and the day before he was seen he had an increase of the pain. When examined he was sweating, pale, and the large, fat abdomen throbbed in a most extraordinary manner. The shock of the impulse was communicated to the patient's body, was visible everywhere from head to foot, and standing against the foot of the bed one could feel distinctly the jarring impulse communicated to it. On palpation the throbbing was violent, but it was trifling in comparison with the extent of visible pulsation. There was a loud systolic murmur, but no thrill. That evening he passed a large quantity of blood from the bowels, and though no definite tumour could be felt, the diagnosis of aneurysm was made. The necropsy shewed a duodenal ulcer placed directly upon the pancreas, and a normal abdominal aorta.

In pernicious anaemia the throbbing in the vessels of the neck and in the subclavians may be so violent as to suggest aneurysm.

Diagnosis from other Tumours.—Skiagraphy has been of great assistance

in differentiating the pulsatile from the solid intrathoracic tumours. Occasionally subcutaneous tumours over the front of the chest have a communicated throbbing suggestive of aneurysm. Particularly is this the case with a cold tuberculous abscess associated with periostitis of the rib or the sternum. There may then be a definite jarring visible in the tumour, but there is absence of that expansile, forcible characteristic impulse, nor is the shock of the heart-sounds felt, and, as a rule, there is no murmur. There are two forms of pulsating empyema : (*a*) in one there is widespread throbbing of a large area of the chest, not unlike the jarring pulsation communicated by a large hypertrophied heart. It is not the strong heaving impulse of a cardiac or aneurysmal beating. Occasionally the pulsation may be very localised. I saw one instance in which the throbbing was entirely above the third rib on the left side. (*b*) In the other form, *empyema necessitatis*, the projecting tumour between the ribs presents a diffuse throb, not a strong heaving expansile pulsation. The *x*-rays here shew the condition very clearly. If necessary, a small needle may be inserted. It is not often now, I think, that an aneurysm, either in the abdomen or perforating the chest-wall, is opened in mistake for an abscess, as in the instances recorded by Ambroise Paré and Morgagni. One of the most deceptive conditions is the ruptured aneurysm of the abdominal aorta, which may form a very large tumour in the back, or the blood may pass down and reach one iliac fossa, and the tumour may be mistaken for abscess or appendicitis. As already mentioned, I know of four instances in which operation was performed in these circumstances, in one for a supposed abscess, in three cases for appendicitis. In a few instances expansile tumours of the bony wall of the chest are met with growing either from the sternum or from the ribs. As a rule, very little difficulty is experienced in determining the nature of the case.

Internal tumours are much more frequently a cause of difficulty in diagnosis. The small solid growths in the posterior mediastinum, connected either with the bronchial glands or with the oesophagus, may give a picture identical with that of the small aneurysm of the transverse arch, causing cough, severe orthopnoea, and paralysis of the left recurrent laryngeal nerve. Nowadays in good hands even a very small pulsating sac may be recognised by the *x*-rays. In any case, in an adult and a male, when there are no signs of external tumour or enlargement of the glands, such symptoms are much more likely to be caused by aneurysm. The small hard tumour of the oesophagus, just at the bifurcation of the trachea, may cause great difficulty, and even the *x*-rays may not be helpful, as is shewn in a case reported by A. L. Scott at the Pennsylvania Hospital. Quite as frequently the very large intrathoracic tumour growing from the mediastinum, the lung or the pleura, may cause symptoms very like those of aneurysm, as in the following case :—

A fairly well nourished woman of forty years, a patient of Dr. Bolgiano's, decubitus on the right side. Even before the night-dress was removed, pulsation

was seen on the left side of the chest.   Turned on her back, a visible impulse extended from the left sternal margin in the second, third, and fourth inter-spaces, lifting the chest-wall with each systole.   There was no bulging.   On palpation there was a diffuse shock, no punctuate impulse, no thrill ; the shock of the second sound was felt over the area of pulsation.   On percussion there was dulness over the manubrium and extending from the clavicle to the fourth interspace.   On auscultation the breathing was feeble and distant ; there was a systolic bruit to the left of the sternum.   The apex-beat was in the fifth inter-space, a little outside the nipple.   There was well-marked tracheal tugging ; the left recurrent laryngeal nerve was paralysed, and the voice was cracked.   Nothing was lacking in the diagnosis but the definite aneurysmal impulse over the area of shock or pulsation.   Had this been my first introduction to the case, I should have been in great doubt, but the conditions had been only too evident.   I had seen the patient months before with a small tumour of the left lobe of the thyroid and an enlarged gland above the clavicle.   In Europe she consulted Professor Kocher, who diagnosed cancer of the thyroid with mediastinal exten-sion.   On her return, Dr. Finney took out a gland in the neighbourhood, which proved to be cancerous.   The mediastinal growth extended to the pleura, and probably to the lung on the left side, the left recurrent became involved, brain symptoms came on, with double optic neuritis.   There was no question here of aneurysm, but the pulsation, the accentuated second sound, the systolic bruit, the complication of the recurrent laryngeal nerve, and the tracheal tugging gave a picture more suggestive of aneurysm than any I had ever before met with in malignant growth.

Very large thoracic aneurysms may simulate tumour, as in the famous case described on p. 661, in which Oppolzer diagnosed aneurysm and Skoda tumour (*vide* also Vol. V. p. 661).   In rare instances aneurysm and sarcoma may coexist ; in Virchow's case the two were in direct connexion.

Other Forms of Pulsatile Tumours.—Rapidly-growing sarcomas of bone and the very vascular tumours of the abdomen may present an expansile pulsation and lead to the diagnosis of aneurysm.   In the case of the bony tumours the situation is usually so definite, the character of the growth so distinctive, and the *x*-ray picture so well defined, that there is rarely any doubt.   More difficulty may arise in the case of a very vascular sarcoma in the abdomen, but the pulsation, though expansile, rarely gives to the hand that sensation of force and strength communicated directly from an aneurysm of the aorta or from one of the larger vessels.

Aneurysms which do not pulsate.—There are two conditions in which an aneurysm does not pulsate : (*a*) When a sac is obliterated with laminated fibrin.   Sometimes met with in aneurysm of the aorta, this is much more frequent in the popliteal and femoral vessels.   In the latter regions it is a serious matter, as the leg may be amputated under the belief that the tumour is a sarcoma.   Such an instance I saw in Montreal : a very large mass in the popliteal space which had neither pulsation nor bruit, after amputation of the leg, proved on dissection to be an obliterated aneurysmal sac.   A remarkable case is reported by Hulke, and the sequel is given by Morrant Baker in his paper " On Aneurysms which do not

pulsate." A huge tumour, which proved at the necropsy to be an aneurysm, occupied the left side of the neck from the trachea to the vertebrae, passed behind the clavicle, filling the axilla, and passed through the superior aperture of the thorax into the left pleural cavity, occupying its upper third and compressing the lung. It sprang from the left subclavian artery. The very large aneurysm referred to on p. 661 did not pulsate. (*b*) The second condition in which an aneurysm may not pulsate is when it ruptures into the neighbouring tissues, forming a diffuse tumour. This may occur in the neck, as in the case reported by Hulke and Baker, but it is much more common in the abdomen. As in two cases which I have reported, the tumour may be of enormous size and present slight or almost imperceptible pulsation. Sometimes no impulse whatever is to be felt. More particularly is this the case when the tumour extends rapidly in the flanks, forming a large solid mass. If the patient survives, as sometimes happens, for weeks or months, the clots become firmer and the pulsation may diminish or even disappear entirely. But even very shortly after the rupture the pulsation may be readily overlooked in the intensity of the other symptoms. The cases may present the features of the acute abdomen, and, as already mentioned, patients have been operated upon for this condition, usually with the diagnosis of appendicitis, without the slightest suspicion on the part of the surgeon that an aneurysm was present.

Skiagraphy.—With a good instrument in skilful hands, obscure and latent cases of thoracic and abdominal aneurysm are now readily recognised. More particularly is the procedure helpful in the aneurysm of symptoms, the small tumour compressing the mediastinal structures. The extent, the localisation, the shape, the relation of the sac to the other parts may all be determined, and even the solidification in it has been followed. Whether the aneurysm is saccular or fusiform and the direction of its growth may be ascertained. Even in cases in which the sac has begun to perforate the chest-wall useful information may be obtained. The accuracy with which a small tumour can be localised is surprising : a young woman was admitted to my ward in a state of cyanosis and orthopnoea, which was relieved by bleeding. The physical examination was negative, except that less air entered the lower lobe of the left lung. There was a history of possible syphilis, but there was really nothing to determine the nature of the tumour compressing her windpipe. The *x*-ray examination shewed a pulsating shadow in the situation of the descending portion of the arch, and after death it was in this position that the aneurysm was found. Occasionally there may be doubt between a small solid tumour or a carcinoma of the oesophagus and aneurysm. Several examinations should be made, but even practised observers may not be able to decide. These, however, are exceptional cases. From an examination of a large number of cases in my wards, F. H. Baetjer classifies the positions as follows : (1) "Aneurysm of the ascending portion of the aorta usually casts a shadow more to the right than to the left of the sternum, above the heart, and by localisation would be found

nearer the anterior than the posterior wall of the chest ; (2) Aneurysm of the transverse arch casts a shadow slightly to the left of the sternum, and the shadow extends upwards, and by localisation would be found nearer the anterior chest-wall ; (3) Aneurysm of the descending part of the arch casts a shadow to the left of the sternum, and by localisation would be found nearer to the posterior than the anterior wall of the chest." We have found the x-ray examination to be of special help in determining whether a case was suitable for surgical treatment ; a tumour which points to the right of the sternum may be a small localised sac growing from the ascending aorta, or it may be only part of a very large aneurysm quite unsuitable for wiring. The diffuse dilatation of the aorta may be distinguished from the dynamic dilatation, as in the latter between the pulsations the shadow disappears, as the aorta contracts and its shadow lies within that cast by the sternum and the spine. Aneurysms of the descending thoracic aorta are not so clearly defined by the x-rays except in these patients, and the same may be said of tumours of the abdominal aorta. In several cases, with the most extreme pulsation of the abdominal aorta, no dilatation could be determined. (*Vide* also art. Vol. I. p. 507.)

Prognosis.—In a majority of cases the outlook is hopeless. The following are the important factors :—*Age.*—Post-mortem experience teaches that in the aged many cases recover ; every museum contains specimens of healed aneurysms (removed from persons dead of other causes), the presence of which was unsuspected during life. A majority of such come from the aged who have lived quietly and in whom the aneurysm has been latent. I have seen in one aorta three completely obliterated sacs. At this time of life the conditions are favourable to repair in the atheromatous variety. An obliterated aneurysmal sac is not often seen in a man under forty years of age. *Form.*—The saccular aneurysm with a narrow mouth offers the best conditions for lamination. The very small and the very large appear to be most often obliterated. Some of the largest aneurysms on record have been the most chronic. No form of aneurysm heals more perfectly than the dissecting, so perfectly indeed that skilled observers have mistaken it for an anomaly of the aorta, and the patient may live for twenty or thirty years after an accident of the most destructive character (*vide* p. 641).

Early and thorough treatment should be carried out, particularly in the young syphilitic subjects, though it must be confessed that nature does more than art in the cure of aneurysm. For one obliterated sac the result of treatment, medicinal or surgical, there are five or six at least in which the condition has been found accidentally.

The prognosis in individual cases is very difficult to estimate. The average duration after discovery is from eighteen months to two years. The most rapidly fatal cases are those in which there have been neither symptoms nor physical signs—the small aneurysm of the arch which perforates rapidly ; almost as many patients die of complications and intercurrent affections as from rupture of the sac. Sudden death, not

common in hospital or private practice in patients under observation, is most frequently met with in medico-legal work. In a few cases the physical signs disappear, the symptoms are relieved and a practical cure is effected ; in others with a persistence of the physical signs, tumour, and pulsation, there is no progress, and the patient is able to follow his occupation for ten or more years ; in others again the chronic course is one of much suffering and discomfort.

The mode of termination in aneurysm is very variable. Sudden death may occur from haemorrhage, syncope, or angina pectoris. Rupture into the air-passages, the pleura, and the peritoneum is less rapidly fatal. Gradual heart-failure, asphyxia, oedema of the lungs, tuberculosis, and a progressive cachexia are responsible for more than a third of the deaths.

TREATMENT.—**Prophylaxis.**—The frequency of aneurysm may be diminished in two ways. The subjects of syphilis should be more thoroughly treated ; but it is difficult to induce men to remain under supervision after all trace of secondary symptoms have disappeared. If every luetic patient were looked upon as a prospective candidate for tabes, general paralysis, or aneurysm, we should be more insistent on this point. In the second place, young men who have had syphilis should be warned not to take too much exercise, and particularly not to make powerful muscular efforts which throw a sudden strain on the vascular system.

In a majority of all cases the treatment is symptomatic ; curative measures are successful in few cases.

Curative Methods.—These are directed towards the healing of the mes-aortitis and the promotion of coagulation in the sac. Iodide of potassium exerts a powerful influence on mes-aortitis, the primary lesion in a large majority of cases ; and although it cannot of course heal a rent of the intima, and probably has very little effect on the sac itself, it relieves pain, promotes cicatrisation of the aortitis, and favours coagulation in the sac by lowering the blood-pressure. Though used before by others, we owe to the late George Balfour of Edinburgh the strong advocacy of this valuable remedy. It is more particularly in young men with a well-marked syphilitic history that good effects are produced by this drug, of which 1 to 2 drams may be given in the 24 hours. When syphilitic infection is recent, or when other visceral lesions are suspected, mercurials may be given at the same time. To promote coagulation in the sac certain methods are followed which, by increasing the coagulability of the blood and slowing the circulation in the sac, favour the natural processes of cure. The oldest is that of Valsalva, reported by Morgagni : " The essence of this treatment was to detain the patient very strictly in bed for forty days, and during this period to subject him to repeated bleedings, while at the same time the diet and drink were carefully ordered, so that the daily allowance, administered in three or four meals, should never be such as to fill up the blood-vessels." " He made it a custom," says Morgagni, " to diminish the quantity of meat and drink

more and more every day, till it was brought down to half a pound of pudding in the morning, and in the evening half that quantity, and nothing else except water, and this also within a certain weight. After he had sufficiently reduced the patient by this method, so that, by reason of weakness, he could scarcely raise his hand from the bed in which he lay, the quantity of aliment was increased again by degrees till the necessary strength returned so as to allow of rising up." It is suggested that the pulsation in the aneurysm can be arrested entirely by this method ; but even should the arrest be not quite complete at first, the disease may still be in course of cure if the patient will continue to submit to a modified and strict, but not quite so severe, regimen for some time to come. The difficulty in carrying out this treatment effectively, as stated by Morgagni (no doubt on the basis of Valsalva's experience, as well as his own), is that " there will be many to whom this method of cure may seem much more insufferable than the disease itself ; especially as the only time when it can be of use (the beginnings of the aneurysm) is when the inconveniences actually felt are slight, so that patients are easily led to delay until continuous and grievous suffering, or even impending death itself, can no longer be avoided by any remedy whatever." The discussion that follows is very interesting, and may perhaps admit of the inference that failures of Valsalva's method were already well known in Morgagni's time, but were attributed (as failures usually are) to the remedy not having been adopted in time. But another inference follows even more clearly from this long discussion by the great pathologist of the eighteenth century, and from the cases submitted in illustration—namely, that with all his respect, amounting almost to veneration, for Valsalva, Morgagni seems to have used the method very tentatively, and with no inconsiderable misgivings (Gairdner). A modification of this was introduced by Bellingham and Tufnell, and is known by the name of the latter surgeon, the omission of blood-letting being the only point of difference from Valsalva's method. The indications are thus stated by Bellingham : " (1) To diminish the distending force of the blood from within (and above all, to diminish the frequency as much as possible of the heart's beats) ; (2) to favour the deposition of fibrin on the walls of the sac ; (3) to maintain this process until the sac is filled up, and thus obliterated ; (4) to bring about these results without deteriorating the quality of the blood, or diminishing too much the patient's strength." Tufnell's diet is : " For breakfast, 2 ounces of white bread and butter, with 2 ounces of cocoa or milk ; for dinner, 3 ounces of broiled or boiled meat, with 3 ounces of potatoes or bread, and 4 ounces of water or light claret ; for supper, 2 ounces of bread and butter, and 2 ounces of milk or tea—making in the aggregate 10 ounces of solid and 8 of fluid food in the twenty-four hours, *and no more*." It is not an easy method to carry out, and requires a good deal of fortitude on the part of the patient. In a young or middle-aged man, with a small aneurysm which the *x*-rays shew to be saccular, it is worth a trial. Tufnell gives some useful hints as to the arrange-

ments. " A light, cheerful, and airy room ; a special attendant always at hand to offer such aid as the patient may require, to read, converse with, or amuse him, are insisted on. The bed must be constructed and arranged so that the evacuations can be withdrawn without disturbance. Upon the bedstead must be placed two hair-mattresses, one upon the other, both full and elastic. Upon these (in proper site to receive the sacrum and hips), a large water-cushion properly but not over-filled ; upon this, a double blanket sewn at the corners and sides to the lower mattress, and upon the blanket a fine linen sheet similarly attached, to prevent all wrinkling in the bed and disturbance of the sheet ; another linen sheet (folded as after a lithotomy) laid transversely to receive the buttocks, and to be drawn from beneath them from time to time. On this bed, when once comfortably settled, the individual must be content to lie, without changing his position further than to turn from side to side, or occasionally round upon his face, should such movement give relief to the dorsal pain, as it sometimes will."

Rest is the important element in the treatment, and it is remarkable how quickly serious symptoms disappear after a week in bed. As Tufnell points out, the recumbent position reduces the heart-beats by about 43,000 in the twenty-four hours. In suitable cases, particularly in young men, these triple measures—rest, low diet, and iodide of potassium —should be given a thorough trial. In the cases which come under observation late with large sacs a symptomatic plan of treatment should be followed.

Surgical Measures.—For a full discussion surgical treatises must be consulted. Ligation of the aorta for aneurysm of the abdominal aorta has been performed in about a dozen cases, always with fatal results. *Distal ligation* of the carotid and subclavian (Brasdor's operation) has been done in many cases of innominate aneurysm, and often with success. *Compression* has been employed successfully in aneurysm of the abdominal aorta. It is only applicable when there is a space for compression above the sac. A case recorded by Dr. W. Murray, of Newcastle-on-Tyne, illustrates the possibility of cure when the aneurysm is in a favourable situation. " In this case the pressure was applied on two occasions at an interval of three days. On the first occasion of two hours' compression difficulties were experienced, and the results as regards the aneurysm were not important ; but it is recorded that the patient passed no urine for nearly thirty hours. On the second occasion five hours were occupied by the pressure, but it was during the last hour only that complete control of the pulsations in the tumour was obtained. This result was evidently of the nature of a surprise. ' To my astonishment,' Dr. Murray says, ' the tumour had now become perfectly pulseless, and every indication of pulsation in the aorta below it had disappeared.' It is certainly very remarkable that the consolidation of the tumour seems to have gone on quite steadily from this moment, and that even the very next day there was ' no pulsation in the tumour, which is now perfectly stationary, hard, resistent, and lessened in size.' The pulse in the

femoral arteries was also gone, but the excretion of urine was in this instance uninterrupted, or was quickly re-established. The details are very interesting, but cannot be further cited here. They shew that a complete cure was obtained ; certainly not without some severe symptoms, but probably with the minimum amount of danger or inconvenience consistent with the method. It is not, however, irrevelent to remark here that the patient (who had remained well and fit for work in the interval) contracted a second aneurysm near the coeliac axis, of which he died suddenly six years later. The necropsy revealed the complete consolidation of the first aneurysm, which was converted into a fibrous mass ; a great shrinking in the trunk of the inferior mesenteric artery ('dwindled to the size of the radial artery'), which arose out of this obliterated aneurysmal sac ; the enormous enlargement, on the other hand, of the superior mesenteric ('as large as the aorta'), and a corresponding increase of size in all the vessels entering into the very highly-developed collateral circulation which had been established through the epigastric, internal mammary, intercostal, and circumflex iliac arteries outside the abdomen, and the hepatic, colica media and sinistra, and haemorrhoidal arteries, etc." (Gairdner).

Introduction of Foreign Bodies.—To extend the surface on which the fibrin may coagulate, Charles H. Moore, of the Middlesex Hospital, in 1864, introduced fine iron-wire into the sac, and since this date various substances have been used—horsehair, catgut, Florence silk. It has not been a very successful method ; for details of procedure surgical manuals may be consulted. In a few cases cure has resulted. Various irritating liquids—iodine, perchloride of iron, etc.—have been injected into the sac without satisfactory results.

Electrolysis alone, or in combination with wiring of the sac, has given the best results. Hunner has collected 23 cases treated by the latter, the Moore-Conradi method, of which 4 cases were cured. Rosenstern's patient was alive seventeen years after the operation. This method has been extensively used at the Johns Hopkins Hospital ; in no case with complete cure, but in several instances life was prolonged. Full details of an improved and safe technique are given by Finney and Hunner.

Needling the Sac.—Sir W. MacEwen very rightly criticises the wiring and electrolytic methods as forming a red thrombus, whereas the natural obliteration of a sac is effected by white thrombi gradually deposited on the wall. To promote their formation he advises needling the sac with one or more pins passed through the cavity, so that the point or points just touch and no more the opposite wall, and with these the internal wall is scratched and irritated. Several successful cases are reported by this method.

To Promote the Coagulability of the Blood.—The calcium salts have been recommended, but they do not seem of much service in aneurysm. Iodide of potassium is believed to favour coagulation in the sac. The gelatin treatment introduced by Lancereaux has had many advocates. He advises the injection subcutaneously every five or six days of 200 c.c. of a

7 per 1000 saline solution containing 5 grams of gelatin.   From thirty to forty injections are given.   Cures have been reported, and in many cases the sac diminishes in size, and the pain lessens.   For several years this method was given a faithful trial at the Johns Hopkins Hospital, but without very favourable results (Futcher).   In a few cases relief of the symptoms followed, but no case was cured.   From contamination of the gelatin tetanus occurred in 2 cases (Rankin).

Symptomatic Treatment.—The early cough and dyspnoea are promptly relieved by rest.   In the robust, full-blooded man, with shortness of breath and signs of venous obstruction, free venesection is most helpful, and ·may be repeated.   In the severe paroxysms of dyspnoea blood should be freely removed, if necessary, from both arms.   Morphine may be given, unless there is oedema of the lungs ; but in any case, when a patient is suffering, and the orthopnoea is extreme, chloroform or morphine should be used.   The question of tracheotomy comes up in these cases.   The pressure is usually near the bifurcation of the trachea, and, theoretically, opening the windpipe would seem to be useless, and so it is in many cases, but temporary relief is sometimes given. The passage of an india-rubber tube may be tried.   Removal of a portion of the sternum may relieve the pressure, and may be per-formed when the x-rays shew the tumour to be central and compressing the windpipe between the spine and the breast bone.   It was done in one of my cases by Halsted, but too late.   For the pain the iodide of potassium is helpful in the syphilitic cases.   It is remarkable with what promptness the neuralgic pains are relieved by it ; indeed, it was in this way that the drug was introduced in the treatment of aneurysm.   Local applications—belladonna plasters, hot poultices, and the ice bag—give relief, but in a majority of cases, when the pain is fixed and severe, morphine must be given.   It is always distressing to see a man com-pletely under the control of this drug, but when the use becomes a necessity we should try to make a virtue of it, and by judicious manage-ment keep the dose at a minimum, and not worry the patient or his relatives with reflections on the disadvantages of the habit.   I have known several patients who have lived in comparative comfort, and apparently with some prolongation of life, as a result of a daily dose of from 3 to 6 grains.   Terrible cases, indeed, are those of aneurysm of the descending or abdominal aorta with nerve-root pressure, as they may require enormous doses of the drug.

<div align="right">W. OSLER.</div>

REFERENCES

History : 1. BENDA. Lubarsch and Ostertag's *Ergelnisse*, 1904.—2. BROCA. *Des anévrysmes*, Paris, 1856.—3. BROWNE, OSWALD. *Aneurysms of the Aorta*, 1885, H. K. Lewis.—4. CHIARI.   *Verhandl. d. deutsch. path. Gesellsch.*, 1904, Tagung vii., 180.—5. CRISP.   *Treatise on Structure, Diseases, and Injuries of Blood-vessels*, London, 1847, 171. — 6. CRUVEILHIER.   *Traité d'anatomie pathologique*, Paris, 1849-64. —7. EPPINGER.   *Pathogenesis, Histogenesis, und Aetiologie der Aneurysmen*, Berlin, 1887. — 8. GALEN.   *Kühn. Medici Graeci.* vii., Leipzig, 1824. — 9. FERNELIUS. *Pathologia*, L. S. c. 12.—10. FREIND, J.   *History of Physic*, 1725.—11. HELMSTEDTER.

Du mode de formation des anévrismes spontanes, Strassburg, 1873.—12. HODGSON. *Diseases of Arteries and Veins*, London, 1815.—13. HUNTER, W. *Medical Observations and Inquiries*, 1757, i. 323.—14. KÖSTER. *Berlin. klin. Wchnschr.*, 1875, xii. 323.— 15. LANCISI. *De Aneurismatibus*, Romae, 1728.—16. MORGAGNI. *De Sedibus et Causis Morborum*, Patav., 1765.—17. PARÉ, AMBROISE. Works, translated by Johnson, London, 1649.—18. ROKITANSKY. *Denkschriften der kaiserlichen Akademie der Wissenschaften*, 1850, iv. Wien.—19. SCARPA. *Sull' Aneurisma : Riflessioni ed Osservazioni Anatomico-chirurgiche*, Paris, 1804.—20. SIBSON. *Collected Works*, edited by W. M. Ord (New Syd. Soc.), 1881.—21. THOMA. *Virchows Arch.*, 1888, cxi. 76 ; 1888, cxii. 259, 383 ; 1888, cxiii. 244, 505.—22. VESALIUS. *Opera omnia*, 2 vols., Lugd. Bat., 1725.—22A. WELCH. *Med.-Chir. Trans.*, London, 1876, lix. 59. —**Etiology :** 23. ALLBUTT, Sir CLIFFORD. *St. George's Hosp. Rep.*, 1871, v. 49.— 24. DICKINSON, W. L. "Aneurysms associated with Hypoplasia of Arteries," *Trans. Path. Soc.*, London, 1894, xlv. 52.—25. FEYTAUD. *Des anévrysmes de l'aorte d'origine rhumatismale*, Paris, 1906.—26. FISCHER. *Deutsch. med. Wchnschr.*, 1905, xxxi. 1713.—27. GAIRDNER, Sir. W. T. "Aneurysm of the Aorta," this *System*, 1st edit., 1899, vi. 345.—28. *Idem*. *Clinical Medicine*, 1862.—29. JACCOUD, *Traité de pathologie interne*, 4th edit., Paris, 1875.—30. LAVERAN. *Sur l'anéurysme de l'aorte pectorale*, Paris, 1873.—31. LEBERT. *Ueber das Aneurysma der Bauch-Aorta und ihre Zweige*, Berlin, 1865.—32. LE BOUTILLIER. *Am. Journ. Med. Sc.*, Phila., 1903, cxxv. 778.—33. MYERS, A. B. R. *Diseases of the Heart among Soldiers*, London, 1870.— 34. THAYER, W. S. "On the Cardiac and Vascular Complications and Sequels of Typhoid Fever," *Johns Hopkins Hosp. Bull.*, 1904, xv. 323.—35. WELCH, F. H. *Med.-Chir. Trans.*, London, 1876, lix. 59.—**Pathology :** 36. BRYANT, J. H. *Clin. Journ.*, London, 1903-4, xxiii. 71, 89.—37. CRISP. *Treatise on Structure, Diseases, and Injuries of Blood-vessels*, London, 1847, 127-269.—38. LEBERT. *Traité d'anatomie pathologique*, Paris, 1857, tome i.—39. MYERS, A. B. R. *Diseases of the Heart among Soldiers*, London, 1870.—40. THOMA. *Virchows Arch.*, 1886, cv. 1, Plate I. fig. 5.— **Dilatation-Aneurysm :** 41. ADAMI. *Montreal Med. Journ.*, 1896, xxiv. 945.—42. BOSTRÖM. *Deutsch. Arch. f. klin. Med.*, 1888, xlii. 1.—43. DALAND. *Trans. Am. Climatological Assoc.*, 1897, xiv.—44. FERNELL. *St. Louis Courier of Medicine*, 1887. —45. GRAHAM, J. E. *Am. Journ. Med. Sc.*, Phila., 1886, xci. 155.—46. LINN. *Medical Records and Researches*, London, 1798.—47. MACALLUM, W. G. "Dissecting Aneurysm," *Johns Hopkins Hosp. Bull.*, 1909, xx. 9.—48. M'CRAE, J. *Journ. Path. and Bacteriol.*, Edinburgh and London, 1905, x. 373.—49. MARTIUS, M. *Rupture de l'aorte*, Paris, 1905.—50. NICHOLLS. *Phil. Trans.*, London, 1729, xxxv. 440. —51. PEACOCK. *Trans. Path. Soc.*, London, 1866, xvii. 50 ; xxv. 59.—52. PENNOCK. *Am. Journ. Med. Sc.*, Phila., 1838-39, xxiii. 13.—53. SCHEDE. "Aetiologie, Verlauf, und Heilung des Aneurysma dissecans der Aorta," *Virchows Arch.*, 1908, cxcii. 52.—54. SHEKELTON. *Dublin Hosp. Reps. and Communications*, 1822, iii. 231.—**Saccular Aneurysm :** 55. DRUMMOND. *Brit. Med. Journ.*, 1893, ii. 299.—56. BOINET. *Bull. Soc. anat.*, Paris, 1906.—57. BROADBENT. *Heart Disease*, 4th edit., London, 1906.—58. GAIRDNER. This *System*, 1st edit., 1899, vi. 386.—59. GEE. *St. Barts. Hosp. Rep.*, London, 1894, xxx. 1.—60. HEAD. *Brain*, 1893, xvi. 56.—61. JANOWSKI. *Deutsch. Arch. f. klin. Med.*, Leipz., xlvi.—62. JOHNSON, Sir G. *Med.-Chir. Trans.*, London, 1875, lviii. 29.—63. MACDONNELL. *Amer. Journ. Med. Sc.*, Phila., 1888, xcv. 251, and 1890, xcix. 598.—64. MACKENZIE, J. *Brain*, 1893, xvi. 321.—65. OLIVER. *Lancet*, London, 1878, ii. 406.—66. OGLE, J. W. *Med.-Chir. Trans.*, London, 1858, xli. 397.—67. SHAW, H. BATTY. *Internat. Clinics*, 1901, 11 ser., I.—68. SOKOLOWSKI. *Deutsch. Arch. f. klin. Med.*, 1877, xix. 623.—69. WALSHE, W. H. *Diseases of the Heart*, 4th edit., London, 1873.—70. WILLIAMS, F. H. "Small Aneurysm of Descending Aorta, x-ray Examination," *Trans. Assoc. Am. Physicians*, Phila., 1899, xiv. 168.—**Aneurysm of the Abdominal Aorta :** 71. BRYANT, J. H. *Clin. Journ.*, London, 1903-4, xxiii. 71, 89.—72. CRISP. *Treatise on Structure, Diseases, and Injuries of the Blood-vessels*, London, 1847, 171 and 235.—73. NUNNELEY. *Aneurysm of the Abdominal Aorta*, Baillière, Tindall, and Cox, 1906.—74. OSLER. *Lancet*, London, 1905, ii. 1089.—75. STOKES. *Diseases of the Heart and Aorta*, London, 1854.—**Arterio-Venous Aneurysm :** 76. BROCA. *Des anévrysmes*, Paris, 1856.—77. HUNTER, WILLIAM. *Medical Observations and Inquiries*, 1757, i. 323. —78. KAPPIS, I. *Deutsch. Arch. f. klin. Med.*, 1907, xc. 506.—79. PEPPER and GRIFFITH. *Trans. Assoc. Am. Physicians*, 1890, v. 45.—80. SIAGO. *Deutsch. Ztschr. f. Chir.*, 1906, lxxxv. 577.—81. STEVENSON, W. F. *Surgical Cases ;*

Government Report, 1905.—82. THURNAM, J. *Med.-Chir. Trans.*, London, 1840, xxiii. 323.—**Diagnosis** : 83. BAKER, W. M. *St. Bart. Hosp. Rep.*, 1879, xv. 75.—84. BRAMWELL, BYROM. *Diseases of the Heart and Thoracic Aorta*, 1884, 723.— 85. BURNS, ALLAN. *Observations on Diseases of the Heart*, 1809.—86. HARE. *Med. Rec.*, New York, 1886, xxix. 558.—87. HULKE. *Trans. Clin. Soc.*, London, 1878, xi. 123. —**Treatment** : 88. BELLINGHAM. "On the Curative Treatment of Aneurysm of the Aorta," *Dublin Med. Press*, 1852, xxvii. 81.—89. FINNEY and HUNNER. *Johns Hopkins Hosp. Bull.*, 1900, xi. 263.—90. LANCEREAUX. *Bull. Acad. de méd.*, Paris, 1897, xxxvii. 784.—91. MACEWEN. *Lancet*, 1890, ii. 1086.—92. MOORE, C. H. *Med.-Chir. Trans.*, London, 1864, xlvii. 129.—93. MURRAY, W. *Ibid.*, 1864, xlvii. 187. — 94. RANKIN. *Ibid.*, 1903, lxxxvi. 377. — 95. TUFNELL. *The Successful Treatment of Internal Aneurysm by Consolidation of the Contents of the Sac*, 2nd edit., London, 1875.

W. O.

PHLEBITIS

By H. H. CLUTTON, M.C.

OPINIONS as to the affinities between phlebitis and thrombosis have varied from the earliest time at which pathology could have for us any meaning up to the present day. Hunter and his followers, with Cruveilhier at their head, laid the greatest stress on the inflammation of the vein ; the latter asserting that whatever the form of the inflammation it is always accompanied by the formation of a clot—thrombosis—but that this coagulation of the blood is a secondary phenomenon. Thus phlebitis at that time held the primary position. In 1856 Virchow completely turned the tables, and was able to convince the vast majority of pathologists that thrombosis was the primary and essential condition. Since then bacteriology has shewn that microbes play a most important part in causing phlebitis, and that thrombosis in such cases is a secondary result (*vide* p. 705).

With our present knowledge we may confidently assert that in all suppurating wounds, where organisms abound, the progress of infection is from without inwards ; that the inflammation around the infected spot involves the outer coat of the vein, and from this inwards till endophlebitis is fully established ; and that this endophlebitis is the cause of the thrombosis in this particular variety of the disease. Arguing from this as an established and easily demonstrated fact, we have been gradually induced to think that many of the cases of so-called primary thrombosis really depend upon an endophlebitis which may either have extended from the outer coat inwards, as in the infective phlebitis above mentioned, or be itself primary and due to a specific cause, such as acute articular rheumatism or gout. We cannot, however, assume that this is always the case ; and Virchow's work on the coagulation of the blood as the cause of phlebitis may explain some of those cases of thrombosis which cannot at present be attributed to phlebitis (*vide* p. 710).

Etiology and Pathology.—The circumstances in which phlebitis may arise, and the different forms it may assume, must now be considered.

Traumatic phlebitis, simple or infective, may arise in any wound, or after any operation, in which a vein has been exposed. The simple form will pass unnoticed and be unaccompanied by thrombosis, if a large vein be not exposed or wounded and ligatured. The inflammation is part of the reparative process which ordinarily takes place in the healing of a wound, and does not spread. If a vein has been ligatured endophlebitis ensues, but it is strictly local for the obliteration of the vein at that spot. On the proximal side of the ligature this obliteration takes place, as far as the next collateral branch, without the formation of a thrombus, as in an amputation stump; but on the distal side, as the vein is full of blood, a clot is formed which, after it has undergone softening and fatty degeneration, is again absorbed. The thrombus on this side of the ligature may indeed extend farther than the nearest collateral vein, but it has no evil consequences in its extension towards the periphery.

The infective phlebitis from a wound, on the other hand, is one of the most serious diseases with which we have to deal; and, according to our present knowledge, admits of many clinical varieties from the different degrees of virulence which the infective organisms present, and the different susceptibility of the individuals infected. As the pyogenetic organisms, to which this infection is due, vary in their mode of growth, and in their virulence, there is a considerable variety in the clinical manifestations of an infected wound. We do not as yet know to which particular pyogenetic organism, or even to what group of such organisms, we are to attribute a particular train of symptoms. When a wound does not heal by first intention, we believe that it has been infected by such organisms of one kind or another; whether by means of instruments, fingers, or ligatures, or, in the case of an accident, by the original injury. It is very difficult to fix upon a particular predominating organism among the many which may be detected in the laboratory. We recognise sometimes a very rapid and acute form of suppuration in which, from the intensity of the inflammatory processes which the organisms have produced, the tissues rapidly break down, and even sloughs come away. In other cases there may be merely a little pus, or even none at all, and a good deal of swelling. The difference in the progress of these cases, the one towards almost certain death, and the other possibly towards ultimate recovery, may be due to the varying degrees of virulence in the same organism, and of suitability of the soil for their cultivation. In either case a vein may be involved; the external coat is invaded by the same organisms which are the cause of the inflammation in the connective tissue, and the extension of this to the internal coat, and the consequent endophlebitis, give rise to thrombosis. For a time the coagulation of the blood in the vein may arrest the general dissemination of the infective process; but, in the more virulent forms of the disease the thrombus is itself invaded by

the bacterial infection, and softens down under its influence. Infective embolism may now take place with its characteristic symptoms of pyaemia, or this may be again prevented by further thrombosis.

In many instances the progress of further infection of the vein is permanently arrested by the formation of a healthy clot; yet too often, if the larger veins be the seat of the trouble, the auto-infection goes on till the whole length of a large vein, or series of veins—like the femoral and iliac veins—are filled with purulent fluid, with or without the consequent phenomena of embolism and pyaemia. Death is produced either by pyaemia, septicaemia, or the extension of the mischief to the vena cava; when, even if some arrest of the disease do take place, it is too late to hope for recovery.

A small vein, if it be shedding infective emboli into the circulation, is as mischievous as a larger one; for each embolus carries with it the organisms of suppuration, and starts again—most often in the lungs— the disease originated at the primary seat of infection. In this way all the characteristic and fatal symptoms of pyaemia (see Vol. I. p. 880) may be produced from what was at first a most insignificant wound or operation. Again, a suppurating wound may be surrounded by a barrier of inflammatory tissue which has arrested the progress of the infecting organism. This barrier of granulation tissue may have in its midst a vein, the walls of which are thus inflamed, and the lumen consequently filled with clot. But if this barrier and vein be not themselves invaded with organisms, we have after all to deal with a simple phlebitis, not with an infective or septic one; although the original cause was perhaps as septic as it well could be.

Other instances of suppurative phlebitis and thrombosis, which are not dependent upon injury or operation but yet arise from a definite form of suppuration, should here also be mentioned.

The lateral sinus and jugular vein may be infected in this way from acute or chronic suppuration of the middle ear. The intervening bone is not necessarily diseased, though this is far from rare. The emissary veins may become involved in the area of infection, exactly as we have described above, and, by the extension of endophlebitis, may eventually produce the same result—endophlebitis and suppurative thrombosis—in the lateral sinus. Before this sinus is blocked by thrombosis, septic emboli may be thrown into the circulation producing the ordinary signs of pyaemia.

Pylephlebitis is another instance of the same kind. In this disease the portal vein becomes the seat of suppurative phlebitis and thrombosis from some suppurative and infective disease in its tributary branches; such, for example, as those coming from a suppurating appendix vermiformis. For this and for other visceral lesions of a similar kind, the reader must be referred to the appropriate section in which such conditions are more fully described (" Pylephlebitis," Vol. IV. Part I. p. 145). They are quoted here as instances to shew the various modes in which suppurative phlebitis and thrombosis may arise.

We have now to consider those cases of phlebitis and thrombosis which are unattended by any suppuration, and which are not due to any injury or operation.

Thrombophlebitis is occasionally seen in *chlorosis*. It occurs both in the cerebral sinuses and in the veins of the extremities, and in the latter is often bilateral. Pulmonary embolism may cause a fatal issue to an otherwise simple femoral thrombosis. This is important, inasmuch as a chlorotic patient may be able to walk about, and should of course be kept in bed when the thrombosis is recognised. (See art. " Chlorosis," Vol. V. p. 713 ; and "Thrombosis," p. 736.)

Phlebitis as a complication of acute or subacute *articular rheumatism* has been more frequently recognised by continental observers than in this country. It has, however, also been referred to by Dr. Poynton, who says that he has seen 7 cases. Rheumatic phlebitis most frequently attacks the veins of the lower extremities, and occurs before, during, or after an attack of acute or subacute articular rheumatism. It is, however, much more common during the convalescence, or after the acute attack has passed off. Sometimes when the phlebitis comes on, the general symptoms of rheumatic fever reappear. As a rule, pain in the part precedes the general swelling and the cord which is finally to be felt in the situation of the vein involved. It seems reasonable, therefore, to suppose that many of these cases are due to endophlebitis, with which both the clinical symptoms and the pathology of the disease would appear to agree (*vide* also p. 731).

Gouty Phlebitis.—To Sir James Paget we are indebted for the first clear account of this condition. "It affects the superficial rather than the deep veins, and often occurs in patches, affecting (for example) on one day a short piece of a saphenous vein, and on the next day another separate piece of the same, or a corresponding piece of the opposite vein or of a femoral vein. It shews herein an evident disposition towards being metastatic and symmetrical ; characters which, I may remark by the way, are strongly in favour of the belief that the essential and primary disease is not a coagulation of blood, but an inflammation of portions of the venous walls." It occurs chiefly in the lower limbs of those who have a marked gouty constitution, or with gouty inheritance ; it may arise during acute gout, in an interval between attacks of gout, in persons of more latent gouty habit, and, indeed, in persons of gouty inheritance in whom no gouty symptoms had previously appeared. Although more frequent in middle and later life, it is far from unknown in younger persons. In this form of phlebitis there is also a constant tendency to recurrence at the same spot—for example, in three or four inches of the internal saphena—till a permanent, thick, hard cord is to be felt which is never completely absorbed, but remains as a constant irritant for future attacks of inflammation. Even in the quiescent stage this chronic phlebitis prevents the patient from taking his usual exercise on account of the pain that is thereby induced ;

and in the acute stage he is forced to take to his bed till the attack is over.

Influenza may, before convalescence has set in, give rise to phlebitis and thrombosis of one or other of the larger veins of the extremities. (*Vide* art. "Influenza," Vol. I. p. 946, and "Thrombosis," p. 729.)

After enteric fever, during the convalescent stage, obstruction of the veins in one of the lower extremities is not uncommon, and more often in the left femoral than in the right. The patient is often extremely weak, and the strength of his circulation is reduced to the lowest ebb. But, besides slowing of the blood-stream, there seems some accumulating evidence to shew that the typhoid bacillus may be the cause of endophlebitis, as it is for the periosteal affections which occur and continue so long after the attack of fever has passed, and the patient has apparently recovered.

At the end of an attack of *appendicitis*, and perhaps more frequently after removal of the appendix, when the patient appears to be perfectly well and with a normal temperature, a simple phlebitis or thrombosis may arise in the left femoral vein and its tributary branches. It clears up quickly and produces very little oedema. It is probable, therefore, that the vein is not completely filled with clot. Occasionally, however, a number of superficial veins on the same side are involved one after the other. The patient is then delayed in his convalescence for a month or two (*vide also* p. 731).

Syphilitic Phlebitis.—Dieulafoy has collected 36 cases ascribed to syphilis, and epitomises its features. It is an early manifestation, and may appear a few weeks after the primary sore. It is often symmetrical and is specially prone to relapse. It is not prone to give rise to embolism.

The *pressure of a tumour* is one of the causes ordinarily assigned for thrombosis. And yet how rare it is for a vein to be thus occluded by clot, unless the tumour be one of those that infiltrates the surrounding tissue as it increases in size,—namely, a malignant growth! Oedema from the pressure of a simple tumour is of course another matter, for this does not prove thrombosis, but only obstruction to the flow of blood. Malignant infiltration of the walls of a vein gives rise to endophlebitis ; the thrombosis is, in that case, secondary, and is itself invaded by the growth in its further extension.

During the convalescence of any serious illness, or in the late stages of a malignant disease causing *cachexia*, the obstruction of a vein is not uncommon. Although not always evident before death, the necropsy in a malignant case may prove that a secondary growth was the cause, in the manner described above. In the other instances in which no obvious cause can be discovered, slackening of the circulation, accumulation of platelets and of white corpuscles along the sides of the vessel, and an alteration of the chemical composition of the blood may be the cause of the thrombosis, which is here supposed to precede the phlebitis. This is often called marantic thrombosis. (See art. "Thrombosis," p. 707 *et seq.*)

The phlebitis and thrombosis which follow upon a varicose condition of the veins fall within the sphere of surgery.

Symptoms.—1. *Of Simple Phlebitis.*—The local symptoms of a non-suppurative phlebitis are ushered in by pain, which gradually increases in severity for some hours or days and then slowly subsides, unless there be further extension of the inflammation or a fresh attack else-where.   On examination of the part at first no swelling is discovered ; but in the course of the day, or of a few hours more, some diffused swelling is felt : if the vein affected be a deep one, like the femoral, some days elapse, as a rule, before a distinct cord-like swelling is found.   If it be one of the superficial veins, like the saphenous, it is more clearly defined, or more quickly recognised.   Oedema of the parts beyond the seat of obstruction is well marked, especially in the foot when the common femoral is the vein concerned ; but it is entirely absent if the superficial veins alone are inflamed and obstructed.   The oedema is generally of the soft variety, easily pitting on pressure, and extends from the foot to the knee.   In cases in which the thrombosis has extended to the highest point in the main vein of a limb, such as the external and common iliac, and has further induced coagulation in its chief collateral branches, the oedema is of a peculiarly hard and brawny consistence, like that which is seen in the "phlegmasia alba dolens" after parturition (see p. 749).   The length of time that the oedema lasts is very variable. In the soft oedema, in which the obstruction is not so extensive or com-plete, a few weeks or months will be a sufficient time to reckon for the complete restoration of the circulation.   But in many cases of "solid oedema" the circulation is never completely restored ; the limb remains larger for the rest of life, and requires support to keep the swelling in check.   Redness of the skin over the parts affected is a very noticeable feature when the superficial veins are inflamed and thrombosed ; but in the case of the veins beneath the deep fascia it does not appear.   When this redness is seen over a superficial vein it is not in a fine line leading to an inflamed gland, as in lymphangitis, but a wide and diffused band.

Suppuration does not occur, even in thrombosis of the superficial veins, unless there be an abrasion of the skin or an ulcer through which organisms can have entered ; occasionally, however, fluctuation may be present when fluid blood lies between two patches of thrombosis in a vein beneath the skin.

The general symptoms of simple phlebitis are those of a mild fever. The temperature rises at the onset, and reaches its highest point, generally about 102° F., on the second day, remains stationary for a few days, and then sinks to normal.   It is not, however, uncommon, especially after typhoid fever, for the onset to be marked by a rigor, and a temperature of 103° F., or higher ; but the rigor is not repeated, and in a few days the temperature falls.   If the temperature persist, either a fresh extension of the phlebitis is taking place, or, especially if at the same time it rises higher, suppuration is to be suspected after all.

Complications.—One of the common features of simple phlebitis is

the possibility of a relapse, when all the symptoms begin again. It may be an extension of inflammation from the part first attacked, or a fresh attack at another part of the body. This is especially frequent in gouty phlebitis, as I have already explained.

Embolism is the most serious complication to which a patient suffering from simple phlebitis is exposed. These only too well-known accidents should always be recollected by every practitioner; they are fully described in a subsequent article. Cases have occurred in which a most sudden and distressing dyspnoea has taken place, and death seemed imminent; but the patient has rallied nevertheless, and a patch of crepitation has been discovered at the base of one lung. An interesting discussion has been raised as to whether clots formed from different causes may shew variations in their tendency to become detached and form emboli. There is no doubt that in the thrombosis of varicose veins embolism is not uncommon, but the victims of this trouble are often walking about. If they were from the onset of the thrombosis put to bed it might not occur. It has also been observed that embolism is a more likely occurrence after thrombosis in influenza ·and rare in enteric fever. But patients are more often able to walk about with influenza than during enteric fever. It seems, therefore, as if movement after the occurrence of thrombosis is the chief cause of embolism, whatever be the origin of the thrombosis. Another complication or sequel should also be borne in mind—namely, the persistence of oedema for months, years, or indeed for the rest of life, in some cases in which the deep femoral vein had been thrombosed.

2. *Of Suppurative Phlebitis.*—The local symptoms are those of suppuration. In most cases pus is already escaping from some wound or abscess cavity, and the known proximity of some large venous trunk causes great anxiety, if more acute general symptoms suddenly appear, or the ordinary febrile attack persists beyond the time during which such an abscess or wound would cause such symptoms. Sometimes, however, there are scarcely any local symptoms, or they are so trivial as to escape observation till the acute general symptoms lead to a more careful examination. The special symptoms are those of high fever, with sharp rises and falls. Shivering, or a distinct rigor, may accompany the sudden rise of temperature; and the fall may be associated with profuse sweating. There may have been a high temperature for a week or more before the fever assumes this particular character; or the hectic may be the first indication of what is taking place. If the ordinary symptoms of phlebitis have already been present for some days, there can be no doubt as to the cause of the symptoms being infection from the thrombus; but in other instances a large inflammatory swelling may have existed without any distinct evidence of thrombosis or phlebitis. A re-examination may shew that, after all, pent-up pus may be the more likely cause of this fever; yet if, after the evacuation of the pus, the same symptoms recur, the suspicion that the vein is involved in the suppuration will be confirmed.

Superficial veins do not give rise to the difficulty described above; for at one or other extremity of the inflammatory swelling the hard cord-like outline of the thrombosed vein can usually be felt. A deep vein, on the other hand, when infected by pyogenetic organisms, is, as a rule, surrounded by so much inflammatory exudation that it may be impossible to find the characteristic local signs of a thrombosed vessel. It is in such places as the groin, the axilla, or the neck that this difficulty arises; but as has been already stated, the diagnosis must be made by the combination of local signs of deep suppuration with systemic infection. In the case of the ear, with suppurative thrombosis of the lateral sinus, there is often a deep swelling in the neck beneath the mastoid process, which is due in part no doubt to inflamed glands from chronic discharge from the ear; but it is also in many cases caused in great measure by the inflammatory swelling round the thrombosed jugular vein, and is one of the most marked indications, if it be not already too late, for surgical interference. The further progress of infective phlebitis and thrombosis is shewn by the persistent character of the fluctuating temperature; if this cease, and the temperature, although high, no longer oscillates in a rapid and irregular manner, we may hope that, although suppuration is still going on in the tissues around, the vein is occluded on the proximal side by a healthy thrombus. Unfortunately this is not often the case; the temperature rises as before in the same sudden and uncertain manner and as quickly falls; variations from 105° to 99° F. in the course of an hour or two being not uncommon. When these variations cover as much as six degrees, septic embolism has probably begun already. Although with nearly every sudden rise of temperature which reaches as high as 104° F. or 105° F. a rigor may be expected, it does not by any means always occur; the sweating with the fall, however, is never absent. The stage at which these violent oscillations of temperature begin is very variable; they are probably coincident with the disintegration of the infected thrombus; it is probable, therefore, that till this occurs there will be no certain sign of suppurating phlebitis, but only the symptoms ordinarily present in an infected wound.

When the sudden changes of temperature have been repeated again and again in the course of a few days, or even, as in some cases, in the course of twenty-four hours, there is little or no prospect of recovery, unless indeed some surgical means can be adopted to shut off the pathway by which the septic emboli are entering the general circulation.

The further history of the case is that of pyaemia, which has been described in the first volume of this work.

Treatment.—1. *Simple Phlebitis.*—Rest in bed, with the limb slightly raised, is the really important treatment for simple phlebitis in its early stage. This does all that is possible to prevent the detachment of any part of the clot. Although embolism is a rare event, the mere possibility of such accidents to which allusion has already been made should make us insist upon absolute rest till all the tenderness on

pressure, and other signs of inflammation, have disappeared. Extract of belladonna and glycerin in equal parts may be applied locally for the relief of pain; but all friction, as in the case of ointments, should be avoided. The constitutional condition should be treated on general principles; if the pain be distressing, small doses of morphine may be given subcutaneously. But aspirin in ten or fifteen grain doses should always be tried first, so as to avoid, if possible, the use of morphine. When the patient is fit to get up and to move about, he may require an elastic bandage or stocking to prevent oedema of the foot or leg, if the obstruction has been in the femoral vein. If much muscular wasting has ensued from the extension of the thrombosis into the collateral branches, massage may be ordered; but great care must be taken to forbid this until all dangers of embolism have long passed away.

2. *Gouty Phlebitis and Thrombosis.*—When the attack of pain and tenderness constantly recurs in the same spot, or a hard cord-like swelling remains and is painful, the part affected should be removed. This is especially advisable when the pain and tenderness seem to prevent any active exercise. The operation should be commenced on the proximal side of the affected part, so as to cut off the connexion with the central part of the circulation. The thickened thrombosed vein can then be quickly excised without any risk of causing embolism. The result of this treatment is very gratifying, as it not only enables the patient to resume his occupation more quickly than he would otherwise do, but it also prevents the constant recurrence of similar attacks in the part previously affected. Moreover, in some of these cases the patients have remained free for many years from gouty phlebitis in any part of the body.

3. *Suppurative Phlebitis.*—As infective phlebitis in wounds and after surgical operations is directly due to suppuration from the introduction of organisms from without, it is clear that no one should undertake the treatment of wounds who is not thoroughly familiar with the various ways in which the organisms of suppuration may be introduced. "Prevention is better than cure."

But when suppuration has taken place, or an abscess has formed (apart perhaps from all surgical interference), everything must be done to stop the further progress of infection. The abscess must be opened, a sinus slit up, or a counter-opening made, according to the surgical necessities of the case. By such means suppuration may be prevented from extending to the neighbourhood of a vein. A deeply-seated and acute abscess is much to be feared as the forerunner of infective phlebitis, and must be treated early if preventive surgery is to take its proper place in our clinical work.

The treatment of suppurative phlebitis has entered upon a new era since it has been shewn that lateral sinus pyaemia from ear-disease may be arrested by the ligature of the internal jugular vein. The vein is completely divided between two ligatures, the proximal end being ligatured as low down in the neck as possible, and the distal end turned

out on the surface of the wound. The sinus is then exposed by trephining the mastoid, and the pus and decomposing clot are scraped out with a sharp spoon. If the distal end of the divided jugular vein has been turned out upon the neck, it can be used as an outlet for washing through from the opening in the lateral sinus. I have recorded a case in which pyaemic infection of the ankle-joint had occurred before this operation was undertaken, and yet after the above proceeding had been carried out the boy recovered. This special subject is fully dealt with in the article on "The Intracranial and Intravenous Infections complicating Ear Disease" (Vol. IV. Part II. p. 494); but it is necessary to state here what has been successfully accomplished for a disease that is otherwise certainly fatal, to illustrate the same principles in dealing with other parts of the body. It is not likely, however, that in the extremities there will be many opportunities of applying this principle of cutting off the connexion of a distant suppurating vein from the central organ of circulation; and, as surgical methods of treatment steadily improve through all ranks of the profession, suppurative phlebitis in the extremities must diminish. The middle ear, on the other hand, will continue to supply such cases so long as the public refuse to treat their ears on modern antiseptic lines. Still, it should be impressed on all teachers of medicine and surgery, that this method of cutting short an attack of pyaemia, due to suppurative phlebitis, is as applicable to the extremities as it has been found to be serviceable in lateral sinus pyaemia. We must remember that reamputation was done many years ago with success for infective osteomyelitis of a stump, when pyaemia had apparently already begun; it is possible, therefore, that at no distant date the division and double ligature of large venous trunks will be a recognised treatment of pyaemia in other parts of the body.

H. H. CLUTTON.

REFERENCES

1. CLUTTON. *Brit. Med. Journ.*, 1892, i. 807.—2. DIEULAFOY. "Phlébite syphilitique," *Clinique Médicale de l'Hôtel-Dieu*, Paris, 1905-6, v. 230.—3. PAGET, Sir J. *Clinical Lectures and Essays*, 1875, p. 293. Edited by Howard Marsh.—4. POYNTON. *System of Medicine* (Osler and M'Crae), 1907, ii. 692.

H. H. C.

THROMBOSIS[1]

By Prof. WILLIAM H. WELCH, M.D.

Definition.—A thrombus was formerly defined as a blood-coagulum, formed in the heart or vessels during life. This definition applies to many cases; but, in order to include agglutinative thrombi as well as coagulative thrombi, and to give due prominence to the participation of blood-platelets and corpuscles, a thrombus may be more broadly defined as a solid mass or plug formed in the living heart or vessels from constituents of the blood. Thrombosis is the act or process of formation of a thrombus, or the condition characterised by its presence.

Coagulation of the Blood.—The outlines of the views now held as to the coagulation of the blood may conveniently be sketched here in the briefest possible manner. The substances necessary for coagulation of the blood are fibrinogen, fibrin-ferment (thrombin), and calcium salts. The fibrinogen and calcium salts are normally present in the blood. Thrombin is not present as such in the blood, otherwise coagulation would occur. It is supposed that it first appears in an inactive form, analogous to other pro-ferments such as trypsinogen, under the designation prothrombin or thrombogen. Much discussion has taken place as to the means by which prothrombin is converted into thrombin. On the analogy of Pawlow's hypothesis of the production of trypsin from trypsinogen by the action of enterokinase, Morawitz and Fuld independently put forward a similar explanation to account for the activation of prothrombin, namely that a zymoplastic substance (thrombokinase, coagulin) in the presence of calcium salts transforms prothrombin into thrombin, and as a result the fibrinogen is coagulated. As to the origin of prothrombin and thrombokinase there is some uncertainty; prothrombin is provided either by the blood-plates or from the leucocytes, and is either secreted by them so that it exists in normal plasma, or results from their destruction. Thrombokinase is derived from the blood-plates, the leucocytes, the vessel-wall, or from the tissues generally, the testis being a convenient source from which to obtain it for experimental purposes (Mellanby). The problem of thrombokinase is further complicated by Loeb's description of coagulins in the vessel-walls and in the tissues, extracts of which, as has long been known, produce coagulation of fibrinogen.

Structure of Thrombi.—The formed elements which may enter into the composition of fresh thrombi are blood-platelets, fibrin, leucocytes, and red corpuscles. These elements may be present in varying number,

---

[1] As Prof. Welch was at the last moment unable to revise his standard article, this has been undertaken while the volume was going through the press by one of the Editors (H. D. R.).

proportion, and arrangement, whence there results great diversity in the appearance and structure of different thrombi.

The two main anatomical groups of thrombi are the red and the white thrombi. Many of the mixed thrombi may be regarded as a variety of the white thrombus. In addition there are thrombi of relatively minor importance composed wholly or chiefly of leucocytes, of fibrillated fibrin, or of hyaline material.

Red Thrombi.—These are formed from stagnating blood, and in the recent state do not differ in appearance and structure from clots formed in shed blood. They are made up of fibrillated fibrin and of red and white corpuscles in the same proportions as in the circulating blood, or the white corpuscles may be somewhat in excess. If any part of such a red thrombus be exposed to circulating blood, white material, consisting of platelets with fibrin and leucocytes, is deposited upon it. This deposit may aid in distinguishing the thrombus from a post-mortem clot.

White and Mixed Thrombi.—Most thrombi are formed from the circulating blood, and are white, or of a mixed red and white colour. The white or grey colour is due to the presence of platelets, fibrin, and leucocytes, occurring singly, or, more frequently, in combination. The admixture with red corpuscles is not an essential character of the thrombus, although it may be sufficient to give it a predominantly red colour.

Fresh white human thrombi, when examined microscopically, are seen to be composed of a granular material, usually in islands or strands of varying shape and size, around and between which are fibrin and leucocytes with a larger or smaller number of entangled red corpuscles. The granular matter, to which the older observers attached comparatively little importance, and which they interpreted as granular or molecular fibrin or the detritus of white corpuscles, is now known to be an essential constituent of the white thrombus, and is composed chiefly of altered blood-platelets. Intact polynuclear leucocytes are usually numerous in the margins of and between the masses of platelets, and may be scattered among the individual platelets. Not less important is the fibrillated fibrin, which is generally present in large amount. It is particularly dense in the borders of the platelet-masses, and stretches between them in anastomosing strands, or as a finer network containing red and white corpuscles. Within the accumulations of platelets in fresh thrombi fibrin is often absent, or is in small amount. These various constituents of the thrombus often present a definite architectural arrangement, and soon undergo metamorphoses which will be described subsequently.

Thrombi of the kind just described, and as we find them at necropsies on human beings, are completed products, and it is difficult, indeed generally impossible, from their examination to come to any conclusion as to the exact manner of their formation; particularly as regards the sequence and relative importance of their different constituents. So long as the knowledge of the structure of thrombi was limited to that derived

from the study of these completed plugs, the coagulation of fibrin was generally believed to be the primary and essential step in their formation ; although Virchow pointed out the greater richness in white corpuscles as a feature distinguishing them from post-mortem clots.

Zahn, in 1872, was the first to make a systematic experimental study, mainly in frogs, of the mode of formation of thrombi. He came to the conclusion that the process is initiated by the accumulation of white corpuscles which, by their disintegration, give rise to granular detritus. This is quickly·followed by the appearance of fibrin, which was readily accounted for by Weigert on the basis of Alexander Schmidt's well-known suggestion of the origin of fibrin-ferment from disintegrated leucocytes. Zahn's views, anticipated in part by Mantegazza in 1869, and confirmed by Pitres in 1876, gained prompt and wide acceptance.

Continued experimental study of the subject, however, especially upon mammals, led to opposition to Zahn's conclusions, and favoured the opinion, now generally accepted, that the ordinary white thrombus starts as an accumulation not of leucocytes but of blood-platelets. The investigators chiefly concerned in the establishment of this doctrine are Osler (1881-82), Hayem (1882), Bizzozero (1882), Lubnitzky (1885), and Eberth and Schimmelbusch (1885-86).

There is no difficulty in producing thrombi experimentally by injury, either mechanical or chemical, to the vessel-wall ; or by the introduction of foreign bodies into the circulation. If the early formation of such a thrombus be observed under the microscope in the living mesenteric vessels of a dog, as was done by Eberth and Schimmelbusch, it is seen that the first step consists in the accumulation of blood-platelets at the seat of injury. These plates, in consequence of their viscous metamorphosis, at once become adherent to each other and to the wall of the vessel, and thus form plugs which may be subsequently washed away into the circulation, but which sometimes so increase in size as to obstruct the lumen of the vessel completely. Red and white corpuscles may be included in the mass of platelets ; but their presence at this stage is purely accidental ; they are not to be regarded as essential constituents of the thrombus in its inception.

The microscopical examination of young experimental thrombi confirms the results of these direct observations, and affords information as to their further development. To obtain a clear idea of this development, thrombi should be examined at intervals of minutes from their beginning to those half an hour old or older. I reported the results of such an experimental study in 1887. The material composing the youngest thrombi formed from the circulating blood appears macroscopically as a soft, homogeneous, grey translucent substance of viscid consistence. Microscopically it is made up chiefly of platelets, which are seen as pale, round, or somewhat irregular bodies, varying in size but averaging about one-quarter the diameter of a red corpuscle.

Leucocytes, which may be present in small number at the beginning,

rapidly increase in number, and within the first fifteen minutes to half an hour they are usually in such abundance that at this stage of its formation they must be considered an essential constituent of the thrombus. They tend to collect at the margins of the platelet-masses and between them. These leucocytes are nearly all polynuclear, and usually do not present any evidence of necrosis or disintegration.

With the accumulation of leucocytes, fibrillated fibrin, which at first was absent, makes its appearance; being, as pointed out by Hanau, especially well marked and dense in the margins of the masses of platelets. Within these masses it is usually absent. The rapidity with which leucocytes and fibrin are added to the masses of platelets varies much in different cases. At the end of half an hour the thrombus may be composed of platelets, leucocytes, and fibrin with entangled red corpuscles, in essentially the same proportions and with the same arrangement as in the human thrombi already described; or even after several hours it may still consist almost wholly of platelets.

The prevailing view is that platelets exist in normal blood, where they circulate with the red corpuscles in the axial current. In accordance with this view, many observers, following Eberth and Schimmelbusch, explain the beginning of a white thrombus by the accumulation of pre-existing platelets upon a foreign body, or, in consequence of slowing or other irregularities of the blood-flow, on the damaged inner wall of the heart or vessels. Contact with the abnormal surface sets up an immediate viscous metamorphosis of the platelets, whereby they adhere to each other and to the foreign body or vascular wall. Eberth and Schimmelbusch designate this process as conglutination, and distinguish it sharply from coagulation, which they regard as a later event in the development of the thrombus.

Those who hold with Löwit that platelets do not exist in normal blood, believe that they are produced at the moment of formation of the thrombus, as the result of injury to the blood; and many who believe that they are in normal blood not as independent elements, but as derivatives from leucocytes or red corpuscles, consider it probable that those in the thrombus are formed, at least in part, in consequence of such injury. Although there are observations which suggest that platelets may be derived from leucocytes, there is no evidence that the masses of platelets found in incipient thrombi come from leucocytes previously attracted to the spot.

Evidence was brought forward by Arnold, F. Müller, Determann, Maximow, and Schwalbe in favour of the origin of platelets from red corpuscles. On the other hand Rufus Cole found that an agglutinating serum for platelets was devoid of this power on red blood-corpuscles—an observation which militates against the view that platelets and red blood-corpuscles are genetically related. J. H. Wright's conclusion that blood-platelets are pinched off from the giant cells in the bone-marrow is fully accepted by Cabot.

The accumulation of leucocytes in the young thrombus may be

explained partly by mechanical causes,—the most evident being the projecting, irregular, sticky substance of the platelet masses associated with slowing and eddies of the blood-stream,—and partly by chemiotactic influences.

Whatever difficulties there may be, in accounting for the fibrin, relate to the general subject of coagulation of the blood rather than to the special conditions of the thrombus. As to the participation of platelets in the production of fibrin, opinion is divided; and upon this point the study of thrombi has not afforded conclusive evidence one way or the other. The usual absence of fibrin within the platelet masses for a considerable time after their formation may be thought to speak against the generation of fibrin-ferment by the platelets. But if, as is probable, the platelets contain nucleo-protein, it would be reasonable to suppose, in accordance with current physiological ideas, that they can yield one of the fibrin factors; and it may be that in these compact masses there is not enough fibrinogen furnished by the plasma to generate an appreciable amount of fibrin. The characteristic dense ring of fibrin immediately around the platelet masses, where there is abundant fibrinogen, could be interpreted in favour of the liberation of fibrin-ferment by the collected platelets. By the time, however, that the fibrin appears, leucocytes have also accumulated in the same situation; and they, either alone or together with the platelets, may be the source of the ferment; although, as already stated, the leucocytes in young thrombi generally shew apparently intact nuclei and cytoplasm.

Does the recognition of the described mode of development of a white thrombus necessitate a radical break, such as that made by Eberth and Schimmelbusch, with the old and still common conception that a thrombus is essentially a blood coagulum? This question applies only to the first stage of formation of a white thrombus, for the completed thrombus is undoubtedly a coagulum. It is, however, of both scientific and practical interest to inquire whether coagulative phenomena usher in the process of thrombosis or are merely secondary. A decisive answer to this question cannot be given until we are better informed than at present concerning the chemistry and morphology of coagulative processes, and the source and properties of the granular material constituting the youngest thrombi. The possibility that this material is already coagulated, and falls into the category of the coagulative necroses, has been suggested by Weigert, but without any proof of this view. There is greater probability that the accumulation and metamorphoses of the so-called platelets in beginning thrombi represent a preparation for coagulation or a first step in the process. As Hammarsten has pointed out, two chemical phases are to be distinguished in the process of coagulation; namely, the formation of fibrin-ferment from its zymogen, and the transformation of fibrinogen into fibrin under the influence of this ferment. Morphological phases may also be distinguished, and the platelet stage of thrombus formation may be interpreted as the first morphological phase of coagulation in circulating blood. According to

Wlassow a similar morphological phase may be recognised in the clotting produced by whipping shed blood.   It would lead too far afield to enter here into a discussion of the arguments in favour of this view ; but much in its support is found in recent chemical and morphological studies of extravascular and intravascular coagulation, and of the anatomical and chemical characters of blood platelets.[1]   It does not appear, therefore, that we are called upon at present to make any such radical revision of the traditional conception of white thrombi as coagula, as has been advocated of late years by some writers.

Leucocytic Thrombi. — As has already been explained, leucocytes, although they do not usher in the process of ordinary thrombosis, make their appearance at an early stage, and often accumulate in such numbers as to constitute a large part of the thrombus.   My studies of experimental and human thrombi have led me to assign to them a more important part in the construction of white thrombi than that indicated by Eberth and Schimmelbusch.   Whether the regular mural white thrombi ever arise as a collection of leucocytes, in the manner described by Zahn, is uncertain.   Such a mode of development, if it occurs, is, I think, exceptional.   Intravascular plugs, however, occur, which are made up wholly or predominantly of polynuclear leucocytes.   These are found mainly in small vessels in acutely inflamed regions, where they are to be regarded as inflammatory and probably chemiotactic in origin. Leucocytic masses may also be found after death in small vessels in leucocythaemia, and in diseases with marked leucocytosis ; but it is probable that these are not genuine obstructing plugs.

Purely Fibrinous Thrombi.—As will be described subsequently, fibrin usually increases in amount with the age of the thrombus.   The masses of platelets may be replaced by fibrin, and leucocytes may degenerate ; so that many old, unorganised thrombi consist of practically nothing but dense fibrin, in places hyaline.   I do not, however, desire now to call especial attention to these old, metamorphosed thrombi.

One sometimes finds in inflamed areas, less frequently under other circumstances, the vessels, particularly those of small size, partly or completely filled with fibrillated fibrin, presenting such an arrangement and configuration as ·to indicate coagulation during life.   Neither leucocytes nor platelets need take part in the formation of these plugs of pure fibrin, although sometimes they are present.   K. Zenker has well described the microscopical appearances in these cases.   Whorls or brush-like clumps of fibrin may spring at intervals from the wall of the vessel, where they are attached especially to necrotic endothelium or to points devoid of endothelium.   The fibrin may be disposed regularly, often in stellate figures, around definite centres in which, perhaps, a necrotic cell or fragment, endothelial or leucocytic, or a clump of platelets can be demonstrated.   The fibrin is often notably coarse. The affected vessels are not usually filled completely with fibrin, and they can be artificially injected.   In croupous pneumonia such fibrinous

[1] This work was critically reviewed by Löwit in Lubarsch and Ostertag's *Ergebnisse,* 1897.

masses are regularly present, both in capillaries and larger vessels of the hepatised area. These purely fibrinous coagula are of anatomical rather than clinical interest.

Agglutinative (Hyaline) Thrombi.—Hyaline thrombi have been known for a long time, being first described by von Recklinghausen, but are now of more importance; for since 1902 Flexner, Boxmeyer, Pearce and others have shewn that they constitute a perfectly distinct form of thrombus, due to agglutination of the red blood-corpuscles, and thus quite different from the thrombi due to coagulation which have been described at length. They are found especially in the capillaries, but may occur also in the smaller arteries and veins. The capillaries are filled with a refractive, homogeneous, translucent, colourless or faintly yellow material, which stains well with Weigert's fibrin-stain. Probably because the red blood-corpuscles have undergone change in the direction of haemolysis, they do not take stains in the ordinary manner. The same material may partly or completely fill the smaller arteries and veins. Balls, as well as cylindrical masses, of this hyaline substance may be found, especially in the cerebral vessels.

This hyaline thrombosis has been observed in a number of conditions, partly general, partly local, of an infective and toxic nature such as bronchopneumonia, pneumonia, the intestine in enteric fever (Flexner), eclampsia, haemorrhagic infarcts, the stomach in carbolic acid poisoning. They have also been produced experimentally in pigs infected with the hog-cholera bacillus (Welch), by injection of *Staphylococcus pyogenes aureus* (Ribbert), by injection of *Bacillus typhosus* (Fisher), *Bacillus diphtheriae* (Flexner), ricin (Flexner), by ergot (v. Recklinghausen), and by freezing (Kriege). Further it has been proved that the injection of bacterial haemagglutinins contained in various cytolytic immune serums give rise to the formation of these hyaline thrombi. It has now been proved (Pearce, Flexner) that these hyaline thrombi are due to the agglutination of red blood-corpuscles; and this view, which was originated so far back as 1873 by Hueter and was supported by Klebs and Welch, is now fully established. The hyaline thrombi are mainly found in the small vessels, especially of the viscera, and are important in that they produce focal necrosis, a subject which has been specially investigated by Pearce. According to Flexner the so-called fibrin-ferment thrombi are really agglutinative (hyaline) thrombi.

It should be noticed that not all hyaline thrombi are agglutinative, some are undoubtedly composed of soft masses of fibrin which have not become fibrillar (Wells).

Growth, Metamorphoses, and Organisation of Thrombi.—Thrombi in their growth assume various characters to which special epithets are applied. A thrombus formed from the circulating blood is at first parietal or mural, but by continued growth it may fill the vessel and become an occluding or obstructing thrombus. A primitive or autochthonous thrombus, caused by local conditions, may be the starting-point of a continued or propagated thrombus, extending in the course of the

thrombosed vessel and perhaps into communicating vessels. A secondary or encapsulating thrombus is one which starts from an embolus of thrombotic material. A continued thrombus is also often spoken of as secondary. Thrombi are, with rare exceptions, adherent, at least in places, to the wall of the vessel or the heart. Mural thrombi appear more or less flattened against the vessel-wall, or they may project in a globular or polypoid form into the lumen. Their free surface is generally rough. Loose thrombi in the heart are called ball-thrombi.

The thrombus grows in length chiefly in the direction of the current of blood ; but it may grow in the opposite direction. The intact and growing end of the thrombus is a flattened blunt cone usually not adherent to the wall of the vessel ; it is sometimes compared in shape to a serpent's head. A venous thrombus extends in the direction of the circulating blood, not only as far as the next branch, but frequently a greater or less distance beyond it, in the form of a mural thrombus. A thrombus is at first soft in consistence and moist ; but by contraction and extrusion of fluid it becomes more compact, firmer, drier, and more granular in texture.

Mural thrombi, especially small ones, such as fresh vegetations on the cardiac valves, may occur without any definite arrangement of the constituent elements. Such thrombi may consist almost wholly of platelets ; but it is most exceptional not to find at least some admixture with leucocytes and fibrin coagulated *intra vitam*.

The larger white and mixed thrombi often present a typical architecture. The stratified structure has long been known and emphasised. More recently Zahn has directed especial attention to the rib-like markings on the free surfaces,[1] and Aschoff to the internal architecture of white and mixed thrombi. Microscopical sections of these thrombi often shew an exquisitely trabecular structure due to irregularly contoured, anastomosing columns and lamellae, of varying size and shape, which spring at intervals from the wall of the vessel and extend, usually in an oblique or twisting direction, toward the free surface of the thrombus, upon which their extremities form the network of whitish lines or the transverse ribs noted by Zahn. If the thrombus be detached from the inner wall of the vessel, similar projecting lines and dots can be seen on its attached surface and often on the inner lining of the vessel. This trabecular framework of the thrombus is composed of masses of platelets with cortical layers of fibrin and leucocytes, as already described. The whole arrangement is aptly compared by Aschoff to branching coral-stems. The spaces between the trabeculae contain blood which during life may be fluid or may have coagulated ; or they may contain only fibrin and leucocytes, or an indefinite mixture of platelets, fibrin, and red and white corpuscles. Between the lamellae and columns, bands of fibrin, with or without platelets, often stretch

---

[1] A number of writers before Zahn observed the markings on the surfaces of thrombi. Bristowe in 1855 spoke of the "peculiar ribbed appearance" of the surface of cardiac thrombi (*Trans. Path. Soc. London*, vii. 141).

loosely and in a curved manner, the concavity of the curve looking toward the axis of the vessel. Aschoff explains the coral-like architecture and the ribbed surface of the thrombus partly by the oscillatory or wave-like motion of the flowing blood, which, as previously suggested by Zahn, may account for the ribs, and partly by slight irregularities of surface-level normally present in the inner lining of vessels. Zahn finds an analogy between the ribs of a thrombus and the ripple-marks in sand at the edge of the sea, or at the bottom of flowing streams. Before Zahn, Wickham Legg, in 1878, described the surface of a cardiac thrombus as "marked by lines resembling the impressions made by the waves on a sandy shore."

The usual explanation of the red and white stratification of mixed thrombi is that the thrombus is deposited in successive layers, of which the red are formed rapidly and the white more slowly. There are manifest difficulties in such an explanation. It is more probable that the red layers are cruor clots formed from blood brought to a standstill. Blood entering crevices, spaces, and clefts resulting from the irregular mode of growth of the thrombus, or from its contraction, or from the blood-stream, often with increase of pressure in consequence of the thrombotic barrier undermining and splitting the white substance, at first soft and later brittle, of the thrombus, may readily stagnate and clot. Indications of such a splitting of the thrombus by the circulating blood are often seen in horizontal white lamellae covering red layers and present within them : these lamellae are apparently split off from the general framework and bent in the direction of the blood-current. The typical architecture of the thrombus may not appear, or may be obscured or destroyed by displacement of its parts through the blood-stream, especially when this is forcible : hence it is often missed in arterial thrombi. White thrombi are, as a rule, microscopically mixed thrombi ; and in colour there is every transition from these to thrombi so red that careful examination is required for the detection of the white substance.

In long propagated venous thrombi smaller white thrombus-masses often alternate in a longitudinal direction with longer red ones. The explanation of this is that a primary white thrombus is formed, often starting from a valvular pocket. This becomes an occluding thrombus, and the column of blood reaching to the nearest branch, or to the confluence of two important veins, is brought to a standstill, and forms a red obstructing thrombus. At the extremity of this, where the blood enters from the branch, another white occluding thrombus may be formed, to be followed again by a red thrombus, and so on. Thrombi are sometimes described as red in consequence of failure to detect the small white autochthonous part of the thrombus. In fact the term mixed thrombus is applied to three different appearances of thrombi : (*a*) an intimate mixture of grey and red substances ; (*b*) stratification in successive grey and red layers ; and (*c*) red propagated clots consecutive to autochthonous white or mixed thrombi.

In old thrombi various metamorphoses have occurred which obscure or obliterate the typical structure and architecture of the younger ones. The masses of platelets, although they may persist a long time, become finely granular, sometimes almost or quite homogeneous in texture. They are invaded by fibrin, especially along the edges of spaces and clefts which appear. Notwithstanding these profound changes a certain configuration and a differentiation in staining properties often enable us to recognise the sites of the original columns and lamellae of plate-lets. The leucocytes, often at an early date, undergo fatty degenera-tion and necrosis, their nuclei disappearing both by caryolysis and caryorrhexis. The leucocytic detritus adds to the granular material of the thrombus. The red corpuscles are decolorised and fragmented. The haemoglobin is in part dissolved and, after organisation begins, is partly transformed into amorphous and crystalline haematoidin. These pigmentary transformations impart a brownish-red colour to red and mixed thrombi. Fibrin increases in amount and becomes coarse and dense. The part of the thrombus adjacent to the vessel-wall is often converted into compact concentric layers of fibrin at a period when masses of platelets are well preserved nearer the lumen. The hyaline material, which is very frequently found in layers and clumps in old thrombi, may be derived both from fibrin and from platelets; perhaps also from red corpuscles and leucocytes. It may stain well by Weigert's fibrin-stain, or only faintly, or not at all. Small spaces and canals, often containing nucleated cells, may be present in the homogeneous fibrin or hyaline substance (canalised fibrin of Langhans).

Of special importance are the liquefactive softenings which may occur in old thrombi. These are distinguished as simple or bland, septic or purulent, and putrid softenings.

The simple softenings occur in bland thrombi, being especially common in globular cardiac thrombi which, when old, regularly contain in their interior an opaque whitish or reddish thick fluid. This in old days was mistaken for pus, and hence the name puriform softening (purulent cysts). The liquid or pulpy material is the result of granular disintegration and liquefaction of the solid constituents of the thrombus, and consists of necrotic fatty leucocytes, albuminous and fatty granules, blood pigment and altered red corpuscles; the varying red tint of the fluid depending upon the number of red corpuscles originally present in the thrombus. Occasionally acicular crystals of fatty acid are present. This form of softening depends on fibrinolysis or solution of the fibrin by proteolytic enzymes derived from the leucocytes. The outer layers of the thrombus are protected by the antitryptic bodies in the blood-plasma, and so are not dissolved. It is not generally supposed that micro-organisms are in any way concerned in the process: bacteria, how-ever, have been found repeatedly in these thrombi; and it may be that they are not so absolutely unconcerned in simple thrombotic softening as is generally thought to be the case.

There is no question as to the participation of bacteria in the other

forms of softening. Septic or purulent softening, met with most frequently in infective thrombophlebitis, is a true suppuration; being the result of the accumulation of polynuclear leucocytes with autolytic liquefaction of the thrombus. The leucocytes are attracted in part from the blood of the thrombosed vessel and in part from the vasa vasorum and surrounding capillaries and veins. Pyogenetic bacteria, most frequently streptococci, are present in the thrombus and the walls of the vessel. Putrid softening is due to the invasion of putrefactive bacteria. Here the thrombus is of a dirty brown or green colour, and of foul odour.

These various softenings often lead to the separation of thrombotic fragments to be transported by the circulation as emboli,—bland, septic, or putrid according to the nature of the process.

White thrombi in veins, far less frequently in arteries, may undergo calcification, forming phleboliths or arterioliths. They are generally approximately spherical, and lie loosely or slightly adherent in the lumen. They are found most frequently in the veins around the prostate and bladder of men, in the plexus pampiniformes of women, and in the spleen.

One of the most interesting adaptive pathological processes is the so-called organisation of thrombi, which is the substitution for the thrombus of vascularised connective-tissue. The thrombus itself takes no active part in the process, but behaves as a foreign body. It is gradually disintegrated and absorbed, largely through the activities of phagocytes. The new tissue springs from the wall of the vessel or the heart, the tissue-forming cells being derived both from the endothelium and from other fixed cells in the wall. New vessels spring from the vasa vasorum. Lacunar spaces in the thrombus, or between the thrombus and the vascular wall, may become lined with endothelium, and also serve as channels for the circulating blood. These new vessels may establish communication with the lumen of the thrombosed vessel above or below the thrombus, or on both sides. The new tissue, which at first is rich in cells, becomes fibrous, and contracts. The result may be a solid fibrous plug, or a cavernous structure with large blood-spaces; or, by disappearance of the septa, a restoration of the lumen, with perhaps a few fibrous threads or bands stretching across it, as in the normal cerebral venous sinuses.

There are great diversities in individual cases as to the rapidity of onset and the course of the organising process; these differences depending upon various circumstances, the most important of which are the location of the thrombus, the condition of the wall of the vessel or heart, the general state of the patient, and the presence or absence of infection. In favourable cases the process may be well under way within a week. The wall of the vessel, or of the heart, may be so diseased as to be incapable of furnishing any new tissue; as is usually the case in aneurysmal sacs, and often in varices and in cardiac disease. The presence of pyogenetic bacteria prevents or delays the process of organisation. This process is a proliferative angiitis. It is this angiitis which leads to the closure of a vessel after ligation. If the ligature be applied

aseptically, and without injury to the internal coats, usually no thrombus is formed, or only a very small one. The formation of a thrombus is of no assistance in securing obliteration of a ligated vessel, in fact it impedes the development of the obliterating endarteritis.

The causes of organisation of thrombi are probably to be sought partly in the influence exerted by the thrombus as a foreign body, and partly in slowing or cessation of the blood-current and lowering of the tension of the vessel-wall (Thoma, Beneke). Whether, in addition, growth of cells may be determined by chemical substances derived from the thrombus is uncertain.

Etiology.—The recognition of the three classes of causes assigned for thrombosis, namely, alterations in the blood, mechanical disturbances of the circulation, and lesions of the vascular or cardiac wall, is not of recent date. The dyscrasic theory is the oldest. John Hunter introduced and Cruveilhier elaborated the conception of primary phlebitis with consecutive plugging of the vein ; and Baillie, Laennec, Davy and others emphasised stasis as a cause of intravascular clotting. Virchow's name, however, is the one especially associated with mechanical explanations of thrombosis. The experiments of Brücke, shewing the importance of integrity of the vascular wall in keeping the blood fluid, led to general recognition of the part taken by alterations of this wall in the etiology of thrombosis.

Whilst it is generally agreed that slowing and other irregularities of the circulation, contact of the blood with abnormal surfaces, and changes in the composition of the blood are concerned, singly or in combination, in the causation of thrombosis, there is much difference of opinion as to the relative importance of each of these factors, and as to the part of each as a proximate, as a remote, or as an accessory cause.

Slowing and other Irregularities of the Circulation.—Diminished velocity of the blood-current is not by itself an efficient cause of thrombosis. The circulation may be at a low ebb for a long time without the occurrence of thrombi. A stationary column of blood included in an artery or vein between two carefully applied aseptic ligatures within the living body may remain fluid for weeks (Glénard, Baumgarten). Slow circulation, however, in combination with lesions of the cardiac or vascular wall, or with the presence of micro-organisms or other changes in the blood, is an important predisposing cause of thrombosis, and frequently determines the localisation of the thrombus. This is evident from the relative infrequency of thrombi upon diseased patches of the inner coat of large arteries in contrast with their frequency upon similar patches in the small arteries and in the veins ; and in general from the predilection of thrombi for those parts of the circulatory channels in which the blood-flow is normal, or as the result of disease, slow. Extensive injury to the walls of arteries may be experimentally produced without resulting thrombosis.

Eberth and Schimmelbusch find that under normal conditions the platelets circulate with the red corpuscles in the axial blood-current, but

make their appearance in the outer still zone when the rapidity of the circulation is sufficiently diminished. Moderate slowing is attended by the accumulation of white corpuscles in this zone, whilst a further slackening of the stream is characterised by fewer leucocytes and more platelets in the peripheral layer. Mere slowing of the circulation, however, does not suffice to form thrombi; there must be some abnormality of the inner lining of the vessel-wall, with which the platelets are brought into contact, in order to induce the viscous metamorphosis of these bodies essential in the formation of plugs. Hence Eberth and Schimmelbusch conclude that it is only by the combination of slowing of the circulation with changes in the inner lining that the formation of white thrombi can be explained. It is conceivable that slowing of the circulation leads to liberation of zymoplastic substance (thrombokinase, coagulin) derived from the vessel-wall, or enables it to act at better advantage.

Von Recklinghausen attaches more importance to a whirling or eddying motion (*Wirbelbewegung*) than to mere slowness of the circulation. He has pointed out that eddies are produced when the blood enters normally or pathologically dilated channels from smaller ones, or passes into a cul-de-sac or over obstructions; and he has considered in an interesting way the special conditions causing this motion and its influence upon the production of thrombi. This irregularity of the blood-current will be referred to again in considering the localisation of venous thrombi (p. 717). Von Recklinghausen's observations make a valuable contribution to our knowledge of the mechanical disturbances of the circulation which favour the development of thrombi.

Thrombi attributed to slowing of the blood-current, often combined with eddying motion of the blood, are called stagnation-thrombi. Of these two groups are distinguished: (a) those due to local circulatory disturbances, as from interruption or narrowing of the lumen of vessels by ligation or compression, or from circumscribed dilatations, as aneurysms or varices; and (b) marantic thrombi resulting from weakened heart's action, with consequent feebleness of the general circulation. Virchow gave the name "marantic thrombi" to all or nearly all thrombi complicating or following anaemic and cachectic states, general infective diseases— as enteric fever, typhus fever, and the like,—and certain constitutional diseases. He considered a condition of marasmus, or great prostration, to be the common underlying factor. As we shall see subsequently, there are serious objections to this explanation of these thromboses, which indeed constitute the class of chief medical interest. The designation "marantic thromboses" for this group is still, however, in common use. Although it is proper in these groups of thrombi to emphasise the mechanical disturbances of the circulation as an important accessory factor, it is evident, from what has been said, that the class of stagnation-thrombi cannot be maintained in the strict sense originally advocated by Virchow. Other factors, especially lesions of the walls of the heart or vessels, enter decisively into their causation.

Contact of the Blood with Abnormal Surfaces. Lesions of the Cardiac and Vascular Walls.—It is universally recognised that the influence of the endothelial lining of the vascular channels in maintaining the fluid state of the blood is of the first importance. This influence appears to be partly physical and partly chemical. The smooth, non-adhesive character of the inner surface of the heart and vessels is the physical property which comes primarily into consideration. Whereas the introduction of such foreign bodies as threads, or bristles with rough surfaces, into the circulation is an efficient cause of thrombosis, perfectly smooth, indifferent bodies, as small glass balls, may be introduced without causing any coagulation (Zahn). Freund has shewn that blood collected with proper precautions in vessels lined with oil or vaseline, remains fluid for a long time. Mere contact with a foreign surface, therefore, does not suffice to induce clotting ; the result depends upon the character of this surface. Freund concludes that the essential thing is that the surface shall be such as to permit adhesion to occur between it and the corpuscles, particularly the red corpuscles ; the normal lining of the blood-vessels being characterised by the absence of this adhesive property. Without adopting Freund's conception of coagulation, which does not here concern us, we can apply, with much satisfaction in the explanation of many thrombi, his observations concerning the importance of adhesive surfaces in causing coagulation. There should also be taken into consideration the damage known to be inflicted by adhesive contact with abnormal surfaces upon platelets or red corpuscles, if these be regarded as the source of the granular material and platelets in thrombi.

Changes, therefore, which impair or destroy the smooth, non-adhesive surface of the normal inner lining of the vessels play an important part in the etiology of thrombosis ; and thrombi thus caused may be called adhesion-thrombi. The efficiency of these lesions in causing thrombi is increased if, by projection into the lumen, they obstruct the blood-flow ; or by their rough, irregular surface set up an eddying motion of the blood.

Although there is no proof that the normal vascular endothelium contains any anti-body (antithrombin) which preserves the fluidity of the circulating blood, there is evidence that lesions of the intima, through chemical as well as physical influences, incite thrombosis. Necrotic endothelial and intimal cells may furnish zymoplastic substance (thrombokinase), which converts prothrombin into thrombin (fibrin-ferment), and so leads to the transformation of the fibrinogen into fibrin.

Strong support for a belief in the participation of chemical substances in the causation of certain thrombi due to intimal lesions is to be found in contrasting the effects of trauma alone with those of trauma combined with infection of the intima. This has been especially brought out in the experimental studies of valvular lesions of the heart. Aseptic laceration of the cardiac valves generally leads to but slight production of thrombi upon the injured surfaces ; whereas the same traumatic lesions, com-

bined with the lodgment and growth of pyogenetic bacteria, are usually attended by the formation of considerable thrombotic vegetations. The differences in the result can hardly be explained by differences in the physical characters of the lesions in the two cases. It has been shewn that bacteria modify coagulation, and that different bacteria differ in their power of coagulating the blood ; thus *Staphylococcus pyogenes aureus* has a much more powerful action than *Bacillus coli*. As this effect is greatly diminished when the microbes are killed by heat, the effect is not identical with that of chemically inert particles, but probably depends on bacterial products (Loeb). We may draw the conclusion that lesions of the intima, apart from their more manifest characters, may possess certain specific properties especially favourable to the production of thrombi.

The most important of the structural changes of the vascular and cardiac walls which cause thrombosis are those due to inflammation, atheroma, calcification, necrosis, other degenerations, tumours, compression, and injury. Here again may be emphasised the importance of retardation and other irregularities of the circulation in rendering these various lesions effective causes of thrombosis. The aorta, for example, may be the seat of most extensive deforming endarteritis, with irregular projecting calcified plates and ragged atheromatous ulcers, without a trace of thrombotic deposit. The forcible pulsating current prevents the adhesion and accumulation of the formed elements constituting the beginning thrombus, or quickly washes them away. The presence in some instances of white mural thrombi in the aorta upon an intima apparently but slightly damaged indicates the importance of certain specific, although little understood, characters of intimal lesions in association with changes in the blood.

Foreign bodies, which have penetrated the blood-channels and set up thrombosis, have been observed repeatedly in human beings, especially in the heart and abdominal veins. Such accidents have followed swallowing fish-bones, needles, nails, bits of wire and the like. A blood-clot or thrombus in a vessel, or projecting into the lumen from a wound of the vessel, may itself be looked upon as a foreign body, and lead to further extension of the thrombus. There seems to be a certain self-propagating power in a thrombus. Similar effects are produced by the entrance of large parasites, such as distomata, by the invasion of tumour-masses, and by the penetration of parenchymatous cells into the circulatory channels (p. 795).

Infective Thrombi. Thrombophlebitis.—Phlebitis, as a cause of thrombosis, has reacquired within recent years so much importance that it is here singled out from other lesions of the vascular wall for special consideration.

In the first half of the last century, mainly through the influence of John Hunter and of Cruveilhier, thrombosis was by many regarded only as an expression of inflammation of the inner lining of the vessels. The material composing the thrombus was considered to be, at least in part,

an exudate of coagulable lymph from the inflamed vascular wall. Virchow, by his monumental work on thrombosis and embolism, dating from 1846, reversed this order of things, and made, for the great majority of cases, the thrombus the primary and essential phenomenon, and the inflammation of the wall, if present, a merely secondary effect. Phlebitis disappeared, as a chapter, from works on internal medicine, and thrombosis took its place. Within recent years, and again chiefly through the work of French investigators, the pendulum has swung back, and phlebitis has once more come to the front as a common and important cause of thrombosis, and resumed an important place in many systematic treatises on medicine. This rehabilitation of phlebitis is due mainly to bacteriological investigations of thrombosed vessels, especially of the so-called marantic thrombi of infective and cachectic diseases.

The distinction between bland thrombi and infective thrombi is an old and important one. The thrombi in septic and suppurative phlebitis, concerned especially in pyaemic processes and surgical affections, were for a long time the chief, indeed almost the only recognised representatives of the class of infective thrombi. There has been a gradual extension of the domain of infective thrombosis, until now many thrombi, previously classified as bland, are considered to be of infective origin. This is notably true of a large number of thrombi, formerly and still often called marantic, complicating many infective diseases, wasting and cachectic conditions, and anaemia. In 1887 Weigert stated that by means of his fibrin-stain he had found unsuspected micro-organisms in marantic thrombi with surprising frequency ; and since then there have been numerous similar observations, as well as not a few negative ones. In an examination of 44 cases, mostly of peripheral thrombi of the so-called marantic type, Harris and Longcope found bacteria in 34. In France the studies of Cornil and his pupils, especially Widal, and of Vaquez have had the greatest influence in developing the doctrine of the mycotic origin of this class of thrombi, and particularly that of primary phlebitis as the cause of these thromboses. It should not be forgotten that Paget, in 1866, contended for the primarily phlebitic nature of thrombosis in gout.

Phlegmasia alba dolens of the puerperium is the prototype of this class of thromboses. In the articles on various infective diseases, particularly enteric fever (see Vol. I. p. 1112) and influenza (Vol. I. p. 946), attention has been called to the occurrence of thrombosis as a complication or sequel. Similar thromboses occur in pneumonia, typhus, acute rheumatism, erysipelas, cholera, scarlatina, variola, tuberculosis, syphilis,—in fact with greater or less frequency in nearly all acute and chronic infections. Likewise in chlorosis, gout, leukaemia, senile debility, and chronic wasting and cachectic diseases, particularly cancer, thrombosis is a recognised complication. The more important associations of thrombosis with these various diseases will be considered more in detail subsequently (p. 727).

These various thromboses, occurring very rarely as primary affections,

usually secondary to infective or constitutional diseases, compose the great majority of those of medical, as distinguished from surgical, interest. Clinically and anatomically they undoubtedly have much in common. Is there any common etiological point of view from which they may be regarded ? Virchow thought so in calling them marantic thrombi, and attributing their causation to enfeebled circulation. The same causative factor still remains the underlying one with those who, like Cohnheim, interpolate nutritive changes in the endothelium between the slow circulation and the beginning of the thrombus.

Impaired circulation cannot serve as a common etiological shelter for this whole class of thromboses. There is no definite and constant relationship between the condition of the circulation and the occurrence of these thrombi. Whilst many appear during great debility, others of the same nature, and often in the same disease, occur when the heart's action is not notably weak. Thrombosis may ensue early in influenza. It is oftener a sequel than an accompaniment of enteric fever. On the other hand, the circulation may be extremely feeble for days without the appearance of thrombosis.

Many of these so-called marantic thrombi are unquestionably of infective origin. Vaquez, in his monograph on phlebitis of the extremities, published in 1894, has brought together the results of the observations of others, and especially those of his own and Widal's investigations, which demonstrate that bacteria are often present in these thrombi and in the adjacent vascular wall. Since the appearance of Vaquez' monograph there have been a number of confirmatory observations. Widal emphasises the importance of searching for bacteria in fresh thrombi, and in the autochthonous part of the thrombus and the adjacent wall of the vessel. The largest contingent of positive results has been furnished by the examination of puerperal thrombi,—many of which indeed are examples of septic thrombophlebitis, and of the marantic thrombi of chronic pulmonary tuberculosis ; but bacteria have also been found in thrombi complicating or following enteric fever, influenza, pneumonia, cancer, and other infective and cachectic conditions.

In relatively few instances has the specific micro-organism of the primary disease, as the typhoid or the tubercle bacillus, for example, been present in the thrombus ; more frequently secondary invaders, especially streptococci and other pyogenetic bacteria, have been detected : so that the thrombosis is considered to be oftener the result of some secondary infection than of the primary one. Colon bacilli have been found in typhoidal and other thrombi ; but as these bacteria are found so commonly in the blood and organs after death from all sorts of causes, no great importance can be attached to their mere demonstration without some further evidence of their pathogenetic activity. As might be expected, streptococci are the bacteria found most frequently in puerperal thromboses. Singer believes that gonorrhoeal infection is also a possible factor.

Not only in thrombi of infective diseases but also in cachectic

thromboses have bacteria, and here again most frequently pyogenetic forms, been demonstrated. Nor is this surprising when we consider the frequency of secondary infections in chronic diseases, especially as a terminal event (Flexner).

The supposition that in all of these cases the bacteria are accidentally or secondarily present, and in no way concerned in the causation of the thrombi, is extremely improbable. They are often in such number, in such arrangement and associated with such lesions, that they must have multiplied in the thrombus and in the vessel-wall.

The problem whether the bacteria have led to thrombosis by first invading the vascular wall and setting up inflammation is not solved by the mere demonstration of their presence. Certainly, in some instances, this sequence of events is plainly indicated by the microscopical appearances; but in many it is impossible to decide to what extent inflammatory changes in the wall antedated the thrombus, for the latter, especially when infected by bacteria, induces a secondary angiitis. Opportunities to study very recent infective marantic thrombi with reference to this point are not common.

In a case, which I examined, of multiple venous thrombosis complicating leucocythaemia, there was a primary mycotic endophlebitis with secondary thrombosis. There was a secondary streptococcic infection. In the intima of the thrombosed vessels were numerous scattered foci in which large numbers of streptococci were present. In these areas there was necrosis of endothelial and other intimal cells, with proliferation of surrounding cells and many polynuclear leucocytes. These foci formed little whitish elevations capped with platelets, fibrin, and leucocytes; the whole presenting an appearance similar to that of endocardial vegetations. There was marked nuclear fragmentation both in the infected intima and in the thrombus. Fresh mixed thrombi, containing fewer streptococci, were connected with these phlebitic vegetations. Although the vasa vasorum were hyperaemic, and were the seat of a moderate migration of leucocytes, streptococci were absent from the adventitia; and the appearances pointed decidedly to the direct penetration of the streptococci from the circulating blood into the intima. I have examined three other similar cases. A similar form of mycotic endophlebitis has been described by Vaquez (endophlébite végétante). Eichhorst has described a case of a multiple arterial thrombosis in which the appearances were in favour of localised arteritis due to infection brought by the vasa vasorum. In other cases the intima is more diffusely inflamed. After a short time there is no distinct line of demarcation between the thrombus and the intima, and all of the coats of the vessel are more or less inflamed.

Although the bacteria found in the intima may gain access from without through the vasa vasorum, or the lymphatics, it is probable that in the class of cases here under consideration they more frequently enter directly from the blood circulating in the main channel. There may be very extensive bacterial inflammation of the venous

wall, even with bulging of the intima into the lumen, without any thrombosis.

We do not possess sufficiently numerous and careful bacteriological examinations of the thrombi of infective and wasting diseases to enable us to say in what proportion of cases they contain micro-organisms. It is certain that in many instances such examinations have yielded negative results. It is quite possible that in some of these negative cases bacteria, originally present, have died out; but although by some authors much use is made of this explanation, it is not in general a satisfactory one. Many of the examinations were of thrombi sufficiently recent to exclude this possibility.

To explain these non-bacterial cases, the French writers assume the existence of a primary toxic endophlebitis, the toxins being either of bacterial origin or derived from other sources. Ponfick, many years ago, called attention to the occurrence of degenerations of the vascular endothelium in infective diseases; and there can be no doubt of the frequency of both degenerative and inflammatory changes of the intima in toxic and infective conditions.

A lesion which I have seen in the intima of veins (less frequently of arteries) in enteric fever, diphtheria, variola, and other infective diseases, is a nodular, sometimes a more diffuse, accumulation of lymphoid and endothelioid cells beneath the endothelium. These cells, as well as the covering endothelium, may undergo necrosis; indeed the appearances sometimes suggest primary necrosis with secondary accumulation of wandering cells and proliferation of fixed cells. These foci are not unlike the so-called lymphomatous nodules found in the liver in typhoid and other infections. They may unquestionably be the starting-point of thrombi, as has been shewn by Mallory in his study of the vascular lesions in enteric fever. Although this form of endophlebitis or endarteritis resembles that demonstrably caused by the actual presence of bacteria in the intima, bacteria are often absent, even in the fresh lesions; so that it is reasonable to suppose that the affection may be caused by toxins. I think that this toxic endangiitis is of importance in the causation of thrombosis complicating infective and cachectic states.

There are, however, instances of so-called marantic thrombosis in which no visible alteration of the intima can be made out at the site of the thrombus, or only the slight fatty degeneration of the endothelium which is such an extremely common condition that it does not afford a satisfactory explanation.

It is obvious that bacteria are likely to find especially favourable opportunities to gain lodgment, and toxic substances to do injury, in situations where the blood-current is slow and thrown into eddies; but the localisation in these situations of thromboses complicating infective and chronic diseases has perhaps been unduly emphasised. These thromboses may occur elsewhere, even in the aorta and larger arteries. Pre-existing diseases of the veins, especially chronic endophlebitis and

varicosities, are conditions predisposing to infective and cachectic thromboses.

Whilst we are justified in assigning a far more prominent place to the agency of micro-organisms and to primary phlebitis in the etiology of thrombosis than, until recent years, has been customary since Virchow's fundamental investigations, recent attempts to refer all thromboses, formerly called marantic, to the direct invasion of micro-organisms and to phlebitis go beyond demonstrated facts. We have not at present sufficient bacteriological and anatomical substratum for so wide a generalisation. The whole field, although difficult, is an inviting and fruitful one for further investigation. The clinical arguments in favour of the phlebitic origin of thrombosis will be considered below (p. 747).

What has been said regarding the relation of phlebitis to thrombosis complicating infective and constitutional diseases applies also to that of arteritis to the similar arterial thromboses which, although less common than the venous, are more frequent than was formerly supposed ; this will appear when we take up the association of thrombosis with particular diseases (p. 727).

It is of course understood that the preceding remarks on the relation of phlebitis and arteritis to thrombosis relate only to the medical thromboses, and not to the septic and suppurative thrombophlebitides of the surgeon, of the bacterial origin of which there is no question ; although these latter may be concerned in diseases, such as suppurative pylephlebitis, which are in the province of the physician.

Chemical Changes in the Blood. Ferment-Thrombi.—The old ideas of chemical changes in the blood as causes of intravascular clotting, embodied in such terms as acre coagulatorium, hyperinosis, inopexia, are now of historical interest only. There appears to be no definite and constant relation between the amount of fibrin obtainable from the blood, or the rapidity of its coagulation in the test-tube, and the occurrence of thrombosis in human beings. Peripheral thrombosis is a less common complication of pneumonia and acute articular rheumatism, which are characterised by high fibrin-content of the blood, than of enteric fever and certain cachectic states in which the fibrin-content is approximately normal or reduced.

In dogs whose blood was rendered incoagulable by injection of "peptone" (albumose) Schimmelbusch produced platelet-thrombi experimentally. On the other hand, Sahli and Eguet observed no collection of platelets or formation of thrombi around hogs' bristles or silk threads inserted into the jugular veins of rabbits having incoagulable blood from injection of leech extract ; although control experiments regularly gave positive results. These latter experiments shew that chemical changes in the blood may influence the process of thrombosis.

The main support of the belief entertained by some that the liberation of fibrin-ferment in the general blood-stream is an important cause of human thrombosis, is based on the results of experiments which

demonstrate that the injection of various substances into the circulation may cause intravascular clotting. The most important of the substances which have been observed to produce this effect are laked blood (Naunyn), biliary salts (Ranke), ether (Naunyn, Hanau), fresh defibrinated blood (Köhler), emulsions or extracts from cells, especially lymphoid cells (Groth, Wooldridge), transfusion of blood (Landois, Ponfick), and snake-venom (art. Vol. II. Part. II. p. 800). The coagulating effect of laked blood is attributable to the stromata of red corpuscles rather than to dissolved haemoglobin (Wooldridge). The coagulating principle here, as well as of the various tissue-extracts, is believed to be a nucleo-protein which, by combination with calcium, forms the fibrin-ferment. It is to the presence of this ferment or the subsequent liberation of the ferment that the dangerous intravascular clots following the injection of defibrinated blood or the transfusion of foreign blood are due. The coagulative effect of snake-venom under certain conditions is referred by Halliburton to proteoses free from phosphorus, and therefore not nucleo-proteins. The action of snake-venom upon coagulation is probably analogous to that of various toxic albumoses, bacterial and vegetable. They are in general to be ranked among anti-coagulating substances; but the result varies with the dose, the manner of injection, and other circumstances. Wooldridge has shewn that thromboses are particularly prone to occur in the territory of the portal system after the injection of various substances favouring coagulation.

Conditions analogous to those set up in these experiments may occur in human beings; but they are, so far as we know, most exceptional. Especially do we lack satisfactory observations, in cases of thrombosis in human beings, of increase of fibrin-ferment in the blood. Considerable quantities of fibrin-ferment, more than are likely to be liberated in any probable circumstances in man, can be injected into the circulation without causing coagulation. Still it is possible that the mechanism by which this excess of fibrin-ferment is neutralised and coagulation prevented may be paralysed under certain conditions. There are certain instances of rapidly-formed red thrombi in vessels with apparently normal walls which, in the absence of other explanation, it would be very convenient to refer to ferment-intoxication. Köhler and Hanau consider that many thrombi, especially those complicating infective and cachectic states, are best explained by supposing a liberation of fibrin-ferment in the blood, and they call them, therefore, ferment-thrombi.

Hayem designates as thrombi from precipitation (thromboses par précipitation) many which others call ferment-thrombi; especially those following injection of various destructive substances into the circulation, and those caused by burns and freezing.

Silbermann and others assert that thrombosis, particularly multiple capillary thrombosis, plays an important part in extensive superficial burns, and in poisoning with various substances destructive to the blood-corpuscles, such as aniline, potassium chlorate, arsenic, phosphorus, sublimate, carbonic oxide, illuminating gas. These views need further

confirmation before they can be accepted, as several observers have obtained only negative results in searching for thrombi in the same class of cases.

Notwithstanding the lack of a substantial basis of demonstrated facts for the opinion that human thrombosis is often caused by liberation of fibrin-ferment in the general blood-stream, it would be quite unreasonable to suppose that chemical changes of the blood are without influence upon the occurrence of thrombosis in man. Indeed, in infective and toxic conditions such changes are doubtless the underlying factors. Both the circulatory disturbances and the alterations in the vascular wall to which we attribute the production of thrombi are the result of damage done to the heart and vessels by bacterial and other toxins. More than this, there is good reason to believe that alterations in the formed elements of the blood, caused directly or indirectly by toxic substances, are of great significance in the etiology of thrombosis. The platelets are in all probability cell-derivatives ; and we may well suppose that damage inflicted upon leucocytes and red corpuscles may favour their production, and that, in consequence of abnormal composition of the plasma, the platelets themselves may more readily undergo viscous metamorphosis, and form plugs. In view of recent observations in favour of the origin of platelets from red corpuscles, the studies of Ehrlich, Maragliano, von Limbeck, and others, concerning degenerations and increased vulnerability of these corpuscles in various diseases, are of interest with reference to thrombosis ; but it must be confessed that we cannot at present make more than a hypothetical application of these results to the explanation of certain forms of thrombosis. Flexner considers that these so-called fibrin-ferment thrombi are agglutinative thrombi formed of red blood-corpuscles.

Increase of Blood-Platelets.—In view of the essential part taken by blood-platelets in the formation of thrombi, it is important to inquire whether thrombosis can be brought into any relation with a pathological increase of these elements. Some observations of the existence of such a relationship are highly suggestive.

Especial difficulties are encountered in the efforts to enumerate the platelets on account of their small size and their viscid consistence, which causes them to clump together. The average number may be taken as 500,000 per c.mm. ; the earlier estimations were considerably lower, between 200,000 and 300,000 (Hayem, van Embden).

There is considerable divergence of statement as to the number of platelets in different diseases. This number is markedly increased in chlorosis (Muir), of which thrombosis is a well-recognised complication. The platelets are increased in post-haemorrhagic anaemia (Hayem, Richardson), which is one of the remoter causes of thrombosis. There is evidence that haemorrhage after childbirth, and in the course of various diseases, favours the occurrence of thrombosis. The platelets are reduced in number in haemophilia, lymphatic leukaemia, and in pernicious anaemia, which, unlike chlorosis, is rarely, if ever, com-

plicated by thrombosis (Hayem, Birch-Hirschfeld, Beugnier-Corbeau). In purpura haemorrhagica there is extreme diminution of platelets, sometimes amounting to total absence (Denys, Hayem, Ehrlich, van Embden), which constitutes the only demonstrated morphological change of the blood in this disease. In febrile infections there is often a correspondence between leucocytosis and the number of platelets. Thus in influenza, pneumonia, erysipelas, meningitis, and septic infections the number of platelets is often increased, in severe cases sometimes diminished; whereas in enteric fever and malaria it is diminished (Hayem, Reyne, Türk, Muir, van Embden). The disappearance of leucocytosis is sometimes followed by increase of platelets. In view of the greater frequency of thrombosis as a sequel than in the course of many acute diseases, the recognition by Hayem of a platelet crisis (crise hématoblastique) is interesting. After the crisis or subsidence of certain infective diseases Hayem observed a rapid and marked increase in the platelets. This was noted after pneumonia and enteric fever. Platelets are said to be often increased toward the end of pregnancy and after delivery (Hayem, Cadet). In various cachectic conditions, in tuberculosis, and, in general, in states of bad nutrition, increase is the rule. Prof. Muir finds that in myeloid leukaemia the platelets are notably increased (art. "Leukaemia," Vol. V. p. 794). In chronic passive congestion, due to heart disease, the platelets are said to be diminished (van Embden). An increase of platelets in various conditions in which they are usually diminished can often be attributed to complications. Upon the whole there is much in support of the view that increase of platelets is an index of lowered resistance of the red corpuscles.

It is fair to say that some of the foregoing statements regarding the condition of the platelets in various diseases need further confirmation, and that in general the subject is difficult and has been insufficiently investigated. Nevertheless we cannot fail to have our attention arrested by a parallelism, in many instances, between disposition to thrombosis and increased number of platelets; and this has appealed so strongly to some that the platelets have been spoken of as thrombocytes. This is an extreme view, for in some instances no such relation between the number of platelets present in the blood and its coagulability is apparent. Pratt indeed found that the number of platelets and the coagulability of the blood did not correspond, and, as lymph which is free from platelets will clot, it is probable that the platelets are one but not the sole source of prothrombin (Wells).

It hardly need be said that the mere increase of platelets is insufficient to explain the occurrence of thrombosis. We are brought back here, as elsewhere, to disturbance of the circulation and changes in the vascular walls as the determinants of the localisation of thrombi; whilst we must recognise changes in the chemistry and morphology of the blood as important predisposing causes.

Calcium-Content.—As is well known, since Arthus and Pagès pointed it out in 1890, the blood cannot coagulate in the absence of calcium

salts. Sir Almroth Wright, who has specially associated the coagulation-time of the blood with its calcium-content, states that by administering calcium salts by the mouth the calcium-content of the blood is raised and its coagulation-time shortened, and conversely that by giving citric acid by the mouth the blood is "decalcified" or has a smaller quantity of calcium salts and a longer coagulation-time. These conclusions, which, however, have been disputed (Addis), have been applied to the explanation of thrombosis in enteric fever. The argument is that the milk, which is rich in calcium salts and forms the staple diet during the fever, raises the calcium-content in the blood, and is thus an important factor in the causation of thrombosis (Wright and Knapp). Briggs refers to a case of recurrent thrombosis with a very short coagulation-time of the blood depending on the presence of fluorine in the beer drunk by the patient.

Localisation.—Thrombosis may occur in any part of the circulatory system. We distinguish therefore arterial, venous, capillary, and cardiac thrombi. Lymphatic vessels may likewise become plugged with fibrin, leucocytes, or foreign material, such as tubercle, cancer, or red corpuscles.

Arterial Thrombi.—The majority of arterial thromboses are caused by some local injury or disease of the arterial wall, or by the lodgment of an embolus. Especially important are the arteriosclerotic thromboses of the brain, heart, and extremities.

Here may be mentioned the varying relations of arterial thrombosis to *gangrene* of the extremities. Thrombosis of arteries, as well as of veins, may be secondary to varieties of gangrene which are not caused by primary plugging of the arteries. Senile gangrene is caused either by embolism which may lead to thrombosis, or by arteriosclerosis, usually associated with thrombosis. In various infective and chronic wasting diseases gangrene may result from primary arterial thrombosis of the class often called marantic. Many of these thromboses are infective in origin; but we have not sufficient information to warrant the assertion that all are caused by micro-organisms.

Of especial interest is the relation of thrombosis to certain forms of so-called "spontaneous" gangrene which may occur in middle life, or even in the young, and are often preceded by definite symptoms indicative of gradual occlusion of the arteries. The condition has been thought to depend on obliterative endarteritis (von Winiwarter, Friedländer), or on primary thrombosis. The latter view is put forward by Buerger who proposes the title obliterating thrombo-angiitis of the lower extremities. This subject is discussed elsewhere (p. 561).

The *action of infective agents* in the causation of focal and diffuse diseases of the arteries is receiving constantly increasing attention. The occurrence of acute and chronic arteritis as a result of various infective diseases—as enteric fever, typhus fever, acute articular rheumatism, variola, scarlatina, pneumonia, endocarditis, septicaemia, syphilis, tuberculosis, leprosy—is now so well established that it is reasonable to

believe that the arterial thromboses complicating or following these diseases are often referable to an infective arteritis.

It cannot be doubted that not a few cases reported in literature as primary arterial thrombosis are to be attributed to embolism which was overlooked. The possible sources of emboli for the aortic system can be usually controlled much more readily than those for the pulmonary arteries; for the latter sources embrace all the systemic veins. These veins may contain mural thrombi, or in places occluding thrombi, which give no signs of their presence. The possibility that an entire thrombus may be detached and transported by the blood-current, so that its original location cannot be determined, is also to be considered. But, after all has been said, it is carrying scepticism to an unjustifiable extreme to refuse to admit the occurrence of primary arterial thrombosis in infective, cachectic, and anaemic states, in circumstances in which the localisation cannot be attributed to arteriosclerosis or other pre-existing arterial disease. Sir Jonathan Hutchinson has reported observations of rapid thrombosis of arteries without obvious disease of the walls.

The most frequent site of arterial thrombosis is in the extremities, and far more frequently in the lower than the upper. Arterial thrombosis, unlike venous, occurs on the right side as often as on the left. Other situations, more or less common, are the cerebral, pulmonary, coronary of the heart, mesenteric arteries, and the aorta and its primary branches.

Venous Thrombi.—These may result from local causes, such as trauma, compression, phlebitis, phlebo-sclerosis, varix, inflammation or other lesion of surrounding parts, and connexion of venous terminals with septic or gangrenous foci.

Vascular thromboses due to general causes are, in the great majority of cases, situated in veins; and to this group the chief medical interest attaches. In special characters of the venous circulation we must seek the explanation of the greater effectiveness of these general causes in veins than in arteries. The physiological peculiarities, partly general and partly local, which come especially into consideration, are—the slower mean speed of the blood in veins than in arteries; the low blood-pressure; the flow from smaller into larger channels; the absence of pulsation; the presence of valves; fixation of the venous wall in certain situations to fasciae and bones; the existence in some places of wide sinuses and ampullary dilatations; the agency of certain subsidiary forces, such as muscular contraction and movements of the limbs, in assisting the flow in the veins; the composition of venous blood, particularly the higher content of CO_2, and perhaps the functions of the capillaries and small veins in the production and absorption of lymph. It is obvious, without detailed explanation, that some at least of these special characters must render the venous system much more favourable than the arterial to the occurrence, under the general conditions known to dispose to thrombosis, of retardation of the blood-current; eddying

motion of the blood, and damage to the vascular wall from impoverished and insufficient blood-supply, or prolonged contact with micro-organisms and toxic substances, the agency of which in the etiology of thrombosis has already been considered.

The best evidence that these mechanical conditions determine the localisation of the majority of thrombi of infective, anaemic, and cachectic diseases is afforded by the marked preference of such thrombi for situations where these conditions are in the highest degree operative. The tendency of venous thrombi to start from valvular pockets has already been mentioned. It is important to note that thrombi due to general causes, unlike those starting from local septic foci, do not begin in the rootlets, but originate usually in the main venous trunks of a member. The very large veins are unusual primary seats of marantic thrombi. Beginning as a rule in a sinus or medium-sized vein, the thrombus may grow centrally into large veins; as from the femoral into the iliacs and vena cava, and peripherally into small veins, not, however, generally reaching the smallest veins. The favourite starting-point of so-called marantic thrombosis of the cerebral sinuses is in the middle of the superior longitudinal sinus at the top of the cranial cavity, whence the thrombus may extend forward, but tends especially to grow toward the torcular Herophili, and into other sinuses and into the cerebral veins. There is, however, no rigid rule in this matter. The plug may begin in other sinuses, or even in the cerebral veins.

In extensive thromboses, such as occur especially in veins of the thigh and leg, it is sometimes difficult to determine the point of origin of the thrombus, and the exact manner of its propagation. Often, however, decisive information can be gained by careful attention to features indicative of the age of thrombi, as already described (p. 700). Thus the autochthonous part of the thrombus is grey, or reddish-grey, and firmly adherent; the continued part often red and more loosely attached, and the older parts frequently softened or liquefied in the centre. By observation of such points as these, the common assumption that a thrombus, occupying continuously both large and small veins, began in the most distal veins and grew thence into the larger channels, can often be shewn to be erroneous. An occluding thrombus may lead to such disturbances of the circulation as to cause the formation of discontinuous multiple thrombi on both the central and the peripheral sides, and these may become connected by red or mixed thrombi. In short, the modes of extension of thrombi are sometimes complicated, and not readily unravelled.

The so-called law of Lancereaux was enunciated by him in 1862 as an explanation of the common site of thrombi in the cerebral sinuses, and at the summits rather than at the peripheries of the extremities; his rule is as follows:—" Marantic thromboses are always formed at the level of the points where the blood has the greatest tendency to stasis, that is, at the limit of the action of the forces of cardiac propulsion and of thoracic aspiration." There are serious physiological objec-

tions to the physical conceptions of the circulation underlying this so-called law, which in any event cannot be accepted in the exclusive form given to it by Lancereaux. Wertheimer has shewn that the effect of thoracic aspiration upon the venous circulation extends to remote parts of the saphenous vein by the side of the tendo Achillis. As the collective sectional area of the veins steadily diminishes from the capillaries to the heart, the average speed of the blood must be greater in the large veins than in the small ones, if the circulation is to continue for any length of time ; and this remains true even when the energy of the blood-current is feeble.

Much more satisfactory, it seems to me, is the explanation offered by von Recklinghausen, of which mention has already been made (p. 703). This explanation places the chief emphasis upon the eddying movement (*Wirbelbewegung*) of the outer lines of flow of the blood-stream when there are counter-currents, or when the blood with retarded flow passes from smaller into larger channels or over obstructions, or especially into spaces relatively too wide for the received volume of fluid. Especially favourable for the appearance of this irregularity of the circulation are the ampullary dilatations just above the insertion of the venous valves, the intracranial sinuses, and the femoral vein near Poupart's ligament, which, in consequence of fixation to bone or fasciae, cannot readily adjust themselves to a lessened volume of blood, and in which counter-currents are set up by the obtuse or right angles at which blood is received from some of the tributary veins. The trabeculae which cross the cerebral sinuses may be a contributory factor. Similar irregularities of the blood-flow must occur with feeble circulation in other situations, as in the pelvic venous plexuses, where wide channels are intercalated between smaller ones, in the recesses of the heart, and in aneurysms and varicose veins. Von Recklinghausen has pointed out that the plexus-like arrangement, the entrance of small veins into large ones, and the close apposition of artery and vein render branches of the renal veins in the kidney susceptible to irregular blood-currents.

The greater frequency of venous thrombosis in the left leg than in the right is attributable to the more difficult return-flow from the former, in consequence of the greater length and obliquity of the left common iliac vein and its passage beneath the right common iliac artery. The presence of an adhesion between the anterior and posterior walls of the left common iliac vein, possibly of developmental origin, which was found in 9, or 29 per cent, of 31 bodies (M'Murrick), may play some part in greater frequency of left-sided thrombosis in the lower limbs. It has been suggested that pressure upon this vein by a distended sigmoid flexure or rectum may likewise contribute to slowing of the blood-current upon this side. The preponderance of thromboses of the left axillary and brachial veins over those of the right is attributed by Parmentier to the greater length and obliquity of the left innominate vein.

As has already been urged, these mechanical disturbances of the circulation are not, by themselves alone, efficient causes of thrombosis. They simply make certain parts of the vascular system seats of election for thrombi. It is quite possible to exaggerate their function in the etiology of thrombosis. The presence of micro-organisms or other changes in the blood may induce lesions of the vascular wall in any part of the circulatory system; and primary thrombi may be formed in situations apparently the most unpromising, so far as the circulatory conditions are concerned; as for instance in the pulmonary veins and in the venae cavae near the heart.

Capillary Thrombi.—The blood in the capillaries remains fluid, even with extensive venous and arterial thrombosis, unless necrosis or gangrene of the tissue ensue, in which case, as in infarctions, the capillaries are always plugged. The interesting fibrinous and hyaline thromboses of the capillaries have already been considered (p. 696).

Cardiac Thrombi.—There is no stranger chapter in the history of pathology than the story of cardiac polypi, from the first observation of fibrinous clots in the heart by Benivieni, in the fifteenth century, until the end of the eighteenth century. It is full of warnings against the uncritical use of post-mortem findings. The cardiac polyps of the old writers were, for the most part, nothing more than ordinary colourless post-mortem clots. Nor has the error of confounding these with genuine thrombi wholly disappeared from medical literature even at the present day. These moist, pale, yellowish, smooth, elastic, uniform, more or less translucent, fibrinous clots, softer or firmer according to their content of serum, non-adherent though entangled with muscular columns and trabeculae, often shewing moulds of the valves or other projecting surfaces with, at least, some red cruor clot at their most dependent parts,—such clots, membranous, polypoid, band-like, or filling the right cavities of the heart and sending worm-like offshoots into the vessels, should never be mistaken for the drier, opaque, grey or reddish-grey, granular, more friable, usually much smaller, adherent, often centrally softened or stratified thrombi.

Although there is a common impression that these fibrinous clots are formed during the death agony, I know of no good reason for such a view. It is much more probable that they are analogous to the buffy coat of clots in shed blood, and are formed after death, when coagulation does not set in until the red corpuscles have settled from the plasma. Liberation of fibrin-ferment, fibrin-content of the blood, sedimentation-time of red corpuscles and coagulation-time,[1] all variable elements, are the leading factors which determine the production of these colourless

---

[1] By "fibrin-content" is meant the amount of fibrin yielded by the blood, and is not of course to be understood as implying the pre-existence of fibrin in the blood. The rapidity of coagulation is an element which is more or less independent of the total yield of fibrin. Red corpuscles settle from plasma or from serum with varying degrees of rapidity in different specimens of blood. Clots also vary much as to their contraction and the separation of serum. Although in using such an expression as "coagulability of the blood" these factors are often confounded, it is important that they should be distinguished.

clots. Most striking examples of colourless clots are found after death from pneumonia and acute articular rheumatism, where the fibrin-content is high, the sedimentation-time rapid, and the coagulation-time slow. The whole doctrine of death from " heart-clot " in these and other acute diseases is based, in my opinion, upon mistaken interpretation of fibrinous post-mortem clots.

The *fresh vegetations* of endocarditis are not generally included in the consideration of cardiac thrombi. Still they are genuine thrombi, and there is no more favourable situation for the study of the formation of mycotic thrombi than the acutely inflamed heart-valve. The first step is the invasion of bacteria, either directly from the blood in the cardiac cavities or through the coronary arteries (Poynton, Coombes), into the endothelial and subendothelial layers. The surrounding cells undergo rapid necrosis with caryorrhexis ; and simultaneously are deposited upon the damaged spot masses of conglutinated platelets followed by leucocytes and fibrin, these masses forming the vegetations. Proliferation of the subendothelial and adjacent cells quickly follows, polynuclear leucocytes migrate into the area, and before long new vessels with organisation of the thrombus make their appearance. A process essentially the same may occur not only in the mural endocardium but also in arteries and veins (vegetative arteritis, vegetative phlebitis, p. 708).

Putting aside these endocardial vegetations, it has been customary to consider the conditions leading to cardiac thrombosis as essentially identical with those of peripheral venous thrombosis ; but there are differences. Cardiac thrombi are found especially in association with chronic diseases of the heart, lungs, arteries, and kidneys ; in all of which, with the exception of pulmonary tuberculosis, peripheral venous thrombosis is uncommon. On the other hand, most of the acute infective diseases, as enteric fever, influenza, pneumonia, which are so important in the etiology of venous thrombosis, are in general of less relative importance in the causation of cardiac thrombosis, although it may occur in these diseases. In cachectic states, especially phthisis and cancer, the conditions as regards the incidence of cardiac and of venous thrombi are more nearly identical, for here thrombi are often enough found in the heart ; particularly when there is well-marked fatty degeneration. Cardiac thrombosis stands in no such peculiar relation to chlorosis and gout as does venous thrombosis, although its occurrence in these diseases is not unknown. The great field for cardiac thrombi is afforded by diseases of the valves and walls of the heart, and especially by dilatation of one or more of its cavities with cardiac insufficiency (asystole of the French school) ; conditions which, in spite of the great retardation of the venous flow, are not often attended by peripheral venous thrombosis, unless in association with diseases known to dispose to the latter.

The seats of election for cardiac thrombi are the auricular appendices and the ventricular apices between the columnae carneae ; the particular situation varying as the cause may affect the whole heart, or only one

side, or one cavity.   In cardiac insufficiency from general or local causes
these recesses and pockets must offer the best possible conditions for
slowing of the blood-current, and especially for the formation of eddies.
That there is no actual stasis of the blood is shewn by the grey or
reddish-grey colour of the thrombi.

The familiar *globular thrombi* (végétations globuleuses of Laennec)
are by far the commonest form of cardiac thrombus.   Varying in size,
usually from a pea to a hazel-nut, they may attain the size of a hen's
egg.   They are usually multiple, and neighbouring ones are connected
by an adherent subtrabecular thrombotic meshwork or membrane, of
which they constitute sessile or pedunculated spheroidal or ovoid projec-
tions.   Their surface may be smooth, or marked by delicate lines or ribs ;
and their interior is usually converted into an opaque grey or brownish-
red grumous fluid, so that the whole resembles a cyst with puriform
contents.   The liquefaction is of the bland variety already described
(p. 700).   Although the projecting covering of these cysts is often only a
thin shell, it rarely bursts.   These thrombi may, however, be the source
of emboli.   Hearts containing these thrombi are often the seat of fatty
degeneration.   Usually no localised mural disease is to be detected with
the naked eye beneath these thrombi, although the microscope generally
shews degeneration or defect of the endothelium.   It is most exceptional
for any trace of organisation to be present in these globular thrombi.

Calcification of cardiac thrombi is a rare event.   Prof. Delépine de-
scribed very fully a cardiolith, and has collected reports of similar cases.
Some of these are probably phleboliths in or derived from varicose veins
which Wagner, Zahn, and Bostroem have described in the wall of the
heart, particularly in the septum auricularum.

Somewhat different as a rule are the *mural thrombi* found on areas of
circumscribed disease of the heart wall ; as on infarctions, fibroid patches,[1]
and gummas, and in partial aneurysm.   These may be identical in ap-
pearance with the ordinary globular cysts ; but often they are flat or
polypoid, stratified, and more intimately incorporated with the cardiac
wall.

Cardiac thrombi may be in the shape of massive or of elongated
polypoid formations, occupying a large part of one of the cavities, and
extending even through valvular orifices into adjacent cavities or vessels.
One of the cavities, usually a dilated auricle, may be nearly filled with
a massive laminated thrombus, as in a case reported by Prof. Osler which
I examined.   There is much resemblance between the clot in these cases
and that found in aneurysms.

Apart from endocardial vegetations not much is known of *infective
thrombi* in the heart, although it is probable that they occur more fre-

---

[1] It is interesting to note that in 1809, Allan Burns in his classical work on *Diseases
of the Heart*, in recording his observations on angina pectoris with calcification of the
coronary arteries and polypi in the left ventricle, called attention to the relations between
disease of the coronary arteries and cardiac thrombosis.   He thus anticipated Weber and
Deguy, and other recent writers, who have emphasised the occurrence of cardiac thrombi in
angio-sclerotic hearts.

quently than is suspected. In a child dead of scarlatina I found, in association with streptococcal mitral endocarditis, softened thrombi containing streptococci in the right auricular appendix. There are a few scattered reports of the discovery of bacteria in cardiac thrombi. Particularly interesting are the observations of Weichselbaum, of Birch-Hirschfeld, and of Kotlar, of tubercle bacilli in white cardiac thrombi. Birch-Hirschfeld found in the appendix of the right auricle, in a case of extensive genito-urinary and chronic pulmonary tuberculosis, a white organised thrombus, which contained many tubercle bacilli and numerous tubercles. In these and similar cases there is difficulty in determining whether the bacteria are the direct cause of the thrombosis, or are secondary invaders. Kotlar interprets his case as the development of miliary tubercles in an organised thrombus.

As there are unquestionable instances of finding emboli derived from venous thrombi in the right heart, the possibility of a thrombus arising secondarily from such an embolus in this situation may be admitted; but I know of no convincing example.

Ball-thrombi, loose in the left auricle, are rare forms of cardiac thrombi. The first observation which I have found of such a thrombus was published by William Wood in 1814, in Edinburgh. As in other typical cases, the loose thrombus was in the left auricle and there was extreme mitral stenosis. The patient, a girl fifteen years old, had the regular symptoms of chronic valvular disease. Death was not sudden. Wood thus describes the appearances: "The substance occupying the sinus venosus of the left auricle, when particularly examined, was found to be of a darkish-red colour, in form completely spherical, measuring rather more than an inch and a half in diameter. It felt firm, but elastic; the surface was everywhere smooth and polished, but having a singularly clotted appearance. Rolling loosely in the auricle, it had no connexion with surrounding parts. When cut open, after having been kept for some days in diluted alcohol, it was found to consist of a sac, one-eighth of an inch in thickness, formed of an immense number of firm, smooth laminae, which could be easily separated from each other. Within the cavity formed by this sac was contained a quantity of coagulated blood." Adherent to the wall of the auricle near the mitral valve was a firm oval thrombus on the free surface of which was a superficial concavity which formed a "kind of socket for the loose ball to roll in." This last feature is a unique observation.

In 1863 J. W. Ogle reported a typical instance of ball-thrombus in the left auricle with extreme mitral stenosis, and accompanied the report with an admirable drawing. In 1877 Dr. Wickham Legg reported, likewise to the London Pathological Society, 2 cases of ball-thrombi in the left auricle with mitral stenosis. He refers to Ogle's specimen which he re-examined, and to a fourth specimen in the museum of St. Thomas's Hospital. One of his cases is unique in the presence of two ball-thrombi in the left auricle. This patient was brought dead to the hospital, and presumably died suddenly in the street. Von Reckling-

hausen's brief description, in 1883, of 2 cases of ball-thrombi is quoted
in the subsequent German records on the subject as the first observa-
tion of this interesting form of cardiac thrombus; although there were
much fuller previous accounts of at least 4 cases, with mention of a
5th, in Scottish and English records extending back as far as 1814;
those of Ogle and Legg being certainly very accessible in the *Transac-
tions of the London Pathological Society.* Macleod's case of loose
thrombus in the right auricle is properly excluded by von Reckling-
hausen from the class of ball-thrombi. If the conception of a ball-
thrombus be simply that of a loose thrombus too large to pass through
the valvular orifice, then van der Byl's case, reported in 1858, should be
included in this class. He found in a case of sudden death "an irregu-
lar, shaggy-looking mass sticking" in the extremely contracted mitral
orifice. When floated out in water this assumed a sac-like appearance,
was about the size of a pigeon's egg, and completed a broken thrombotic
sac in the auricular appendix. This embolus must have been freshly
detached, and had not assumed the typical spherical or ovoid shape of the
ball-thrombus. There have been later reports of ball-thrombi, by Hertz
(2 cases), Osler (2 cases), Arnold, von Ziemssen, Redtenbacher,
Krumbholz, Rosenbach, Stange, and Eichhorst (3 cases mentioned
without any details), making 20, without including Macleod's and
van der Byl's cases.[1] Of these, 15 are reported with sufficient details
for analysis. This form of thrombus, therefore, although rare, is not so
much of a curiosity as has been generally supposed.

Three characters, in my opinion, should enter into the definition of
a ball-thrombus: (i.) entire absence of attachment and consequent free
mobility; (ii.) imprisonment in consequence of excess in the diameter of
the thrombus over that of the first narrowing in the circulatory passage
ahead of it; and (iii.) such consistence and shape that the thrombus must
not of necessity lodge as an embolus in this passage. The third point
does not prejudice the question of the possibility of a ball-thrombus
lodging as an embolus; but it excludes from the group such detached,
shaggy, irregular masses (as in van der Byl's case) as must necessarily be
caught at once as emboli in the narrowed passage in front. According
to this definition a ball-thrombus might, theoretically at least, occur in
any circumscribed or sac-like dilatation of the circulatory system; indeed
von Recklinghausen considers loose phleboliths and cardiac ball-thrombi
as analogous.

All of the cardiac ball-thrombi—as thus defined—hitherto reported,
were in the dilated left auricle; and, with one exception, were associated
with mitral stenosis. In Stange's case there was aortic stenosis, with
slight insufficiency of the mitral valve without stenosis. The agency of

[1] I have also not included Schmorl's case, mentioned by Stange, as it is evidently identical
with that of Krumbholz, nor Fürbringer's case of numerous globular thrombi, the largest
the size of a cherry, in the right auricle, although he reports it as belonging to the group of
ball-thrombi. He is evidently under a misconception of the nature of ball-thrombi. There
was not the slightest reason why these small bodies, many of them indeed minute, if they
were really loose during life, should not have travelled on with the blood-stream.

mitral stenosis in the production of ball-thrombi is not only that it prevents the escape of detached thrombi which might pass the normal orifice, but also that it favours the formation of thrombi in the left auricle, particularly in the appendix; and doubtless also, through the particular disturbance of the circulation, aids in their detachment, increases the tendency to their rotary motion, and prevents the complete emptying of the left auricle during systole, thus rendering more difficult the lodgment and fixation in the valvular orifice of thrombotic masses which at first may be irregular in shape.

The thrombi have varied in size from that of a small walnut to that of a hen's egg; in Wood's case the thrombus was over an inch and a half in diameter, and in Ogle's the weight was more than 4 drams. In 10 the shape was spherical; in 4 ovoid; in 1 (probably of recent separation) a somewhat irregular flattened hemisphere. In 6 the surface was smooth and polished; in 6 marked by granules, lines, ribs, or little depressions; in 2 smooth and knobbed; and in 1 (Redtenbacher's) beset with very fine, grey, fibrinous villi. 9 were centrally softened; 4 solid throughout; and for 2 there is no statement on this point. The colour was grey or reddish-grey; in Wood's darkish-red. In the majority of cases it is said there were adherent thrombi in the left auricle, usually the appendix; and where this is not expressly stated they may have been present. In 5 cases only was there a rough or projecting spot on the surface of the ball indicative of the previous attachment; and in 2 this spot was not at all smoothed off: so that the detachment was evidently very recent, possibly indeed during the necropsy, as in one of the two loose balls in Legg's first case. Krumbholz says that the surface of his thrombus was covered with endothelium. In none, however, was any distinct evidence of organisation detected, for von Ziemssen's statement on this point is too indefinite to be considered.

Ogle, in 1863, clearly recognised the mode of production of a ball-thrombus "by the constant and free agitation of a fragment of fibrinous coagulum separated from some part of the endocardium, and uniformly increased by fresh material at its circumference precipitated from the surrounding blood-stream." Von Recklinghausen has given the fullest and most satisfactory explanation of the spherical shape and smooth surface, in noting that at least some ball-thrombi have a globular shape when first detached; and that irregular bodies, of the consistence of thrombi, rotating in a cavity and growing by successive accretions, assume a spherical shape by a process of moulding, and not by the grinding or breaking off of corners and projections, as was suggested by Hertz to account for the smooth roundness of ball-thrombi. In two or three instances in which the ball-thrombus has consisted of a central irregular nucleus enveloped in a concentrically laminated capsule, it has been assumed that the former represents the original detached part, and the latter successive accretions during free rotation in the auricle. Whilst suggestive of such an interpretation, this structure may, however,

exist in still adherent globular thrombi. It seems to me probable that most ball-thrombi are smooth and at least approximately spherical when first detached. It is difficult to say how much a thrombus may have grown after its separation. ·

In nearly all cases the loose thrombus apparently came from the left auricular appendix, where adherent thrombi were rarely missed when it is expressly stated that they were searched for. In Wood's case the dark-red colour, central blood-clot, and polished surface suggest the possibility that the loose body was a separated polypus resulting from haemorrhage in the wall of the auricle or from a varix; and this opinion is strengthened by the socket-like depression in the adherent thrombus, for it is not clear how such a socket could be formed by a thrombus loose in the auricle; but it might have been the impression left by a polypus attached at some other point.

As regards the clinical significance [1] of cardiac ball-thrombi, Wickham Legg expressed the notion which would probably at first occur to most persons. "A loose thrombus," he says, "in the left auricle would at any time be ready to act as a ball-valve, and stop the circulation in the mitral orifice"; and in this opinion he was strengthened by the presumably sudden death of his patient. Von Recklinghausen, however, who at the time knew only of his own 2 cases and the 2 of Hertz, in criticising a similar opinion expressed by the latter, brought forward several arguments opposed to this notion. The main points of his argument are that instances of sudden death are not infrequent in extreme mitral stenosis without ball-thrombi; that lodgment of the thrombus in the mitral orifice has not been observed, and, even if it were found lying loosely over the orifice at the necropsy, that this would not indicate its position at the moment of death; that the funnel of the stenosed mitral orifice is elliptical in cross-section and shallow, so that a rolling sphere of the consistence of a ball-thrombus could neither completely occlude it nor get wedged in it, nor, if the ball should enter the shallow funnel, is there anything to hold it there, so that the next moment it would roll out. To these points may be added Arnold's argument that the thrombus cannot be horizontally pressed by the auricular contractions against the orifice; for during its systole the dilated auricle does not completely empty itself of blood through the stenosed orifice.

The histories of the cases of cardiac ball-thrombus support in general the position of von Recklinghausen. No symptoms were observed which may not occur in mitral stenosis. Death was gradual in all except 4. In only 1 of these 4 cases of sudden death was there any conclusive evidence that the thrombus was the cause. This was Prof. Osler's second patient upon whom the necropsy was made in my laboratory by Dr. Flexner. The patient, a woman aged twenty, was seen in good condition a few hours before death. At 4.30 A.M. she was found by the nurse

---

[1] In order to complete without interruption the description of ball-thrombi I introduce here their clinical significance, although the consideration of the symptoms of thrombosis is taken up subsequently.

very cyanotic, she gave a gasp or two, and died in a few moments. At the necropsy were found marked hypertrophy and dilatation of the left auricle, right ventricle, and to a less extent of the right auricle ; without dilatation or hypertrophy of the left ventricle. The segments of the mitral valve were thickened, adherent, and drawn down by great shortening of the chordae tendineae, so as to form the wall of a distinct funnel. There were no fresh vegetations and no oedema. The stenosis was not extreme, the mitral orifice readily admitting the index finger. The other valves and the coronary arteries were normal. An ovoid ball-thrombus, resembling a thick chestnut, measuring 4 × 3·5 × 3 cm., was found, upon opening the heart, occupying with its smaller end and completely blocking the funnel-shaped mitral orifice, from which it was readily removed by the fingers. At one pole of the thrombus was an irregular, roughened spot indicating a former attachment, probably to a thrombus in the appendix. There can be no reasonable doubt that the thrombus in this case was the cause of the sudden death, which is certainly not a common occurrence with such moderate uncomplicated mitral stenosis at the age of this patient. Indeed sudden death is less common in uncomplicated mitral stenosis than in aortic valvular disease ; as the former occurs often in young women, and is usually unassociated with disease of the coronary arteries. In the three other instances of sudden death with ball-thrombus the ages were twenty-one, twenty-two, and thirty-nine years respectively. Only in one of these was the thrombus a perfect sphere ; so that it would appear that an oval thrombus is more likely to plug the mitral orifice than a spherical one. This view is strengthened by the fact that of the four observations of ovoid thrombi in three death was sudden. In the light of our case it seems clear that a ball - thrombus may "act as a ball-valve and stop the circulation in the mitral orifice," as suggested by Legg ; but it is certain that this is an exceptional occurrence.

Under the name of cardiac *pedunculated polyps* various formations have been described. Some of these are ordinary unorganised or partly organised polypoid thrombi, about which nothing more need be said ; but others are very remarkable structures which occupy an entirely exceptional position, not only among cardiac thrombi but among thrombi in general. In the older records some of the latter were described as fibromatous or myxomatous polyps,—two as haematoma ; but in the later reports most have been recognised as organised thrombi. They are often called true polyps in distinction from the false polyps of the older writers.

The literature of the subject begins with Allan Burns in 1809. References to many of the cases will be found in the papers of Hertz, zum Busch, and Pawlowski. Among the noteworthy observations since Hertz are those of Czapek, Voelcker, Bostroem, and Ewart and Rolleston. I have found records of 33 cases, at least 20 of which were well-characterised, organised, pedunculated polyps. 25 sprang from the wall of the left auricle, usually the septum ; 4 from the right auricle ; 4 from the left ventricle.

The following are the more notable features of these curious formations :—In many instances no cause whatever could be found for their occurrence. The hearts containing them were often otherwise entirely normal, with the exception of changes manifestly secondary to the polyp, such as nodular fibroid thickening of the mitral segments and dilatation and hypertrophy of the left auricle and right ventricle. Unlike other cardiac thrombi they are solitary formations, and often unassociated with ordinary thrombotic deposits. The vast majority of these polyps spring from the septum of the left auricle near the fossa ovalis with a short pedicle, sometimes narrow, sometimes broad. They are firm or gelatinous, elastic, ovoid or pear-shaped formations, in several instances hanging down into the left ventricle with a constriction corresponding to the mitral orifice. The surface is usually glistening, smooth, and covered by a distinct membrane which often resembles the endocardium. It may present calcified, atheromatous, or pigmented patches ; and upon it may be irregular knobs and depressions. The colour is described as yellowish-grey, dark-red, or brownish-red ; the colour often varying in different parts of the polyp. A prevailing dark-red colour has been observed in a large number of the cases. In distinction from nearly all other cardiac thrombi, these polyps are more or less organised by connective tissue and vessels ; the organisation in some being little marked, in others so far advanced that the structure resembles that of a fibroma or myxoma. The central part is often unorganised or less organised than the base and periphery. In the incompletely organised forms the substance of the polyp is composed of red corpuscles, fibrin, granular detritus, yellow blood-pigment, leucocytes, and other cells between the blood-vessels and fibrous septa. Laminated fibrin may be present in the peripheral layers. Unless ordinary thrombi are likewise present, emboli are usually missed. A further distinction from the ordinary cardiac thrombi is that many of these polyps, by encroaching upon the mitral orifice, are of as much clinical as anatomical interest ; the diagnosis during life in these cases being mitral disease, usually stenosis.

We have no satisfactory explanation of these pedunculated polyps. The ordinary causes of thrombosis are generally absent. Their commonest site of origin, the septum of the left auricle near the oval fossa, is not a usual situation for ordinary thrombi. They stand in no demonstrable relation to patency of the foramen ovale or to circumscribed endocarditis in this situation.

Bostroem has suggested that an explanation may be found in the existence of varicose veins which have been observed repeatedly in the septum, usually near the posterior quadrant of the foramen ovale. A difficulty with this explanation is that nine out of ten of the varicosities observed by Wagner, Zahn, Rindfleisch, and Bostroem were on the right side of the septum. In one instance, however, Bostroem found in the left auricle a spherical, dark-red polyp, 13 mm. in diameter, attached by a short narrow stem to the septum on the posterior lower margin of the completely closed foramen ovale. This proved to be a varix con-

taining a phlebolith. In another case a similar thrombosed varix had broken from its pedicle on the septum of the right auricle, and was lodged as an embolus in a branch of the pulmonary artery. He suggests this as a possible source of ball-thrombi. Of still greater significance is Bostroem's demonstration in an old museum specimen, labelled "thrombosis of the right auricle (pedunculated cardiac polyp) peripherally organised," of an enormous completely thrombosed varix almost filling the right auricle. In still another case he proved conclusively that a broad-based, nearly spherical polyp, occupying a large part of the right auricle, was a haemorrhage in the wall of the auricle. Choisy and Nuhn long ago interpreted the polyps, which they observed, as the result of haemorrhage in the septum of the left auricle.

In the light of Bostroem's interesting investigations, more attention than has been customary should be given to the possibility that pedunculated polyps are the result of haemorrhage or are thrombosed varices. Most competent investigators, however, have unhesitatingly pronounced the polyps which they have examined to be organised thrombi. It would appear, therefore, that the nature of these formations is not always the same. At any rate the great majority of the typical pedunculated polyps, to which the preceding description applies, occupy a position quite apart from ordinary cardiac thrombi. As already remarked, by no means all of the cases described as true cardiac polyps belong to this peculiar group. Some, as in Krumm's case, are ordinary partly organised thrombi attached to diseased patches of the heart-wall.

Association with certain Diseases.—Thromboses may be divided, as regards their clinical relations, into the following groups : (i.) those resulting from direct injury of vessels, including the penetration of foreign bodies ; (ii.) referable to diseases of the vascular wall, as to arteriosclerosis, syphilitic arteritis, aneurysm, varix ; (iii.) caused by lesions of neighbouring parts ; (iv.) thromboses of arteries and veins whose terminal branches end in septic and gangrenous areas ; (v.) complications or sequels of (*a*) infective diseases, (*b*) cachectic and anaemic states, (*c*) cardiac disease, (*d*) certain constitutional diseases ; (vi.) idiopathic and primary infective thromboses. Several of these groups, being mainly of surgical interest, will not be considered here. The thromboses embraced in the fifth and sixth groups are of such special medical interest that it is proper in this article to give them particular attention ; although it is manifestly impossible within reasonable limits to take up all in detail. Some of them are noticed in other parts of this work.

Enteric Fever.—Cardiac thrombosis is a rare complication of enteric fever. In 2000 fatal cases of enteric fever in Munich there were only eleven instances of acute endocarditis (Hölscher). Girode, Viti, Carbone, and Vincent have found the typhoid bacillus in endocardial vegetations ; and vegetative endocarditis has been produced experimentally by intravascular injections of pure cultures of the typhoid organism combined

with injury to the valves. More frequently the endocarditis has been due to secondary infection. In rare instances in the course of enteric fever globular thrombi are formed in the auricular appendices and ventricular apices; and these, as well as the endocardial vegetations, may be the source of emboli.

Arterial thrombosis is a still rarer event, but, in consequence of its gravity, an important one. Bettke, in 1420 cases, found 4 of gangrene of the extremities; but in 2000 Munich necropsies no instance is recorded, a result in contrast with 59 of thrombosis of the femoral vein in the same series. Keen, in his admirable monograph, has collected and analysed 115 cases of gangrene associated with enteric fever and due to plugging of the arteries. In 21 cases arterial thrombosis was observed without gangrene, the absence of which is much more common with thrombosis of arteries of the upper extremity than of the lower. The earliest appearance of the gangrene was on the fourteenth day; the latest in the seventh week. In the great majority of cases the thrombus was seated in the arteries of the extremities; and in those of the lower far more frequently than of the upper. In 8 out of 11 cases of arterial thrombosis of the lower extremities, collected by Barié, the posterior tibial artery was concerned. In contrast with venous thrombosis the right side is the seat as often as the left.

Other arteries, as the pulmonary, the superior mesenteric, and the cerebral, may become thrombosed. Fatal cases of typhoidal thrombosis of the middle cerebral artery, or its branches, have been reported (Huguenin, Barberet and Chouet, Vulpian, Osler (2 cases), and Thayer); and other cases have been recorded in which the diagnosis of cerebral thrombosis was made from the symptoms. In Dr. Osler's first case, in which Dr. Flexner and I examined the brain, the middle cerebral artery was open; but the ascending parietal and parieto-temporal arteries and their branches were occluded by adherent, firm, mixed thrombi. The adjacent brain-substance was studded with punctiform haemorrhages, but not much softened. Typhoid bacilli were widely distributed in the body.

The arterial thrombosis may be secondary to embolism; but in the great majority of cases it has been reported as autochthonous. In the older records the thrombosis has been usually regarded as marantic; whereas the tendency now is to refer it to an infective arteritis; a view which is probable, although we have few conclusive observations in its support. Rattone and Haushalter claim to have demonstrated the typhoid bacillus in the walls of occluded arteries; and Gilbert and Lion, Crocq, and Boinet and Ramary have produced an acute aortitis experimentally, by injuring the vessel-wall and then injecting typhoid bacilli into the circulation. The bacteriological studies are too meagre and unsatisfactory to warrant any definite statements as to the specific cause of arterial thrombosis in enteric fever. As has been mentioned on p. 714, the liability to thrombosis has been connected with an increased calcium-content in the blood and a corresponding shortening in the coagulation-tone of the blood in convalescence (Wright and Knapp).

The far commoner venous thrombosis of enteric fever has been considered in Vol. I. p. 1112 ; and the points bearing on its causation have been presented under Etiology. Richardson has called special attention to the " marantic " thromboses of intracranial veins complicating enteric fever.

Influenza.—Nearly all of our knowledge of thrombosis in influenza dates from the pandemic of 1889-90, which led to the recognition of countless complications, among which those of the circulatory system occupy a less prominent place than the respiratory and nervous. *Arterial thrombosis*, although far from common, is still not an extraordinarily rare complication or sequel of influenza. It is more common in this disease than in any other acute infection. In a few instances it appeared as early as the third to the fifth day, but in most during convalescence. Over 40 cases of arterial thrombosis or of gangrene accompanying or following influenza have been reported. References to many of these will be found in the monographs of Leichtenstern, of Lasker and of Eichhorst ; but their lists are far from complete. In a partial collection of the cases I find that the popliteal artery was occluded in 6 ; the femoral in 4 ; the iliacs, the axillary, the brachial, the pulmonary, and the renal each in 2 ; and the central artery of the retina (embolism being probably excluded) in 1. The cerebral arteries were repeatedly invaded. In several instances there were multiple thrombi. Symmetrical gangrene following bilateral plugging was observed in a number of cases. Gangrene was observed in all the cases of occlusion of the arteries of the lower extremities, but not regularly with that of the upper.

It is difficult to say in how many cases the occlusion was due to embolism. Endocarditis is a rare but recognised complication of influenza, and globular cardiac thrombi have also been observed. In the great majority of cases it seems clear that there was primary arterial thrombosis.

Venous thrombosis is a far commoner result of influenza ; and has been the subject of a special memoir by Chaudet, and of numerous articles in the medical journals of all countries. 25 cases are recorded in Guttmann and Leyden's collective investigation, and many additional ones are to be found in the vast literature on influenza. Dr. Goodhart, in his article on "Influenza" (Vol. I. p. 946), notes the frequency and the occasional diagnostic value of this complication, which may appear during the course of the disease or weeks afterwards, and in mild as well as severe cases. In the great majority of instances the femoral vein was attacked ; but the veins of the upper extremity were thrombosed more frequently than in other acute infective diseases. Leichtenstern notes the acute onset and course in some of the cases. There are records of thrombosis of the cerebral sinuses in influenza. Klebs and Kuskow describe capillary thrombi in the lungs.

Few observers are satisfied with the explanation of either the arterial or the venous thromboses of influenza as marantic. Leyden suggests as a cause increase of blood-platelets from disintegration of leucocytes. Evidences of such disintegration, or of masses of platelets in the blood,

have been noted by Klebs, Chiari, and Bäumler. Maragliano observed the onset of necrobiotic changes of the red corpuscles in influenza almost immediately after withdrawal of the blood. French writers for the most part attribute the thrombosis to infective arteritis or phlebitis (artérite grippale, phlébite grippale). Rendu, however, in his case of arterial thrombosis rejects this explanation, as he found the walls of the thrombosed arteries entirely normal (nothing is said of a microscopical examination) ; and he attributes the thrombosis to feeble circulation. In his case there was also a thrombus with softened centre in the left ventricle, and the occlusion of the artery may have been due primarily to an embolus. Gerhardt attributes the gangrene in his case to spasm of the arteries, considering it therefore analogous to symmetrical or arterio-spastic gangrene. In support of the more probable view that the thrombosis is the result of some change in the vascular wall, directly referable to infection or intoxication, Kuskow observed with great frequency degeneration, proliferation, and desquamation of the vascular endothelium in influenza. In a fatal case of influenzal phlegmasia alba dolens Laveran found streptococci in the blood. These organisms have often been found in the blood and organs of those dead of influenza.

In a remarkable case of multiple thrombotic vegetations present in large numbers in the pulmonary artery, especially in the left main branch, and also on the pulmonary valves (other valves normal), Flexner in my laboratory found in the thrombus, chiefly enclosed within polynuclear leucocytes, very numerous, extremely delicate bacilli, which were identified as the influenzal bacilli of Pfeiffer. This establishes the occurrence of an acute arteritis and thrombosis due to the bacillus of influenza.

Pneumonia.—The sixteenth-century error of mistaking for ante-mortem coagula the firm, yellowish-white cardiac clots, intimately intertwined with the columnae carneae, and found after death from pneumonia more frequently than from any other disease, had not wholly disappeared at the end of the nineteenth century. Genuine ante-mortem thrombi in the cavities of the heart occur in pneumonia, but they are rare ; being much less common than in many diseases in which death from "heart-clot" is not mentioned as a special danger. Acute valvular endocarditis is a well-recognised complication of pneumonia. The frequency of the pneumococcus in the blood of cases of pneumonia enables the arteries and veins to become directly infected, with resulting pneumococcal arteritis and phlebitis. Mention has already been made of coagula in pulmonary vessels directly connected with the inflamed lung (p. 696).

Eichhorst has collected 10 cases of gangrene of the extremities in pneumonia, and in the cases of this sequel recorded by Cruveilhier, Benedikt, Brunon, Rendu, Leyden, Zuppinger, Blagden, and McGregor, there was arterial thrombosis. Blagden's patient was a woman ninety-two years old, and M'Gregor's patient a male aged eighteen. In Leyden's case there was thrombosis of the lower end of the abdominal aorta. Dr. M'Gregor has collected a number of cases of gangrene of the extremities in pneumonia due to embolism.

Venous thrombosis is more frequent than arterial as a complication or sequel of pneumonia. In 3066 cases of pneumonia thrombosis occurred in 20, or once in 150 cases (H. Mackenzie). In 1902 Steiner collected 41 cases of thrombosis. The femoral vein is the one nearly always affected, and most often the left one. It usually occurs during convalescence, and so may be considered as a sequel rather than a complication of pneumonia. The affection, if one may draw any conclusion from so small a number of cases, is more common in women than in men. Of 367 cases of pneumonia, observed by Dickinson, peripheral venous thrombosis occurred in 7, of which 4 were in young women, 2 of these being chlorotic. Laache ranks pneumonia next to influenza and enteric fever as regards the frequency of occurrence of peripheral thrombosis; but this event is far commoner in the last two diseases. The affection presents the same general characters as the phlegmasia alba dolens of enteric fever. Da Costa very plausibly attributes it to a primary infective phlebitis, and pneumococci have been found in the thrombi (Mya, Gaultier).

Acute Articular Rheumatism.—There was a time when rheumatic phlebitis ranked in importance next to the puerperal form; but it is now recognised that most of the cases of thrombosis attributed by the older writers to rheumatism had nothing to do with acute articular rheumatism. Schmitt and Vaquez have sifted the reported cases, and they find that, while phlebitis or venous thrombosis is to be recognised as a complication of genuine acute rheumatism, it is a rare one. The infrequency of this event is noteworthy in view of the fibrinous state of the blood and the frequency of acute endocarditis. According to Blumer there are not more than 25 to 30 authentic cases on record; the process is generally due to phlebitis, and usually, though not exclusively, occurs in the lower extremities. Legroux reports an instance of thrombosis of the brachial artery without gangrene in acute articular rheumatism.

Appendicitis.—Mention may be made of the occurrence of thrombosis in appendicitis, as this affection is of medical as well as surgical interest. Besides the septic thrombophlebitis of the mesenteric and portal veins, thrombosis of the iliac and femoral veins may occur on the left side as well as on the right. Among 3334 cases of appendicitis in various London Hospitals, 29 shewed thrombosis (Haward). The published reports indicate that this is more common on the right side; but the left femoral vein is comparatively frequently affected, and some statistics shew that it is more often involved; thus in 1000 cases of appendicitis at the London hospital, there were 12 instances of femoral venous thrombosis, 11 of the left leg, and 1 of the right (Treves and Lett). It is interesting to note the analogy of appendicitic thromboses to puerperal thromboses, where we also have septic and suppurative thrombi in veins immediately adjacent to the inflamed organ, and less manifestly infective thrombi in the veins of the lower extremities. It is probable, however, that the latter thrombi in appendicitis, as well as in the puerperal cases, are frequently caused by bacteria, and oftenest by streptococci, which are

concerned in both affections with great frequency. Among the 29 cases of thrombosis mentioned above, pulmonary embolism occurred in 7.

Other Acute Infective Diseases.—It would lead too far to continue a detailed inquiry into the association of thrombosis with other acute infective diseases. It must suffice to specify typhus fever, relapsing fever, dysentery, erysipelas, suppurative tonsillitis, diphtheria, variola, scarlatina, measles, Asiatic cholera. In many instances thrombosis, as associated with specific infective diseases, has been due to a secondary septicaemia, streptococci being the commonest secondary invaders. The disposition in or after typhus fever to arterial as well as to venous thrombosis should be especially emphasised.

Tuberculosis.—The consideration of thrombosis directly referable to tuberculous processes adjacent to vessels need not detain us. The occurrence of intimal tubercles, where the evidence is conclusive that tubercle bacilli have penetrated the inner lining of vessels directly from the circulation in the main channel, may be mentioned not only as a cause of thrombosis, but also as an interesting illustration of this mode of infection of the vascular wall. Several instances of endocarditis caused by the tubercle bacillus have been described, and mention has already been made of tuberculous cardiac thrombi (p. 721). Michaelis and Blum have produced vegetative tuberculous endocarditis experimentally, by injuring the valves in rabbits and then injecting tubercle bacilli into the ear veins. Particularly demonstrative of infection taking place through the vascular endothelium are the rare instances of tuberculous foci in the aortic intima, without invasion of the outer coats, and without tuberculosis of neighbouring parts. Instances of this form of aortic tuberculosis have been described by Flexner, Blumer, Stroebe, Aschoff, Schmorl, and Krumbhaar who has recently discussed the subject and collected a number of cases. These rare instances are cited because they furnish conclusive proof that bacteria may penetrate the inner lining of vessels from the main channel, even where the blood-current is forcible ; and may set up inflammation of the intima with secondary thrombosis. Hektoen's interesting observations of changes in the intima of vessels in tuberculous meningitis furnish additional evidence along the same lines.

Arterial thrombosis, apart from the forms just mentioned, which are of pathological rather than clinical interest, is a rare event in tuberculosis. Most common are the instances of thrombosis of the pulmonary artery or its main branches in phthisis. Dodwell mentions an instance of thrombosis of both popliteal artery and vein. Vaquez, in chronic pulmonary tuberculosis, described an interesting case of thrombosis of the left subclavian, axillary and brachial arteries with gangrene of the arm ; he found streptococci in the plug and in the wall of the vessel, including the vasa vasorum, but no tubercle bacilli.

On the other hand, peripheral venous thrombosis in advanced phthisis is a comparatively common and well-recognised ailment. In the great majority of cases veins of the lower extremities, the left

oftener than the right, have been plugged; but the thrombus may be in the inferior vena cava, or other veins, or the cerebral sinuses. Dodwell, in his valuable paper on this subject, places the proportion of cases of phthisis with this complication at about 3 per cent. In about 1300 necropsies of phthisical patients at the Brompton Hospital there were 20 cases of thrombosis of veins of the lower extremities (1·5 per cent). In 1778 cases analysed by Ruge and Hierokles there were 19 cases, or 1·1 per cent, of thrombosis. The upper extremities are very rarely affected; Blumer (189) records 1 case, and refers to 10 collected by Aldrich.

The peripheral venous thromboses of advanced phthisis are usually cited as typical examples of the marantic or cachectic form. Dodwell, however, while recognising enfeebled circulation as a factor, is inclined to refer the thrombosis to some unknown change in the vascular wall set up by a complicating septicaemia. He emphasises the infrequency of venous thrombosis with the acute and the very chronic forms of phthisis, and its relative frequency with an intermediate type with remittent or continued fever. He also noted association with intestinal and laryngeal ulceration in a larger percentage of the thrombotic cases than the average. As is well known, secondary septicaemias, usually streptococcal, are very common in pulmonary tuberculosis.

There are several records of bacteriological examination of the peripheral thrombi in phthisis, which shew that they may be of mycotic origin. Vaquez found tubercle bacilli, without other micro-organisms, in a thrombus of the left profunda and femoral veins. They were present also in the wall immediately beneath the endothelium, but were absent from the media and adventitia. Sabrazès and Mongour in two instances found tubercle bacilli both in the plug and in the wall of a thrombosed iliac vein; they were associated with micrococci. Tuberculous endophlebitis has been found in thrombosis of the superior vena cava (Banti) and of the inferior vena cava (Griffon). More frequently micrococci, presumably pyogenetic, have been found, without tubercle bacilli, in the thrombi and vascular walls: examples of this are recorded by Vaquez. Notwithstanding these suggestive bacteriological observations it would be quite premature to conclude that all the peripheral venous thromboses of phthisis are referable to direct infection of the venous wall by bacteria. In a rather old thrombus of the iliac and femoral veins in pulmonary tuberculosis I failed to find any micro-organisms, either by culture or by microscopical examination.

Hirtz has called attention to the occurrence of phlebitis in the initial stage of pulmonary tuberculosis. Some cases so reported have appeared to be chlorotic in origin.

Syphilis.—Thrombosis secondary to endarteritis is well known. Syphilitic thrombophlebitis is rare, and Dieulafoy states that 36 cases are the total reported. This subject is referred to on p. 685.

Gonorrhoea.—Here again thrombosis is rare; Heller has collected 26 cases. Three-quarters of the cases occur in men, and usually in the third decade of life.

Cachectic States.—Of other marasmic or cachectic states, in which thrombosis is somewhat frequent, may be especially mentioned those resulting from cancer, dysentery, chronic diarrhoea, gastric dilatation, prolonged suppurations especially of bone, anaemia from loss of blood, and syphilis. Pulmonary tuberculosis has just been considered. It is especially in the young and the very old that these conditions are most likely to produce thrombosis. Thromboses of the cerebral sinuses, and of the renal and other veins, in marasmic infants, particularly after diarrhoea, are well recognised. Peripheral venous thrombosis is more often associated with the waxy kidney than with other forms of Bright's disease. The thrombi occasionally found in the renal veins in chronic diffuse nephritis are probably due to local causes, and not to cachexia.

There is a French thesis by Rigollet on thrombosis in malaria, and Pitres, Bitot, and Regnier have likewise called attention to the subject. It is doubtful whether there is any relation between malaria and thrombosis. In over 2000 cases of malaria observed at the Johns Hopkins Hospital there was only one example of thrombosis (Futcher).

Trousseau attached some diagnostic significance to the occurrence of thrombosis in cancer. There have been instances of latent cancer of the stomach in which peripheral venous thrombosis was the first symptom to attract attention, as indeed it was in Trousseau himself who died of gastric cancer. Gouget has reported a case of widespread venous thrombosis, of eight months' duration, which was the only affection observed during life ; at the necropsy a small cancer of the stomach was found. In a latent case of gastric carcinoma, with profound and progressive anaemia and absence of gastric symptoms, there were thrombi in fourteen or fifteen veins and great muscular tenderness (Osler and M'Crae).

The principal seats of cachectic thromboses are the auricular appendices, between the columnae carneae of the right heart, in the veins of the lower extremities, the cerebral sinuses, the pelvic veins, and the renal veins. Lancereaux has strongly urged that this form of thrombosis never occurs in the arteries. Doubtless in not a few reported cases embolism has not been satifactorily excluded ; but older observations of Charcot and von Recklinghausen, and several recent ones, leave no doubt of the occurrence of genuine so-called marantic or cachectic thrombi in arteries, even in the aorta.

Whilst pre-existing vascular disease, particularly arteriosclerosis and varicose veins, are predisposing conditions, these plugs are often seated upon intimae which shew very slight alteration. Indeed competent observers have repeatedly described the vessel-wall beneath marantic thrombi as normal. Whilst secondary septic infections often participate in the causation of cachectic thromboses, the view that all have this origin is at present unsubstantiated. It is clear that enfeebled circulation is of importance in their causation ; but, for reasons already stated, there must be some additional element, which, in many cases at least, cannot well be other than changes in the composition of the blood. The nature of these changes is not known. Possibly increase of platelets, or

a special vulnerability of cells, perhaps of the red corpuscles from which platelets may be derived, is concerned.

Cardiac Incompetency.—I have already had occasion in this article to speak repeatedly of the importance of feebleness of the general circulation in the causation of thrombosis. Thrombi in the heart itself have been considered (p. 718). In this respect attention is called to the occurrence of peripheral venous thrombosis in chronic passive congestion due to cardiac incompetency, chiefly from valvular disease. Especially noteworthy, in view of the slow venous circulation and the frequency of cardiac thrombi in this condition, is the infrequency of peripheral thrombosis. Hanot and Kahn, in reporting an instance of thrombosis of the right subclavian vein, say that they were able to find in the French literature, which is exceptionally rich in clinical contributions to the subject of thrombosis and phlebitis, only five additional observations of peripheral venous thrombosis in cardiac disease. I do not think that this complication is quite so rare as would appear from this statement; for, without any pretence to completeness, I collected in 1900 the published accounts of 23 cases, which, with 4 cases from the Johns Hopkins Hospital, made up a total of 27 cases. In 1906 Desquiens collected 39 cases, and Dévé, who embodied these cases, states that in 38 out of 40 cases some branch of the superior vena cava was involved. The most notable point concerning my 27 cases is that 23 were thromboses of veins of the neck or upper extremity or both, far more frequently of the left than the right side. Of my 23 cases of thrombosis of the upper extremities, 14 were unilateral and 7 bilateral; in only 2 cases was the right side exclusively affected. In addition to the reasons given on p. 717 for the greater frequency of thrombosis on the left upper extremity, I would urge pressure, direct or indirect, on the left subclavian vein exerted by the dilated left auricle and the dilated pulmonary vessels. It may be that femoral thrombosis is more common in heart disease than would appear from these figures; it is less likely to be reported than thrombosis of the neck and arms, and, on account of the oedema attributable to cardiac insufficiency, may more readily be overlooked both at the bedside and the autopsy table. When, however, we consider that Bouchut places the ratio of thrombosis of the upper extremity to those of the lower at 1 to 50, the relatively large number of the former associated with cardiac disease is certainly most striking. My four cases of venous thrombosis of the lower extremities in heart disease had little in common with the other 23 cases; 2 were in old people, and 2 were imperfectly reported. Of the 23 patients with thrombosis of the upper extremities, 17 were females, and of those whose ages are given nearly half were between fifteen and thirty years of age. The associated valvular disease of the heart is almost invariably mitral, stenosis with or without incompetence taking the lead. Of the 23 cases, 19 were fatal and 4 recovered.

The relative freedom from peripheral venous thrombosis in cardiac disease, in spite of conditions of the circulation apparently favourable

to such an occurrence, may perhaps be attributable partly to the reduction in platelets in this condition (which has been noted by van Embden), and partly to the absence of von Recklinghausen's *Wirbel-bewegung* (p. 717), an irregularity of the circulation which occurs especially in vessels too wide in proportion to the amount of blood which they receive.   Hanot and Kahn refer the thrombosis to a cachectic state developing in the last stages of cardiac disease.   Huchard likewise attributes it to cardiac cachexia associated with secondary infection.   Three cases are referred by Dr. Poynton to rheumatic infection, but the bacteriological examination was negative (209). Although there is little proof available, the thrombosis is most probably due to bacterial activity.

As will appear later (p. 812), there is evidence that arterial plugging associated with mitral stenosis is due oftener to primary thrombosis than is generally supposed.

Chlorosis.—The association of thrombosis with chlorosis is of peculiar interest.   Sir Clifford Allbutt, in his article on Chlorosis (Vol. V. p. 713), has sketched the more essential features, but has referred some points for consideration here.   In the older literature there are reports of plugging of the veins in young women which undoubtedly pertain to chlorosis.   Thus William Sankey, in 1814, says "I have met with 2 cases in young women, not after parturition ; both were severe and well marked ; both had obstructed menses."   But Trousseau, with his pupil Werner, in 1860 was the first to draw distinct attention to this associa-tion.   References to the more important records, up to 1898, will be found in an article by Schweitzer, from Eichhorst's clinic.

Although thrombosis is not a common complication of chlorosis, it is sufficiently frequent to indicate a special tendency to its occurrence in this disease ; a tendency calculated to arrest attention on account of the age and the class of the patients, the obscure causation, and the unexpected and calamitous termination which it may bring to a disease ordinarily involving no danger to life.   Some idea of the frequency of chlorotic thrombosis is perhaps afforded by the statements that von Noorden observed 5 instances in 230 chlorotics, and Eichhorst 4 in 243. The list of reported cases was brought by Proby in 1889 to 21, by Bourdillon in 1892 to 32, and by Schweitzer in 1898 to 51.   I have found reports of 30 additional cases not included in these lists, and am indebted to Dr. W. S. Thayer for an unpublished personal obser-vation ; making a total of 82.   (References will be found at the end of this article.)   I have also seen 12 other cases mentioned, but without sufficient detail for statistical analysis ; and I have come across several references to articles on the subject not accessible to me. Slavonic and Italian literatures have not been searched, and the American to only a small extent.   I have no doubt that mention or reports of over 100 cases of thrombosis chlorotica could be gathered by thorough overhauling of medical books and periodicals.   31 of my cases are from French literature, 25 German, 18 British, 3 Scandinavian, 2 American,

and 1 Italian. It would, however, be quite unwarrantable from this literary inequality to infer any difference in the incidence of the affection according to race or country.

The statistical study of these 82 cases brings out a number of interesting points, of which some only are directly pertinent to this article. Thrombi in the heart are very rarely mentioned in the post-mortem reports. There were only four instances of primary arterial thrombosis, two being of the middle cerebral arteries (Vergely); one of the pulmonary without thrombosis elsewhere (Rendu), and one of the right axillary with gangrene of the hand and recovery (Tuckwell). Tuckwell reported his case as one of embolism; but it is usually included among the arterial thromboses, and probably with as much or as little right as the others.

All the remaining 78 cases were venous thromboses. There was thrombosis of the cerebral sinuses in 32 cases (39 per cent), 6 (19 per cent) of these being associated with thrombosis of the lower extremities. In four instances thrombi extended from the sinuses into the internal jugular veins. Unquestionably sinus-thrombosis is represented by too high percentage figures in my list, for the obvious reason that reports of an affection of such gravity and such interest, especially to neurologists, are much more likely to get into print than those of ordinary femoral thrombosis. Still the figures are impressive, and indicate that sinus-thrombosis is not of great rarity in chlorosis; to which malady a leading place among the causes of spontaneous thrombosis of the cerebral veins and sinuses in women must be conceded.

In 51 of the 82 cases there was venous thrombosis of the extremities (62·2 per cent—too low a percentage, as already explained); 50 being of the lower and 3 of the upper, of which only 1 was limited to the upper extremity. Of the 50 cases of thrombosis of the lower extremities (which are probably involved in at least 80 per cent of all chlorotic thromboses), the process was bilateral in 46 per cent, and unilateral in 54 per cent—34 per cent being left-sided and 20 per cent right-sided. The usual preference of femoral thrombosis for the left side is shewn by the beginning of the affection in the left leg in 64 per cent of the thromboses of the lower extremities, in the right leg in 29 per cent, and on both sides simultaneously in 7 per cent. There is in the list 1 case (Kockel's) with meagre history, in which no mention is made of thrombi outside of the upper part of the inferior vena cava; death ensued from pulmonary embolism. This I have not included among the thromboses of the extremities.

So large a proportion of thromboses involving both lower extremities merits emphasis as a characteristic of chlorotic thrombosis. So again the repeated observations of multiple and successive thromboses, relapses and recurrent attacks (it may be after weeks or after years), all point to the peculiar and widespread tendency to thrombosis in some cases of chlorosis. The most remarkable example of this is Huels' case, in which various large veins of the extremities, trunk, and neck

became thrombosed in quick succession, until finally only the jugular and right subclavian veins remained free. The patient recovered. In 5 cases examined after death the inferior vena cava was plugged; and in a few of those who recovered the symptoms indicated extension of the thrombus from the iliacs into this vein.

Although the prognosis of chlorotic sinus-thrombosis is extremely bad, Bristowe and T. Buzzard each report an instance of recovery. Such a possibility has been questioned, but I see no reason to doubt it. Not very infrequently after death one or more of the intracranial sinuses are found to contain thrombi which had not occasioned any recognisable symptoms during life, nor any lesions of the brain.

A fatal issue of uncomplicated thrombosis of the extremities is due almost always to pulmonary embolism, which occurs oftenest in the second to the fourth week after the onset, and usually after some movement of the body. In my collection of cases there are thirteen instances of pulmonary embolism (25 per cent of the 52 cases with venous thrombosis outside of the cerebral sinuses). All but two terminated fatally. In some other cases there were symptoms suggestive of embolism; and doubtless emboli lodged in smaller pulmonary arteries without giving any indication of their presence. After making due allowance for the undoubtedly disproportionate representation of embolism of the large pulmonary arteries in published records, this catastrophe remains sufficiently frequent to impart a certain gravity to the prognosis even of simple femoral thrombosis in chlorosis.

There are almost as many hypotheses of chlorotic thrombosis as of chlorosis itself. None of these introduces any factors which have not been considered already under etiology. The principal causes which have been assigned, either singly or in combination, may be grouped as follows : (i.) feeble circulation due to weakness of the heart, sometimes intensified by congenital hypoplasia of the blood-vessels (Virchow); (ii.) alteration of the vascular endothelium, especially fatty degeneration (Eichhorst, Renaut); (iii.) primary phlebitis of unknown causation (Vaquez); (iv.) increase of platelets (Hanot and Mathieu, Buttersack); (v.) some fault in the composition of the blood, variously defined as lowered specific gravity, deficiency of salts (?) (Renaut), presence of extractives derived from muscular activity (Proby), increase of fibrin-ferment (Birch-Hirschfeld); (vi.) secondary infection (Villard, Rendu, Oettinger, von Noorden).

It is not necessary here to discuss all these views in detail. The data for estimating their value have for the most part already been presented in this article. Such primary lesions of the vascular wall as have been noted in the thrombosed veins have usually been trivial, and are common enough without thrombosis. There is at present no bacteriological basis for the infective supposition. Villard's much-quoted observation is unconvincing; in his case a small piece of a peripheral thrombosed vein was excised and examined by Nepveu for micro-organisms with a negative result. Villard adds that Bossano found

micro-organisms in the blood, but gives no details ; and there is no evidence that these micro-organisms may not have come from the skin. Perhaps more weight should be attached to a few observations in which some source of infection, such as furuncle, was present. Proby, Löwenberg, von Noorden, and other observers have examined the thrombi and blood of chlorotics without finding any micro-organisms. Nevertheless von Noorden and others are favourably disposed to the infective hypothesis, on clinical grounds. Sometimes the onset of chlorotic thrombosis is ushered in by a chill or chilly sensations ; usually there is fever, which may be well marked ; and in general the symptoms are thought by some to indicate infection. It does not seem to me imperative to interpret these symptoms as necessarily indicative of infection by micro-organisms.

There are difficulties with all of the hypotheses which have been suggested. I think that there may be some significance for the etiology of chlorotic thrombosis in the increase of platelets. Another element which may enter into the causation is some little understood irregularity of the circulation, other than retarded flow, which is manifested in the venous thrills and hums ; and which may in certain situations, where thrombi most frequently form (sinuses, femoral vein), lead to the eddies shewn by von Recklinghausen to be of importance in the causation of thrombosis ; although I confess that the fulness of the veins in chlorosis does not support this suggestion.

Gout.—Since the publication of the classical paper on gouty phlebitis by Paget in 1866, followed by those of Prescott Hewett and Tuckwell, this affection has been well recognised (see art. on "Gout," Vol. III. p. 153). Its causation is unknown. Paget with much reason regards the ailment as a primary phlebitis with secondary thrombosis ; and in this he has been followed by most writers on the subject. Although deposition of urates has been found in the sheaths of veins, there is no evidence that gouty phlebitis is caused in this way. Sir W. Roberts, on p. 136 of the article just quoted, ingeniously suggested that the presence of scattered crystals of sodium biurate in the blood may constitute foci around which thrombi may be formed. Blumer points out that there are two forms of thrombophlebitis in connexion with gout : (*a*) occurring during an attack of gout ; (*b*) in persons supposed to be gouty ; and that the latter group must be considered in the relation with idiopathic thrombosis.

Idiopathic Thrombosis.—Paget says that the occurrence of phlebitis in elderly persons without any evident external cause warrants the suspicion of gout ; and that this is perhaps the most common form of idiopathic phlebitis. There remain, however, rare instances of apparently spontaneous thrombophlebitis, occurring in previously healthy individuals, which cannot be explained in this way. Daguillon has observed and collected a number of such cases. Briggs has described a group of cases of idiopathic recurrent thrombophlebitis, and suggests that the causal factor is phlebosclerosis. Possibly the liability to this affection may, like arteriosclerosis, be hereditary in some instances.

Primary Infective Thrombosis.—There are rare instances of arterial and

venous thrombosis, generally widespread, which present the characters of an acute infective disease without anatomical lesions other than the thrombophlebitis, or thrombo-arteritis, and the changes consecutive to the vascular obstruction and to the vascular or general infection. The thrombosis may be referable to a primary infective angiitis, or to a general infection with changes in the blood and circulatory disturbances. The former class of cases may be considered analogous to mycotic endocarditis, the localisation being in the vascular intima instead of in the endocardium. In the latter group, which probably is not strictly separable from the former, the veins or the arteries are plugged with thrombi, which are often extensive and multiple. The venous is more common than the arterial form. Vessels both of the extremities and of the viscera may be invaded. The affection appears as an acute infective fever with the special localisation of the process in the blood-vessels.

As belonging to the group of primary infective thrombophlebitides I should interpret a case reported by Dowse. A woman, forty-three years old, previously in good health, was suddenly seized with chills, fever, and great prostration, accompanied by the rapid onset of severe pain and oedematous swelling of the right leg. Death occurred after two and a half weeks. At the necropsy the iliac, femoral, popliteal, and deeper veins were found to be filled with mixed, adherent, predominantly red thrombi. The tissues around the thrombosed vessels were suffused with blood.

Prof. Osler reported an instance of the arterial form of primary infective thrombosis. A man, aged twenty, who had recovered from typhoid fever two years previously, presented fever, rapid pulse, diarrhoea, and abdominal pain, followed by gangrene of both legs extending to the middle of the thighs. He died about two weeks from the beginning of the illness. At the necropsy was found thrombosis of the femoral and iliac arteries, of the lower two inches of the abdominal aorta, and of two large branches of the splenic artery. The spleen was enlarged, and contained large infarcts, one the size of an orange, which had given rise to peritonitis. There were infarcts also in the right kidney. Numerous micrococci were found in the splenic infarct, and in the exudate covering it. The heart, the intestine, the brain, and the lungs shewed no lesions.

Post-operative Thrombosis.—Cordier has collected 232 cases of post-operative thrombophlebitis; of these 213 were on the left side, either in the femoral or saphena vein.

Pregnancy.—Although this is not a morbid condition, a brief reference may be made here to the occurrence of thrombosis during pregnancy as apart from the puerperium. Although it is not rare, Goldsborough was not able to collect more than 11 cases of thrombosis of the inferior extremities. It may occur on both sides, and when unilateral is more often seen on the left side. In his case the thrombosis was thought to be connected with the pressure exerted by a corset which the patient had worn to conceal her condition.

EFFECTS AND SYMPTOMS.—The lesions and the symptoms produced by thrombi are referable to the obstruction of the circulation caused by the plug, and to the local and constitutional effects of irritative or toxic substances which may be present in the thrombus or vascular wall. It is obvious that these effects must vary with the functional importance of the part supplied by the obstructed vessel; with the rapidity, extent, and completeness of the obstruction; with the location of the plug in heart, artery, capillary, or vein; with the size of the vessel; with the readiness of establishment of a collateral circulation; with the nature of the thrombus, and with associated local and general morbid conditions. Thus the obstruction of each important vessel produces its own anatomical and clinical picture. The thromboses of certain vessels, as the intracranial sinuses, the portal vein, the femoral vein, are well characterised and distinct affections, which receive separate consideration in medical books. But I know of no modern work which presents in a systematic and thorough way the anatomical and clinical characters of occlusion of each of the important vessels of the body; although scattered through medical literature is a large and to a considerable extent unutilised casuistic material for such monographic treatment. In this article, treating of the subject as a whole, the more general considerations concerning the effects of thrombosis, with special reference to certain common and clinically important localisations which do not receive separate treatment elsewhere in this work, will be presented. Widely different are the effects according as the thrombosis is cardiac, arterial, capillary, or venous.

Of Cardiac Thrombosis.—If the presence of globular cardiac thrombi could be determined during life, it would be generally recognised as an index of grave impairment of the heart's action. But, apart from furnishing emboli, ordinary globular thrombi are not known to occasion any symptoms. There may be instances in which during life cardiac thrombi may be suspected as more probable sources of emboli, particularly of those causing pulmonary infarction, rather than either endocardial vegetations or venous or arterial thrombi; but beyond conjecture the diagnosis can hardly go. Gerhardt attributed to the pressure of thrombosed auricular appendices upon the pulmonary artery or aorta murmurs heard over the arterial orifices of the heart; but other causes of such murmurs are commoner and better recognised. The encroachment of massive thrombi and of pedunculated polyps upon the orifices of the heart may occasion murmurs, thrills, and symptoms indistinguishable from those of valvular disease. In three such cases, involving the mitral orifice, von Ziemssen observed gangrene of the feet, which he was inclined to refer to arterial thrombosis rather than to embolism; but this symptom has not the diagnostic value which he assigns to it, for in other cases it was present only exceptionally, and it may occur in ordinary mitral stenosis. Unless the orifices are encroached upon, the mere presence even of large thrombi usually occasions little or no disturbance of the heart, or none which can be distinguished from that of associated

valvular or mural disease.   The clinical features of ball-thrombi have already been considered (p. 724).

Of Arterial Thrombosis.—The effects of arterial thrombosis are so much like those of embolism that it will be convenient to defer the detailed consideration of their manifestations in common to the article on Embolism (p. 770), and here to speak only of the more distinctive features and clinical types of arterial thrombosis.

Whether the occlusion of an artery be by a thrombus or an embolus, the result, apart from possibly infective properties of the plug, depends upon the possibility of establishment of an adequate collateral circulation.   If the anastomoses are such as to permit the ready development of a collateral circulation, an arterial branch may be plugged without any mechanical effects.   In the case of certain visceral arteries, as the terminal cerebral, branches of the splenic, and of the renal, a collateral circulation sufficient to nourish the part supplied by the occluded artery cannot be established, even with a slowly-forming thrombus.   In some situations, however, arteries whose abrupt obstruction by an embolus may cause the gravest lesions and symptoms, may be closed gradually by thrombosis without serious consequences.   This has been observed in thrombosis of various arteries of the extremities, neck, and trunk, such as the femoral, the iliac, the carotid, the mesenteric, the coeliac axis, a main division of the pulmonary artery, and even the aorta.   But in order to secure whatsoever advantage may accrue from its slower formation, the thrombus must find other conditions favourable for the development of a collateral circulation ; and often enough these conditions, of which the most important are integrity of the arterial walls and vigour of the general circulation, are absent.   Furthermore, thrombosis is often rapid in attack, and hence, whether the plug be a thrombus or an embolus, the result is frequently the same.

In the differential diagnosis between arterial thrombosis and embolism emphasis is properly laid in the former upon the more gradual appearance of the symptoms of vascular occlusion and pre-existing arterial disease, and upon sudden onset and the detection of some source for an embolus, particularly cardiac disease, in the latter (see Diagnosis of Embolism, p. 788).   But mistakes in diagnosis are sometimes unavoidable ; for all the clinical phenomena which attend the one may occasionally be associated with the other form of arterial obstruction.   Nor can the distinction always be made, with the desired precision, at the necropsy, although generally this is decisive.   Hence cases are reported as arterial thrombosis which are doubtless embolism, and conversely.

Within recent years primary arterial thrombosis, occurring independently of chronic diseases of the arteries, has been recognised as a more frequent and important affection than had been generally supposed since the acceptance of Virchow's doctrine of embolism.   Of especial medical interest are the primary arterial thromboses, arising oftener as a sequel during convalescence than as an accompaniment of various infective diseases, particularly of enteric fever and influenza.   The

associations and localisation of these thromboses, as well as the prevailing view that they are infective and referable to an acute arteritis, have already been considered.

Arterial Thrombosis of the Extremities.—When, as is usual, arteries of the lower extremities are affected, the first symptom is pain in the limb. This is often severe and paroxysmal, and is increased by pressure at certain points in the course of the vessel. The obliterated artery may be felt as a hard, sensitive, pulseless cord ; and below it pulsation may be feeble or cease altogether. Before obliteration the pulsations may be of wider amplitude than normal, in consequence of lack of arterial tone (Gendrin, Barié). The leg, especially about the foot and ankle, becomes pale, cold, mottled with blush-red spots, numb, and paretic. With loss of tactile sensation there is often increased sensitiveness to painful impressions. There may be diminution or loss of muscular reaction to both galvanic and faradic currents. There may be increased moisture of the skin, and some oedematous swelling of the affected leg. Unless adequate collateral circulation be speedily developed the termination is gangrene. While the extent of the gangrene is in relation to the seat of the obstruction, it varies also according to the collateral circulation ; so that with occlusion of the femoral or iliacs it may affect only the foot or even a toe ; or with closure of the popliteal or tibial arteries it may extend as high as the point of obstruction. The gangrene is usually dry ; but if septic inflammation or closure of the veins occurs it is likely to be moist. Recovery may follow with loss of the gangrenous part ; or death may result from exhaustion, from extension of the mortification, from septicaemia and toxaemia.

The rarer arterial thrombosis of the upper extremities may likewise lead to gangrene ; but here the chances for restoration of the circulation through the collaterals are much better.

I have already referred to the relations of thrombosis to senile, spontaneous, and other forms of gangrene (p. 714). Heidenhain and Naunyn hold that arteriosclerotic thrombosis is the usual cause of diabetic gangrene ; but further investigations into the causes of this form of gangrene are needed. Thrombosis of the abdominal aorta presents a group of symptoms which will be described under Embolism (p. 809).

The complex of symptoms called by Charcot "intermittent claudication" may be observed with thrombosis of arteries of the lower extremities, or of the iliacs or abdominal aorta ; but it is more common with arteriosclerosis. The term "intermittent claudication" (boiterie) is used by French veterinarians to describe similar symptoms in horses affected with thrombosis of the iliac arteries, which is not a rare disease in these animals. In these cases the lower extremities receive enough blood for their needs during repose, but not during active exercise. The slighter manifestations consist only in some muscular weakness and numbness of the legs after exercise ; but in more severe cases, after walking a quarter of an hour or perhaps less, occur great muscular weakness, numbness, and pains and cramps in the legs, which may become

cold, exsanguinated, sometimes cyanosed in the periphery, and almost
pulseless.   All of these symptoms disappear after repose, perhaps of but
a few minutes' duration.   Charcot's syndrome has in a number of reported
cases been a precursor of arteriosclerotic gangrene, but it may exist for
years without this event.   The phenomena are unilateral or bilateral,
according to the seat of the arterial obstruction.   Spasm of the arteries
is evidently an important element in the pathogeny of intermittent
claudication (*vide* p. 611).

Other evidences of inadequate collateral circulation with arterial
thrombosis of the extremities may be muscular atrophy and so-called
trophic disturbances, which are generally the result of trauma or of some
infection in the member whose natural resistance is lowered by the
imperfect blood-supply.

Thrombosis of the visceral arteries may produce lesions and symptoms
identical with those following embolism, such as sudden death from
thrombosis of the pulmonary artery, of the coronaries of the heart, or of
the basilar ; ischaemic cerebral softening, and infarctions of the lungs,
heart, spleen, kidneys, retina, and intestine, with their attendant
symptoms.

Thrombosis of the Pulmonary Artery.—It is especially to be noted that
thrombosis of the pulmonary artery, both in its principal divisions and
in the smaller branches, is often entirely latent, both as regards resulting
lesions in the lungs and the symptoms.   Thrombosis of the main trunk
or primary branches may, however, produce sudden or rapid death ; or
a subacute or chronic affection characterised by dyspnoea, cyanosis,
haemoptoic infarctions and incompetency of the heart, as in the cases
reported by Blachez and by M'Phedran and Mackenzie.

Dr. Newton Pitt believes that thrombosis of the pulmonary arteries
is far more frequent than is generally supposed, even going so far as to
say "that thrombosis in the pulmonary artery, so far from being very
rare, possibly occurs more frequently than in any other vein or artery
in the body."   This opinion is based partly upon failure to find a source
for an embolus in the right heart or systemic veins, and partly upon
absence of folding, fracture, or other appearances of the plug suggestive
of an embolus, as well as upon association with general conditions known
to dispose to thrombosis.   A similar remonstrance against the current
interpretation of so many plugs in the pulmonary arteries as embolic in
origin was made by Bristowe in 1869.   In my experience sclerosis and
fatty degeneration of the intima of the pulmonary vessels is not par-
ticularly uncommon ; and I also believe that primary thrombosis of the
pulmonary arteries, particularly of medium-sized and smaller branches,
is more frequent than is usually represented in textbooks.   Dr. Box has
brought forward seven cases to shew that a thrombus in the main trunk
of the pulmonary artery may become detached and form a riding embolus
occluding the two main branches.   Still, for reasons to be considered
under Embolism (p. 798), the evidence seems to me in favour of the
usually accepted opinion that the majority of plugs found in the pul-

monary artery and its main divisions in cases of sudden death are emboli.

Thrombosis of the mesenteric arteries will be considered with embolism of these arteries (p. 804).

Thrombosis of the coronary arteries has been described on p. 119.

Thrombosis of the renal arteries is rare. Halperin described a case with thrombosis, due to endarteritis obliterans, and a large infarct, and refers to v. Recklinghausen's case of thrombosis of the renal artery following injury. Widespread thrombosis of the cortical arteries in the kidney has been recorded in three almost exactly similar cases in women shortly after delivery (Bradford and Lawrence, Herringham and Griffith, Lloyd). The cortical areas of both kidneys were necrosed, and the symptoms were the same as those of calculous anuria; there were no symptoms of uraemia. Dr. Parkes Weber, however, suggests that the arterial thrombosis was secondary to, and not the cause of, the acute parenchymatous changes.

Thrombosis of the cerebral vessels will be described in Volume VIII.

Here may be mentioned the interesting observations of recent years concerning the dependence of certain diseases of the spinal cord upon affections of the blood-vessels of the cord, arterial thrombosis being an especially important factor in many of these cases.

Capillary Thrombosis.—In consequence of the abundant anastomoses, it is only when all or nearly all of the capillaries of a part are thrombosed that any mechanical effects result. Experimentally, Pearce has shewn that hyaline thrombi, induced by the injection of haemagglutinins, give rise to necroses in the liver. In swine infected with the hog-cholera bacillus I found thrombosis of the renal capillaries chiefly of the glomeruli; in extreme cases there was complete anuria. Although in many cases I have seen similar hyaline thromboses in human kidneys, they were never so extensive as to seem likely to cause recognisable symptoms. Several years ago I drew attention to the presence of hyaline thrombi in capillaries and arterioles in the walls of some fresh gastric ulcers, and since then I have been able to repeat the observation in three or four instances.

Effects of Venous Thrombosis.—Thrombosis is so pre-eminently an affection of veins that chapters in textbooks treating of the general subject usually pay scant attention to its occurrence in other parts of the circulatory system. In the veins thrombosis occupies the field of intravascular plugging almost alone, for it is only in the portal system, and in the rare instances of retrograde transport, that embolism enters into consideration; such extraordinary occurrences as embolism of the azygos vein, resulting from thrombosis of the inferior vena cava, reported by Löschner, being mere pathological curiosities.

The direct effects of venous thrombosis, as of arterial, are referable to the mechanical obstacle to the circulation and to the properties of the thrombus. The mechanical effects result from inadequacy of the collateral circulation. The free venous anastomoses in many parts of

the body prevent any disturbance of the circulation as a result of venous occlusion by simple or benign thrombi. Such innocuous thromboses are particularly common in the pelvic veins. In some situations veins, whose rapid occlusion may cause serious lesions and symptoms, may be slowly plugged by a thrombus without manifest harm. For example, it is not uncommon to find at necropsy the main trunks of the renal veins completely thrombosed, without consequent alteration of the kidney or corresponding symptoms during life ; although we know that ligation of these veins causes haemorrhagic infarction of the kidney with albuminous, bloody urine.

Frequently, however, the contrast between the effects of ligation and those of thrombosis of veins is in the other direction ; the thrombosis being followed by venous congestion, and the ligation of the same veins being without evident disturbance of the circulation. The latter difference is not always easy to explain ; but the factors to which we can often appeal with more or less success, in attempting to account for the absence of sufficient collateral circulation with venous thrombosis, are the extent of the occlusion, general debility, feebleness of the circulation in consequence of coexistent anaemia, infection, cachexia or constitutional disorder, generally high venous pressure and low arterial pressure, lack of muscular movement and perhaps of other subsidiary forces aiding venous circulation, phlebosclerosis, inflammation or some less evident affection of blood-vessels called upon for extra work, and irritative or toxic properties of the thrombus. The importance of these, and perhaps other accessory conditions, in explaining the passive congestion of many venous thromboses in human beings is made evident, not only by the inability to produce similar effects experimentally by correspondingly slight or moderate degrees of venous obstruction, but also by the varying effects of thrombotic processes with the same localisation and extent in different persons and under different conditions. Thus femoral thrombosis may be attended by absolutely no oedema or passive congestion, or may occasion extreme degrees of oedema and venous engorgement.

The consequence of the passive hyperaemia caused by venous thrombosis is local dropsy. This constitutes the characteristic symptom of uncompensated venous obstruction by a thrombus, as local necrosis does that of uncompensated arterial thrombosis. In addition to the oedema, there may be diapedesis of red corpuscles, but this occurs to a perceptible degree only when the obstruction to the venous flow is extreme, or the capillaries unusually permeable. Such haemorrhages are very rare in peripheral venous thrombosis, but are common with thrombosis of the portal and mesenteric veins, the cerebral veins and sinuses, the splenic, the retinal, and some other visceral veins. Actual necrosis may likewise result from thrombosis of the mesenteric, cerebral, and splenic veins ; but, if it occurs at all with thrombosis of veins of the extremities it is extraordinarily rare, and probably due to complications.

In addition to these effects, due directly to the blocking of the venous

circulation, even so-called benign or simple thromboses often set up an acute inflammation in the venous wall and surrounding part; or, as already explained, this inflammation may antedate the thrombosis. These chemical, as distinguished from mechanical, effects consist chiefly in arterial hyperaemia, inflammatory oedema, pain, implication of nerves, and constitutional symptoms, such as chills, fever, and quickened pulse. The occurrence of these irritative or toxic effects, even with the so-called marantic thromboses, is an argument (in addition to those already considered) in favour of the infective nature of many of these plugs, and of their primarily phlebitic origin. But while undoubtedly significant of such an interpretation, it can hardly be considered conclusive; for it is possible that certain thrombi may possess irritative properties not attributable to the presence of micro-organisms or their products, and that the phlebitis, as well as the periphlebitis, may be secondary. However this may be, the old distinction between benign and infective thrombi no longer appears so sharply marked as was once supposed.

In rare instances the venous medical thromboses associated with anaemic, infective, cachectic, and constitutional diseases are plainly septic, and give rise to phlegmons, and perhaps pyaemia or septicaemia. The suppurative or septic thrombophlebitis, which with its attendant pyaemia was in pre-antiseptic days such a common and formidable wound complication, belongs to the surgeon's domain, or, in puerperal sepsis, to the obstetrician's. (See art. "Pyaemia," Vol. I.) To the borderland of medicine and surgery belong certain septic thrombophlebitides of visceral veins, of which the most important medical group, those of the portal system, has been considered in the article "Diseases of the Blood-vessels of the Liver" (Vol. IV. Part I. p. 141). Thrombosis of the umbilical vessels, which may occur either before or after birth, may be either simple or septic. The latter is an important affection, the consideration of which belongs to treatises on diseases of infants.

There is perhaps no pathological phenomenon which, on the face of it, appears simpler of explanation than the local oedema, consequent upon venous obstruction, but which, the more it is investigated, turns out to be, or at least is made to appear to be, more complicated. The explanation which has often been given is that the oedema is due simply to increased filtration of serum from the blood, in consequence of the rise of intravenous and intracapillary pressure resulting from the obstruction to the venous circulation. It is certain that this simple explanation does not suffice, at any rate for most venous thromboses, and that factors other than the mere rise of blood-pressure in the veins and capillaries are concerned; but as to the nature of these other factors there is great difference of opinion. The whole problem is wrapped up with that of the hypotheses of lymph-formation and lymph-absorption, which has been discussed in the article on "Dropsy" (Vol. IV. Part I. p. 510). Corresponding to the two classes of these hypotheses, we have mechanical hypotheses and vital or secretory hypotheses of the oedema of passive congestion. The mechanical explanations are at least easier of compre-

hension.   Cohnheim attributed this form of oedema to increased venous and capillary pressure, combined with increased permeability of the capillary wall due to malnutrition.[1]   Starling and Cohnstein advocated a similar explanation.   Dr. C. Bolton, from experimental obstruction of the superior and inferior venae cavae, concludes that the intracapillary blood-pressure is not increased when oedema occurs, and that the essential factor in the production of oedema is not increased pressure, but the alteration of the vessel-wall consequent on stagnation of blood and privation of oxygen.   With these conclusions Prof. Starling is in accord.

The secretory hypothesis of oedema has been advocated by Hamburger and by Dr. Lazarus-Barlow, who finds that all the physical explanations of the oedema of passive congestion are inadequate ; and, upon the basis of interesting experiments, he concludes that a principal factor is increased secretion of lymph by the capillaries incited by starvation of the tissues and accumulation of waste metabolic products.

Doubtless several factors, although not all necessarily operative in the same case, are concerned in the causation of the oedema of venous thrombosis.   Those which seem to me most apparent are the following : (i.) increased intravenous and intracapillary pressure, with consequent increased transudation of serum (not alone sufficient, for tying the femoral vein or inferior vena cava generally causes no oedema) ; (ii.) increased permeability of the capillary walls, which may be due to various causes, such as stretching from larger content of blood, starvation and asphyxia of capillary endothelium from lack of fresh supply of nutriment and oxygen, and injury from abnormal composition of blood in anaemic, infective, cachectic, and constitutional disease, or from inflammatory irritants ; (iii.) diminished absorption of lymph in consequence of lack of muscular movement, of imbibition of the capillary walls with fluid, and especially of retarded capillary and venous flow ; (iv.) arterial dilatation from irritative or inflammatory influences emanating from adjacent thrombosed veins, probably also from the asphyxiated tissues, and acting either directly upon the arterial wall, or directly upon vasomotor nerves, or reflexly (here the conditions resemble those in Ranvier's well-known experiment of tying the inferior vena cava or femoral vein, and producing vasomotor paralysis by section of the sciatic nerve) ; (v.) sometimes a watery condition of the blood rendering it easier of filtration.   Experiments of Dr. Lazarus-Barlow indicate that changes in the chemical composition of the tissues and tissue-fluids are also a factor in the production of the oedema.   To these changes, as the cause of alterations in osmotic pressure, Loeb assigns the chief importance in the production of oedema. The influence of hydrostatic pressure is evident from the greater frequency

---

[1] Cohnheim is sometimes quoted as considering increased pressure a sufficient explanation of mechanical oedema, although in his *Allgemeine Pathologie*, Bd. i. S. 494, he expressly recognises as an additional factor " unknown influences on the part of the living vessel-wall." As I had opportunity, when working in his laboratory on a problem concerning oedema, to become familiar with his views on this subject, I may be permitted to say that he often spoke of increased permeability of the capillary wall as an essential factor in the explanation of the oedema of passive congestion.

of oedema with thrombosis of the lower than of the upper extremities, and from the effect of position upon the amount of the oedema. Whilst these various factors can be conceived as essentially physical and chemical in their action, the living capillary wall upon which they act, either directly or indirectly, is to be thought of as something different from a dead animal or artificial membrane.

The oedema of phlegmasia alba dolens is by no means all due to venous congestion. Much, sometimes most of it, is an inflammatory oedema spreading from the thrombosed veins. This is evident partly from the hard, brawny, painful, at times warm character of the swelling (oedema calidum) ; and partly from its location in the part of the extremity nearest the affected veins. The oedematous swelling may begin above and extend downwards, instead of in the usual direction from below upwards. The hydrarthrosis often associated in moderate degree with phlegmasia is probably also referable to an inflammatory serous exudate rather than to passive transudation from venous obstruction. It occurs especially in the knee-joint.

Thrombosis of Veins of the Extremities.—Clinically the most familiar form of venous thrombosis is that of the extremities ; the lower much oftener than the upper. Its various sites and clinical associations have already been considered (pp. 718 and 727). The affection may be entirely latent ; or may be recognised by a slight or moderate unilateral oedema without general or other local symptoms ; or may be in the form of well-marked phlegmasia alba dolens ; or rarely may assume a severely infective character, with chills and high fever ; or, exceptionally, may lead to phlegmon and pyaemia or septicaemia. There is every transition between the extremes. The latent and milder types occur especially with tuberculosis, cancer, and other cachexiae ; the more severe manifestations with phlebitis of the puerperium, infective diseases, and chlorosis ; but there are many exceptions to this rule.

In the more acute and well-characterised cases the general symptoms are chiefly manifest at the onset ; and consist in moderate elevation of temperature, rarely preceded by a distinct chill, oftener by chilly sensations and quickened pulse. Increased frequency of the pulse may antedate the rise in temperature, and the pulse may remain rapid after the temperature falls. This disproportion between pulse and temperature is of diagnostic value (Mahler, Wyder, Singer), but it is not always present. Singer has made a careful study of the pulse-curve in puerperal thrombosis. A step-like acceleration of the pulse-curve often precedes other manifestations of thrombosis by several days. These general symptoms of the initial stage, which may persist for days, are often overlooked ; or they are masked by an existing febrile disorder. They are probably present in some degree, even in mild cases, oftener than the clinical records shew.

The characteristic symptoms are the local ones in the affected leg. Pain, often paroxysmal, is usually the first to attract attention ; but sometimes it is the oedema. The pain may be severe. It is more or less

generalised, with especial tenderness in the groin, the inside of the thigh, the popliteal space, and the calf. Often it is first noted and may remain localised in the calf; as is true of the oedema also. There may be sensations of numbness or of "pins and needles." The cardinal symptom, oedema, sometimes descending sometimes ascending, gives rise to the firm, painful swelling of the limb, covered with tense, shiny, smooth, white or mottled skin, marked often by dilated veins, whence comes the name milk-leg or white leg. The oedema in typical phlegmasia alba dolens is hard and elastic, pitting but little on pressure. Occasionally the skin has a more livid, cyanotic hue, or it may be of a brighter red. In the more acute cases the surface temperature is elevated; in others it is often lowered. Muscular movements are naturally restrained, and it is said there may be actual paresis. The thrombosed vein, if accessible to palpation, can often be felt as a hard, tender cord; but it is best not to attempt to gain this information, which in most cases is of little practical importance. The sensation obtained from palpating the vein may be misleading in consequence of the periphlebitis, or of the soft character of the thrombus. Certainly, in view of the manifest danger of detaching an embolus, only the gentlest manipulations are permissible. If the thrombosed vein be superficial, it may sometimes be seen as a line of livid redness beneath the skin. It is not always tender on palpation.

The great and usually the only danger from peripheral thrombosis is fatal pulmonary embolism. It occurs oftenest between the second and fourth weeks, but may occur earlier or later. The danger may be considered to be past at the end of six weeks, if the local symptoms have subsided; although there are exceptional instances of pulmonary embolism at a later period. It is to be noted that pulmonary embolism may result from latent and mild forms of venous thrombosis as well as from those of the well-marked examples; it is, however, rare with the cachectic thromboses of tuberculosis and cancer. Small pulmonary emboli usually cause no lesions or symptoms, yet they may give rise to haemorrhagic infarction, pleurisy, or embolic pneumonia.

Nervous phenomena are sometimes so prominent as to have led to the recognition of a neuralgic type of phlebitis (Graves, Trousseau, Quénu). There may be even a mild peripheral neuritis associated with the venous thrombosis. This is probably caused by the direct action of inflammatory irritants spreading from the inflamed veins; but it has also been attributed to thrombosis of small veins in the nerve-trunks, to the bathing of the nerves in the oedematous fluid, and to reflex irritation. In thromboangiitis obliterans, in which the veins may also be similarly affected, the clinical picture may closely resemble Raynaud's disease and erythromelagia (Buerger). Occasional sequels of femoral thrombosis, for the most part very rare, are varicose veins, leg ulcers, persistent chronic oedema, elephantiasis, muscular hypertrophy, muscular atrophy, and clubfoot. Small phleboliths in the superficial veins of the shin may resemble subcutaneous rheumatic nodules (Rolleston).

There has been much discussion on the possibility of gangrene being caused by thrombosis of the femoral or iliac veins. Cases have been reported in which no other cause of the gangrene was found than venous thrombosis; but with peripheral venous thrombosis this is such an exceptional occurrence that it seems clear that, when gangrene results, complicating factors—such as arterial disease, pressure upon arteries, arterial spasm, great feebleness of the circulation or septic inflammation—must be associated with venous thrombosis. It is true that surgeons are familiar with gangrene after ligation of the femoral vein, but here also the result is exceptional and attributable to some complication. Braune, upon anatomical grounds, attempted to demonstrate that gangrene is to be expected after closure of the femoral vein near Poupart's ligament, but the clinical evidence does not support this view. Galliard has reported a case and has collected from the records others in which gangrene had followed venous without arterial thrombosis.

The thromboses of the upper extremities are usually of shorter duration and milder type than those of the lower; unless referable to some persistent cause, such as the pressure of a tumour. They are often accompanied by some cervical oedema.

Thrombosis of the Inferior Vena Cava. — Since the days of Richard Lower occlusion of the inferior vena cava has been the subject of much experimental and clinical study. There are reports of at least 140 cases of this affection in human beings. The principal records are cited in the monographs of Vimont, Thomas, and Krause, although the bibliography is by no means complete. The causes may be tabulated as follows :—(i.) Pressure from without by a tumour; enlarged glands due to malignant disease, tuberculosis, or other causes; an aneurysm. (ii.) Invasion of the wall of the vein by malignant disease; a primary endothelioma of the vein giving rise to thrombosis has been recorded (Unruh). (iii.) Disease of the wall of the vein, such as infective phlebitis, syphilitic phlebitis, or tuberculous endophlebitis (Griffon). (iv.) Extension of a thrombus from the pelvic or femoral veins through the iliac veins. (v.) Extension of new growth along the renal or suprarenal veins in primary malignant disease of those organs. (vi.) Autochthonous thrombosis is rare. (vii.) Syphilitic disease of the liver implicating the inferior vena cava (Bosanquet, MacCallum). (viii.) Congenital cases, due to errors of development. It may occur without any symptoms or without symptoms suggestive of the diagnosis, especially when the thrombosis is partial and does not occlude the vessel. The characteristic symptoms are oedema of both lower extremities and of the abdominal walls, and the development of a typical collateral circulation. When the renal veins are likewise occluded there may be albuminous, bloody urine; but with thrombosis of these veins this symptom is oftener lacking than present. The diagnosis rests especially upon the appearance of dilated anastomosing veins coursing upwards from the groins and flanks over the abdominal walls and lower part of the thorax. These tortuous, varicose veins, sometimes as big as the little finger, make a very striking and characteristic picture. The

superficial veins concerned in carrying on the collateral circulation are the inferior and superior superficial epigastric, the long thoracic, the superficial circumflex iliac, the external pudic, the lumbo-vertebral anastomotic trunk of Braune and numerous unnamed anastomotic veins. The direction of the circulation is of course from below upwards. In addition there is a deep collateral circulation through various visceral veins with dilatation of the azygos veins. Sometimes the circulation is almost wholly through the deep collaterals, and there may be little or no dilatation of the visible superficial veins. In fact, in not a few cases, by the absence of visible dilated collaterals, the diagnosis is rendered difficult or impossible. Schlesinger has collected 18 cases in which the oedema was in one leg only. This may be due to the previous establishment of a collateral circulation on one side from a former iliac thrombosis, or to unilateral iliac thrombosis with parietal thrombosis of the vena cava, or to congenital duplication of the vena cava. In a remarkable case the symptoms simulated those of a perforated gastric ulcer (Phillips).

Thrombosis of the Renal Veins.—This affection is fairly common. It may be an extension of a thrombotic process in the vena cava, or on the other hand the latter may be secondary to renal thrombosis. Marantic thrombosis of the renal veins is not unusual in infants with cerebral symptoms, or exhausted by diarrhoea. In adults thrombosis of the renal veins is observed not very infrequently in chronic Bright's disease, particularly the waxy kidney ; and in malignant tumour of the kidney. The renal veins rank among those predisposed to marantic thrombosis. I once made a necropsy on a case of primary genito-urinary tuberculosis in which a caseous mass had broken into a renal vein which contained an adherent greyish-red thrombus extending into the vena cava. Tubercle bacilli were present in the caseous mass and the thrombus. There was acute miliary tuberculosis. The lesions and symptoms which one would expect to find with thrombosis of the main trunk of the renal vein are oftener absent than present. The various collateral veins, communicating through the capsule and along the ureters with the lumbar, diaphragmatic, adrenal, spermatic, and other veins, suffice for adequate return flow. Still a number of cases have been observed with more or less haematuria and albuminuria which have been referred to thrombosis of one or both renal veins, and genuine haemorrhagic infarction may occur.

Thrombosis of the Mesenteric Veins.—Thrombosis of veins in the intestinal wall is often associated with ulcers and other morbid conditions in the intestine. The thrombus may extend into the small mesenteric veins, or the latter may be attacked independently. These small thrombi are important chiefly as a source of infective emboli transported to the liver.

Thrombosis of the large mesenteric veins is less frequent than embolism or thrombosis of the mesenteric arteries. In 1898 I collected 32 cases with pronounced symptoms, and a few cases without symptoms referable to the thrombus and without intestinal lesion. In 1904 Jackson, Porter, and Quinby collected 77 cases. The superior mesenteric vein was thrombosed much oftener than the inferior. In many cases with

symptoms, the thrombosis was ascending and secondary to inflammation, ulceration, or some other disease of the intestine ; it may also occur in strangulated hernia (Corner) and after surgical operation, such as herniotomy or splenectomy, or abdominal injury.   It may be associated with disease of the mesenteric glands (Bradford) or tuberculous peritonitis (Johnson).   In some instances it was descending from thrombosis of the portal or splenic vein ; in a few it was secondary to enteric fever or some marasmic or cachectic state ; and in one it was attributed to a calcareous plate adjacent to the vein.   The symptoms are the same as with occlusion of the mesenteric arteries, and are more fully described on p. 804.   Two groups of cases may be recognised : cases with an acute onset and course, and those with an insidious onset and chronic course.   In the acute group, which includes most of the cases, the symptoms are as follows : sudden onset of very intense, colicky, not definitely localised abdominal pain ; tender, distended, tympanitic abdomen due to intestinal paralysis ; vomiting, which may be bloody ; obstipation or bloody diarrhoea ; and rapid collapse with cold sweat and subnormal temperature.   The diagnosis is likely to be acute intestinal obstruction, and laparotomy to be performed.   Death generally occurs within two or three days.   At the necropsy are found haemorrhagic infarction and gangrene of the intestine, haemorrhages in the mesentery, bloody fluid in the peritoneal cavity, and sometimes, although not regularly, peritonitis.   The cases without symptoms have been usually thrombosis of slower formation, but this does not appear to have been always the case.

In a case reported by Dr. Rolleston, the superior mesenteric vein was filled with softened, canalised clot ; and in addition the inferior mesenteric vein, the internal and external iliac veins on both sides, and the splenic vein were completely thrombosed, and a partly occluding thrombus extended into the portal vein.   The thrombus in the superior mesenteric vein was regarded as the oldest.   There was old and recent inflammation of the intestine, but no intestinal infarction.

Of interest is the relation of thrombosis of the mesenteric veins to portal thrombosis.   In several instances of the latter thrombosis of the mesenteric veins occurred without haemorrhagic infarction of the intestine.   Doubtless the explanation is that a sufficient collateral circulation had been established after the portal thrombosis to prevent the usual effects of a subsequent mesenteric thrombosis.   That this, however, is not always the case is shewn by the sudden or more gradual termination of some instances of portal thrombosis with haemorrhagic infarction of the intestine, in consequence of the extension of the thrombus into the mesenteric veins.   This has occurred especially in the more acute cases of portal thrombosis, but it may occur also in those of several months' duration.   Acute portal thrombosis may cause haemorrhagic infarction of the intestine without mesenteric thrombosis ; or the infarction may be over a larger extent of intestine than corresponds to the thrombosed mesenteric veins.   On the other hand, the infarcted area

may be much smaller than that supplied by the thrombosed vein. The symptoms may be of slower development and of milder type when thrombosis of the mesenteric veins is secondary to portal thrombosis than when it is primary. The sequence of events in Fitz's case is interesting—globular thrombi in the left ventricle, embolism and infarction of the spleen, secondary thrombosis of the splenic vein, extension of the thrombus into the superior mesenteric vein, haemorrhagic infarction of the intestine terminating fatally. There was no obstruction in the mesenteric arteries.

Pylethrombosis.—*Vide* Vol. IV. Part I. p. 142.

Thrombosis of the Splenic Vein.—Primary thrombosis of the splenic vein and its radicles is rare. Autochthonous thrombosis may be secondary to calcification of the wall of the splenic vein. Thrombosis of veins within the spleen, extending sometimes into the main trunk, is common with infarction, abscess, and certain other morbid processes in this organ. Thrombosis of the main trunk may be caused by suppurative or haemorrhagic pancreatitis, or by cancer of the pancreas. As has already been mentioned, thrombi may extend from the portal or mesenteric veins into the splenic, as well as from the latter into the former. There is the possibility of thrombosis secondary to retrograde embolism of the splenic vein.

Köster has reported the rare complication of enteric fever with thrombosis of the radicles and main trunk of the splenic vein ; the evidence being conclusive that the oldest part of the thrombus was in the spleen. The evidences of occlusion of the main vein appeared at the beginning of convalescence. The spleen was enormously swollen and the pulp of a diffuse reddish-black colour. The capsule and surrounding tissues were suffused with blood. As there were thrombi in the small mesenteric veins near the ulcerated ileum, there was a possibility of retrograde embolism ; but Köster thinks it more probable that the process originated within the spleen.

Thrombosis limited to the extra-splenic part of the vein may be completely or nearly compensated by the collateral venous circulation, so that no changes or only a moderate passive congestion occur in the spleen.

Thrombi occupying intra-splenic veins may cause haemorrhagic infarction. Dr. Rolleston has observed two instances of anaemic infarcts of the spleen in association with thrombosis of the splenic vein. Litten probably goes too far in attributing most genuine haemorrhagic as distinguished from pale infarcts of the spleen, to venous thrombosis rather than to arterial embolism. Extensive necrosis and haemorrhagic infarction may be caused by torsion of the pedicle of a movable spleen.

In cases of thrombosis of the splenic vein in which life is not rapidly brought to a close, the clinical features may imitate chronic splenic anaemia of adults. The spleen is much enlarged, and there may be periodic gastro-intestinal haemorrhages (Langdon Brown, Dévé)—cases of this kind have been cited by those who combat the existence of chronic splenic anaemia as a definite disease (*vide* also Vol. V. p. 765).

Obliteration of the Superior Vena Cava.—Since the admirable studies by

Duchek (1854) and by Oulmont (1856) of the causes and symptoms of obliteration of the superior vena cava a considerable number of instances of this condition have been reported. In 1903 Prof. Osler recorded 3 cases and gave a tabular statement of 29 additional recorded cases collected by Hume; these with the cases recorded by Sir D. Duckworth and Dr. Garrod, and by Dr. Poynton, make up a total of 35 cases. They fall into two groups: (1) of thrombosis, due to disease inside the vein, such as simple phlebitis, propagation of a thrombus from the periphery (Duchek), and tuberculous endophlebitis, as in Banti's remarkable case, in which nearly the whole length of the superior vena cava was completely filled by a neoplastic tuberculous mass projecting into the right auricle. The outer walls of the vein were intact. The condition seems to have been analogous to the tuberculous cardiac thrombi already described (p. 721). Primary thrombosis of the superior vena cava is so rare as to be a pathological curiosity. Dr. Poynton has reported an instance of thrombotic occlusion of the upper two-thirds of the superior vena cava in association with chronic and acute valvular endocarditis, and in a second case of valvular disease he found a mural thrombus in this vein. In both cases there was tricuspid insufficiency. About a third of the cases fall into this category. (2) Most of the cases depend on causes outside the vein, such as the pressure exerted by tumours, enlarged glands, or aneurysms; or on mediastinitis or syphilis. The characteristic symptoms are oedema and cyanosis of the upper half of the body—face, neck, arms, and thorax—and dilatation of deep and superficial veins, especially marked over the anterior wall of the thorax and upper part of the abdomen. In one of Prof. Osler's cases, the anterior surface of the chest was covered with large, spongy bunches of enormous varicose veins, in one of which a phlebolith could be felt. Other symptoms, which may be present, are oedema of conjunctival and buccal mucous membranes, exophthalmos, watery secretion from the conjunctivae, nose-bleeding, and such signs of venous congestion of the brain as headache, vertigo, and ringing in the ears, especially on bending over. In the light of the whimsicalities of venous thrombosis it is hardly necessary to add that the symptoms may be less marked, and may deviate from what might naturally be expected. In one case of complete obliteration there was an absence of any swelling of the head and neck (Duckworth and Garrod).

Thrombosis of the Innominate, Subclavian, and Jugular Veins.—The more important literature of this subject is cited in the papers of Pohl, Hirschlaff, and Helen Baldwin. The occurrence of these thromboses in cardiac disease, and from compression, has already been mentioned (p. 735); other rare causes are infection, empyema, acute rheumatism, tuberculosis, marasmus, and trauma. The symptoms are the usual ones of venous congestion, oedematous swelling, pain in the regions from which the veins convey blood, dilatation of collaterals, and, in the case of the cervical veins, recognition of the thrombosed vein by palpation, which, however, should be done with great care.

Thrombosis of the pulmonary veins may be mentioned as a rare source of embolism in the aortic system. It is usually secondary to some pulmonary disease, as gangrene, malignant tumours, abscess, infarction, tuberculosis, pneumonia. It has been observed with extensive emphysema of the lungs (Schmale).

Thrombosis of the cerebral sinuses will be considered in connexion with diseases of the brain in a subsequent volume.

O. Wyss has described a remarkable instance of extensive haemorrhagic myelitis caused by widespread hyaline and platelet thrombi in veins within the spinal cord. The thrombosis was secondary to a glioma of the dorsal cord. Rosin has likewise observed thrombosis of veins extending the whole length of the spinal cord, consecutive to a tumour of the cervical cord.

Thrombosis of the corpora cavernosa penis has been described as a cause of persistent priapism (Weber), and has been thought to be of gouty origin (Duckworth).

Multiple Thromboses.—Finally may be mentioned the cases in which many veins in different parts of the body become thrombosed, as in Huels's case of chlorotic thrombosis ; Prof. Osler's, of thrombosis secondary to cancer of the stomach, already cited (p. 734) ; and cases of infective thrombophlebitis (p. 740). Erlenmeyer has described as "jumping thrombosis" (*springende Thrombose*), in distinction from the ordinary creeping form, cases in which the process attacks first one vein and then another, in a different region, until finally various veins in the extremities, trunk, and brain may become plugged. Buerger has found that migrating thrombophlebitis in the territory of the long saphenous vein is not uncommonly associated with the symptoms of thrombo-angiitis.

Treatment.—The treatment of thrombosis of the extremities is about all that needs special consideration in this article. In view of the part played by enfeebled circulation and secondary infections in the causation of thrombosis, prophylactic measures should be directed toward maintaining good nutrition, strengthening the heart's action, and warding off secondary infection, so far as may be, or treating accessible foci of infection antiseptically.

Sir A. E. Wright and Lieut. Knapp have urged that in enteric fever, as soon as the danger of intestinal haemorrhage has been surmounted, citric acid should be given as a prophylactic, or that citrated milk, which contains less calcium salts than ordinary milk, should be given. They argue that the citric acid removes calcium salts or decalcifies the blood, and that the liability to coagulation is thus diminished. The citrated milk, which is partially decalcified, is made by adding to milk 0·25 to 0·5 per cent of citrate of sodium (20 to 40 grains to the pint).

The general indications for treatment are to secure as speedily as possible an adequate collateral circulation, in order to ward off the danger of tissue-necrosis or gangrene from arterial thrombosis and the effects of passive congestion from venous thrombosis ; and, above all, in the case of venous thrombosis, to guard against the detachment of emboli. These

indications are best met by absolute rest, suitable position and immobilisation of the thrombosed extremity, and nourishing diet.

Stimulated by the success of Carrel's experimental arteriotomy and suture of arteries, surgeons have followed this plan in arterial thrombosis for removal of the clot. Lecène reports a case in which a clot re-formed in a brachial artery, which had been opened and emptied of clot due to arteritis. In thrombo-angiitis obliterans an attempt has been made to divert the blood-stream from the affected arteries into the veins. According to Buerger arterio-venous anastomosis had been done four times with one successful result up to 1909. It is essential that the veins should be healthy, and unfortunately they are often similarly affected in thrombo-angiitis obliterans.

With venous thrombosis of a lower extremity the patient should lie on the back with the limb elevated on an inclined plane, or in a trough well lined with cotton wool. The limb should be kept warm by wrapping in cotton wadding, and hot fomentations of lead-water and laudanum, or some similar preparation, may be applied. If the condition of the heart indicate it, digitalis or other cardiac tonic may be given. At the height of the process the pain may be so intense as to require the use of opium or some of its derivatives.

It is all-important to know what not to do. The patient should be cautioned against moving the leg, especially against any sudden jerk. Palpation of the affected veins should be of the gentlest sort, and is better omitted altogether. All unnecessary movements and manipulations should be avoided. Nothing is gained, and harm may be done by resorting, before all danger of embolism is past, to the old-fashioned treatment of rubbing in mercurial or belladonna ointment. The length of time that the patient should remain quiet in bed will vary according to the severity of the case. Although the thrombotic process does not usually progress after the tenth or twelfth day, it is a general rule that the patient should not be allowed to walk in less than forty days. A large number of the deaths from pulmonary embolism have occurred when the patient first walks, or goes to stool, or takes a bath.

Light bandaging of the lower part of the leg assists the circulation ; but, if applied at all, it should be with only minimal compression. After the danger of embolism is passed, massage and bandaging may be employed to advantage, or a long elastic stocking worn.

If gangrene result from arterial thrombosis, the time and site of operation should be determined upon surgical principles.

WM. H. WELCH. 1899.
H. D. ROLLESTON. 1909.

REFERENCES[1]

Coagulation: 1. FULD. *Zentralbl. f. Physiol.*, 1903, xvii. 529.—2. LOEB. *Med. News*, New York, 1905, lxxxvi. 577.—3. MELLANBY. *Journ. Physiol.*, Cambridge,

---

[1] The references are only to authors cited in the text, and are not intended to be a complete bibliography of the subject. The references to authors cited under different headings in the text will usually be found only under the first heading in which the reference appears.

1909, xxxviii. 28.—4. MORAWITZ. Hofmeister's *Beitr. z. chem. Physiol. u. Path.*, 1903, v. 133.

Structure of Thrombi: 5. ARNOLD. *Virchows Arch.*, 1897, cl. 445.—6. BIZZOZERO. *Ibid.*, 1882, xc. 261.—7. COLE, RUFUS. "On the Production of an Agglutinating Serum for Platelets," *Johns Hopkins Hosp. Bull.*, Balt., 1907, xvii. 261.— 8. DETERMANN. *xvi. Kongr. f. inn. Med.*, 1898, 237.—9. EBERTH und SCHIMMEL-BUSCH. *Die Thrombose nach Versuchen u. Leichenbefunden*, Stuttgart, 1888.—10. FOREL. Lubarsch u. Ostertag's *Ergebnisse der allg. Path.*, 1903, ix.—11. HAMMARSTEN. *Ztschr. f. physiol. Chem.*, 1896-97, xxii. 333.—12. HANAU. *Fortschr. d. Med.*, 1886, iv. 385.—13. HAYEM. *Compt. rend. de l'Acad. des sc.*, July 18, 1882.—14. KEMP, CALHOUN, and HARRIS. (Platelets) *Journ. Am. Med. Assoc.*, Chicago, 1906, xlvi. 1091.—15. KLEBS. *Allg. Path.*, Th. ii., Jena, 1889.—16. LÖWIT. *Arch. f. exp. Path. u. Pharm.*, 1887, xxiii. 1, and xxiv. 188.—17. LUBNITZKY. *Ibid.*, 1885, xix. 185.— 18. MANTEGAZZA. *Gazz. med. lombard.*, 1869.—19. MAXIMOW. *Arch. f. Anat. u. Physiol.*, 1899, 33.—20. MOSSO. *Virchows Arch.*, 1887, cix. 205.—21. MÜLLER, FR. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1898, xxii. 498.—22. OSLER. *Seguin's Arch. of Med.*, Feb. 1881.—23. *Idem. Centralbl. f. med. Wiss.*, July 29, 1882.—24. *Idem. Cartwright Lectures*, 1886.—25. PAGNIEZ. "Aperçu sur l'état actuel de la question des plaquettes sanguines," *Arch. des mal. du cœur*, Paris, 1909, ii. 1.—26. PITRES. *Arch. d. phys. norm. et path.*, 1876, 230.—27. VON RECKLINGHAUSEN. *Handb. d. allg. Path. d. Kreislaufs u. d. Ernährung*, Stuttgart, 1893.—28. VIRCHOW. *Gesammelte Abhandlungen*, Frankf., 1856.—29. WEIGERT. *Virchows Arch.*, 1877, lxx. 483, and 1880, lxxix. 87.—30. *Idem.* "Thrombose," in *Eulenburg's Real-Encyclopädie.* —31. WELCH. "The Structure of White Thrombi," *Trans. Path. Soc. of Phila-delphia*, 1887, xiii.—32. WLASSOW. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1894, xv. 543.—33. ZAHN. *Virchows Arch.*, 1875, lxii. 81.—34. ZENKER, K. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1895, xvii. 448.—35. ZIEGLER. *Lehrb. d. allg. Path. u. spec. path. Anat.*, 9te Aufl. Bd., i. 149.

Agglutinative (Hyaline) Thrombi: 36. BOXMEYER. *Journ. Med. Res.*, Boston, 1903, ix. 146.—37. FLEXNER. *Univ. Penn. Med. Bull.*, Phila., 1902, xv. 324.—38. *Idem. Journ. Med. Res.*, Boston, 1902, viii. 316.—39. HUETER. *Deutsche Ztschr. f. Chir.*, 1873-74, iv. 105, 330.—40. KLEBS. *Die allgemeine Pathologie*, 1887, ii. 113.— 41. KRIEGE. *Virchows Arch.*, 1889, cxvi. 63. — 42. PEARCE. *Journ. Med. Res.*, Boston, 1904, xii. 1, 329, 1906, xiv.—43. PEARCE and WINN. *Am. Journ. Med. Sc.*, Phila., 1904, cxxviii. 669.—44. VON RECKLINGHAUSEN. *Handbuch d. allg. Path. d. Kreislaufs u. d. Ernährung*, 1893.—45. RIBBERT. *Die path. Anat. u. d. Heil. d. durch Staph. pyog. aur. hervorgeruf. Erkrank.*, Bonn, 1891.—46. WELCH. *Brit. Med. Journ.*, 1902, ii. 1112.—47. WELCH and CLEMENT. "Remarks on Hog Cholera and Swine Plague," *Proc. 30th Ann. Convention, U.S. Vet. Med. Assoc.*, etc., Chicago, 1893.—48. WELLS, H. G. *Chemical Pathology*, 1907. The W. B. Saunders Company.

Growth, Metamorphoses, and Organisation: 49. ASCHOFF. *Virch. Arch.*, 1892, cxxx. 93.—50. BENEKE. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1890, vii. 95.— 51. LEGG, WICKHAM. *Trans. Path. Soc.*, Lond., 1878, xxix. 50.—52. THOMA. *Lehrb. d. path. Anat.* i., Stuttgart, 1894.—53. ZAHN. *Virchow-Festschrift. Internat. Beitr.* ii. 199.

Etiology: 54. ADDIS. "The Effect of the Administration of Calcium Salts and of Citric Acid on the Calcium-content and Coagulation-time of the Blood," *Quart. Journ. Med.*, Oxford, 1909, ii. 149.—55. ARTHUS et PAGÈS. "Nouvelle théorie chinique de la coagulation du sang," *Arch. de physiol. norm. et path.*, 1890, sér. 5, ii. 739.—56. BAILLIE, MATTHEW. *Trans. Soc. Improvement Med. and Chir. Knowledge*, 1793, i. 119.—57. BAUMGARTEN. *D. sogen. Organisation d. Thrombus*, Leipz., 1877. —58. BEUGNIER-CORBEAU. *Gaz. méd. de Liège*, 1890, p. 348.—59. BIRCH-HIRSCH-FELD. *Kongr. f. inn. Med.*, 1892, 28.—60. BRODIE and RUSSELL. *Journ. Physiol.*, 1897, xxi. 390.—61. BRÜCKE. *Brit. and For. Med.-Chir. Rev.*, 1857, xix. 183.—62. COHNHEIM. *Vorles. üb. allg. Path.*, Bd. i., Berl., 1882.—63. CRUVEILHIER. *Anat. path.*, Paris, 1829-42.—64. DAVY, JOHN. *Researches, Physiological and Anatomical*, Lond., 1839.—65. DENYS. *Centralbl. f. allg. Path. u. path. Anat.*, 1893, iv. 174.— 66. EGUET. *Mitth. a. Klin. u. med. Inst. d. Schweiz*, 1894, ii. Hft. 4.—67. EICHHORST. "Über multiple Arterialthrombose," *Arch. f. klin. Med.*, 1904, lxxx. 75.—68. VAN EMBDEN. *Fortschr. d. Med.*, 1898, xvi. 241, 281.—69. EHRLICH und LAZARUS. *Die Anämie*, 1. Abth. Wien, 1898.—70. FLEXNER. *Journ. Exp. Med.*, 1896, i. 559.—71. FREUND. *Wien. med. Blätter*, 1886, xix. 296.—72. *Idem. Wiener med. Jahrb.*, 1888,

259.—73. GLÉNARD. *Contrib. à l'étude des causes de la coag. spontan. du sang,* Thèse, Paris, 1875.—74. GROTH. *Ueb. d. Schicksale farbloser Blutkörperchen,* etc., Inaug. Diss., Dorpat, 1884.—75. HALLIBURTON. *Journ. Physiol.,* 1893, xiii. 806 ; xv. 90, and (with PICKERING) xviii. 285.—76. HAYEM. *Du sang et de ses altérations anatomiques,* Paris, 1889.—77. *Idem. Wien. med. Zeit.,* 1897, Nos. 17-19.—78. HUNTER, JOHN. "Obs. on the Inflam. of the Intern. Coat of Veins," *Trans. Soc. Improvement Med. and Chir. Knowledge,* 1793, i. 18.—79. KÖHLER. *Ueb. Thrombose u. Transfusion.* Inaug. Diss., Dorpat, 1877.—80. LAENNEC. *De l'auscult. médiate,* etc., Paris, 1819.—81. LANDOIS. *Die Transfusion d. Blutes,* Leipz., 1875.—82. VON LIMBECK. *Prag. med. Wchnschr.,* 1890, xv. 351, 365.—83. LOEB, L. *Journ. Med. Research,* Boston, 1903, x. 407.—83a. *Idem. Med. News,* N.Y., 1905, lxxxvi. 577.—84. MALLORY. *Journ. Exp. Med.,* 1898, iii. 611.—85. MARAGLIANO und CASTELLINO. *Ztschr. f. kl. Med.,* 1892, xxi. 415.—86. MARTIN, C. J. *Journ. Physiol.,* 1893, xv. 380.— 87. MUIR. *Journ. Anat. and Physiol.,* 1890-91, xxv.—88. NAUNYN. *Arch. f. exp. Path. u. Pharm.,* 1873, i. 1.—89. PAGET, J. *St. Barth. Hosp. Rep.,* 1866, ii. 82.— 90. PONFICK. *Deutsche Klinik,* 1867, Nos. 20-26.—91. *Idem. Virchows Arch.,* 1874, lx. 153.—92. *Idem. Ibid.,* 1875, lxii. 273.—93. PRATT. *Journ. Med. Research,* Boston, 1903, x. 120. 94. RANKE. *D. Blutvertheilung u. d. Thätigkeitswechsel d. Organe,* Leipz., 1891.—95. RICHARDSON. *Journ. Med. Research,* Boston, 1905, xii. 99.— 96. SAHLI. *Centralbl. f. inn. Med.,* 1894, 497.—97. SCHIMMELBUSCH. *Ueb. Thrombose im gerinnungsunfähigen Blute,* Inaug. Diss., Halle, 1886.—98. SILBERMANN. *Virch. Arch.,* 1889, cxvii. 288.—99. SINGER. *Arch. f. Gynäk.,* lvi. 218.—100. TÜRK. *Klin. Untersuch. üb. d. Verhalten d. Blutes bei Infectionskrankh.,* Wien, 1898.—101. VAQUEZ. *De la thrombose cachectique,* Thèse, Paris, 1890.—102. *Idem.* "De la phlébite," in *Clin. méd. de la Charité,* Paris, 1894, 751.—103. WEIGERT. *Fortschr. d. Med.,* 1887, v. 231.—104. WELLS, H. G. *Chemical Pathology,* 1907, Phila., and London.—105. WIDAL. *Étude sur l'infection puerpérale,* Thèse, Paris, 1889.— 106. WOOLDRIDGE. *On the Chemistry of the Blood and other Scientific Papers,* arranged by Sir Victor Horsley and Ernest Starling, Lond., 1893.—107. WRIGHT and KNAPP. "Causation and Treatment of Thrombosis in Typhoid Fever," *Med.-Chir. Trans.,* Lond., 1903, lxxxvi. 1.

Localisation : 108. ARNOLD. *Beitr. z. path. Anat. u. z. allg. Path.,* Jena, 1890, viii. 29.—109. BENIVIENI. *De abditis nonnullis ac mirandis morborum et sanationum causis,* Florent., 1507.—110. BIRCH-HIRSCHFELD. *Deutsch. med. Wchnschr.,* 1892, xviii. 267.—111. BOSTROEM. *Deutsch. Arch. f. klin. Med.,* 1895, lv. 219.—112. BUERGER, L. "Thrombo-angiitis obliterans," *Amer. Journ. Med. Sc.,* Phila., 1908, cxxxvi. 548.— 113. BURNS, ALLAN. *Obs. on some of the most frequent and important Diseases of the Heart,* Edinb., 1809.—114. ZUM BUSCH. *Ueb. d. Zusammensetzung d. Herzthromben,* Inaug. Diss., Freiburg, i. B., 1891.—115. VAN DER BYL. *Trans. Path. Soc.,* Lond., 1858, ix. 89.—116. CHOISY and NUHN, cited from No. 120.—116a. COOMBES. *Lancet,* London, 1909, i. 1377.—117. CZAPEK. *Prager med. Wchnschr.,* 1891, xvi. 458.—118. DELÉPINE. *Trans. Path. Soc.,* Lond., 1890, xli. 43.—119. EWART and ROLLESTON. *Trans. Clin. Soc.,* Lond., 1897, xxx. 190.—120. HERTZ. *Deutsch. Arch. f. kl. Med.,* 1885, xxxvii. 74.—121. HUTCHINSON, JONATHAN. *Arch. of Surg.,* Apr. 1898, 100.— 122. KRUMBHOLZ. *Arb. a. d. med. Klinik zu Leipzig,* 1893, 328.—123. KRUMM. *Deutsch. Arch. f. klin. Med.,* 1895, liv. 189.—124. LANCEREAUX. *Traité d'anat. path.,* i. 604, Paris, 1875-77.—125. KOTLAR. *Prag. med. Wchnschr.,* 1894, xix. 78, 97.—126. LEGG, WICKHAM. *Trans. Path. Soc.,* Lond., 1878, xxix. 49.—127. MACLEOD, N. *Edinb. Med. Journ.,* 1883, xxviii. 696.—128. M'MURRICK. *Brit. Med. Journ.,* 1906, ii. 1699.—129. OGLE. *Trans. Path. Soc.,* Lond., 1863, xiv. 127.—130. OSLER. *Johns Hopkins Hosp. Rep.,* 1890, ii. 56.—131. *Idem. Montreal Med. Journ.,* 1897, xxv. 729.—132. PARMENTIER. *Arch. gén. de méd.,* July 1889.—133. PAWLOWSKI. *Ztschr. f. klin. Med.,* 1894, xxvi. 482.—133a. POYNTON. *Lancet,* London, 1909, i. 1556.—134. VON RECKLINGHAUSEN, No. 27, and *Deutsch. Arch. f. klin. Med.,* 1885, xxxvii. 495.— 135. REDTENBACHER. *Wien. klin. Wchnschr.,* 1892, v. 688.—136. ROSENBACH. *Die Krankh. d. Herzens,* Hft. i. 180, Wien u. Leipz., 1893.—137. STANGE. *Arb. a. d. path. Inst. z. Göttingen,* 1893, 232.—138. VOELCKER. *Trans. Path. Soc.,* Lond., 1893, xliv. 31.—139. WAGNER. *Arch. d. Heilk.,* 1861, ii. 364.—140. WEICHSELBAUM, cited from No. 110.—141. WERTHEIMER. *Arch. de physiol.,* 1895, 5. s., vii. 107.—142. VON WINI-WARTER. *Arch. f. kl. Chir.,* 1878, xxiii. 202.—143. WOOD, WILLIAM. *Edinb. Med. and Surg. Journ.,* 1814, x. 50.—144. ZAHN. *Virch. Arch.,* 1889, cxv. 55.—145. V. ZIEMSSEN. *Kongr. f. inn. Med.,* 1890, 281.

Association with Certain Diseases : *Enteric Fever.*—146. BOINET et ROMARY. *Arch. d. méd. exp.*, 1897, ix. 902.—147. CARBONE. *Gazz. med. di Torino*, 1891, No. 23.—148. CROCQ. *Arch. d. méd. exp.*, 1894, vi. 583.—149. GILBERT et LION. *Bull. méd.*, 1889, iii. 1266.—150. GIRODE. *Ibid.*, 1889, iii. 1392.—151. HAUSHALTER. *Mercredi méd.*, Sept. 20, 1893.—152. HÖLSCHER. *Münch. med. Wchnschr.*, 1891, xxxviii. 43, 62.—153. KEEN. *Surgical Complications and Sequels of Typhoid Fever*, Philadelphia, 1898 (consult for other references to Arterial Thrombosis in Enteric Fever). 154. RATTONE. *Morgagni*, 1887, xxix. 577.—155. THAYER, W. S. *Johns Hopkins Hosp. Bull.*, Balt., 1904, xv. 323, and *New York State Journ. Med.*, 1903 (Literature).—156. VINCENT. *Mercredi méd.*, 1892, iii. 73.—157. VITI. *Atti d. r. Accad. d. fisiocrit. di Siena*, 1890, 4. s., ii. 109.

Influenza.—158. BÄUMLER. *Kongr. f. inn. Med.*, 1890, 305.—159. CHAUDET. *La phlébite grippale*, Paris, 1892.—160. CHIARI. *Prager med. Wchnschr.*, 1890, xv. 124.— 161. EICHHORST. *Deutsch. Arch. f. klin. Med.*, 1901, lxx. 544.—162. GUTTMANN und LEYDEN. *Die Influenza-Epidemie*, 1889-90, Wiesbaden, 1892. — 163. KLEBS. *Deutsch. med. Wchnschr.*, 1890, xvi. 278.—164. KUSKOW. *Virch. Arch.*, 1895, cxxxix. 406.—165. LASKER. Inaug. Diss., Freiburg, 1897.—166. LEICHTENSTERN, in Noth-nagel's *Spec. Path. u. Therap.*, Bd. iv. Th. ii. Abt. i., Wien, 1896.—167. LEYDEN. *Charité-Ann.*, xvii. and xviii.—168. RENDU. *Bull. méd.*, 1892, vi. 50, 296.

Pneumonia.—169. BLAGDEN. *St. Barth. Hosp. Journ.*, 1897-98, v. 122.—170. DA COSTA. *Philadelphia Med. Journ.*, 1898, ii. 519. — 171. DICKINSON, LEE. *Brit. Med. Journ.*, 1896, i. 149.—172. EICHHORST. *Deutsch. Arch. f. klin. Med.*, Leipzig, 1901, lxx. 547.—173. FABRIÈS. *Sem. méd.*, 1888, viii. 144.—174. GAULTIER. *Bull. et mém. Soc. anat. de Paris*, 1904, lxxix. 565.—175. LAACHE. *Deutsch. med. Wchnschr.*, 1893, xix. 785.—176. LEYDEN. *Centralbl. f. inn. Med.*, 1887, 25.—177. M'GREGOR. *Glasgow Med. Journ.*, 1908, lxx. 81 (References).—178. MACKENZIE, H. *Proc. Roy. Soc. Med.*, London, 1908, i. (Med. Sect.) 14.—179. OSLER. *The Principles and Practice of Medicine*, 1905, 181.—180. *Traité de médecine*, t. v. 374, 432 (for other references to arterial and venous thrombosis in pneumonia).—181. STEINER. *Johns Hopkins Hosp. Bull.*, Balt., 1902, xiii. 130.

Acute Articular Rheumatism.—182. GATAY. *Contrib. à l'étude de la phlébite rhumatismale*, Thèse, Paris, 1895.—183. LEGROUX. *Gaz. hebd. de méd.*, 1884, 140. —184. SCHMITT. *De la phlébite rhumatismale*, Thèse, Paris, 1884.

Appendicitis.—185. HAWARD. *Lancet*, London, 1906, i. 652.—186. TREVES and LETT. *Med.-Chir. Trans.*, London, 1905, lxxxviii. 449.

Tuberculosis. — 187. ASCHOFF. *Verhandl. d. deutsch. path. Gesellsch.*, 1899. — 188. BLUMER. *System of Medicine* (Osler and M'Crae), 1908, iv. 512. — 189. *Idem.* *Yale Med. Journ.*, 1909, xvi. 296. — 189a. *Idem.* *Amer. Journ. Med. Sc.*, Phila., 1899, cxvii.—190. DODWELL. *Ibid.*, 1893, cv. 641.—191. FLEXNER. *Johns Hopkins Hosp. Bull.*, 1891, iii. 120.—192. HEKTOEN. *Journ. Exp. Med.*, 1896, i. 112.—193. HIRTZ. *Mercredi méd.*, 1894, No. 40.—194. KRUMBHAAR. *Bull. Ayer Clin. Lab. Pennsylv. Hosp.*, Phila., 1908, No. 5, 66.—195. MICHAELIS und BLUM. *Deutsche med. Wchnschr.*, 1898, xxiv. 550.—196. SABRAZÈS et MONGOUR. *Rev. méd. de l'est*, Nancy, 1897, 306.—197. STROEBE. *Centralbl. f. allg. Path. u. path. Anat.*, 1897, viii. 998.

198. *Syphilis.*—DIEULAFOY. *Clinique médicale de l'Hôtel-Dieu*, Paris, 1905-6, v. 230.

199. *Gonorrhoea.*—HELLER. *Berlin. klin. Wchnschr.*, 1904, xli. 609.

Cachectic States.—200. CHARCOT. *Union méd.*, 1865, xxvi. 165.—201. FUTCHER. *Johns Hopkins Hosp. Bull.*, Balt., 1908, xix. 49.—202. GOUGET. *Bull. Soc. anat.*, 1894, No. 13.—203. OSLER and M'CRAE. *Cancer of the Stomach*, 1900, 57.—204. PITRES, BITOT, et REGNIER, cited from No. 102.—205. VON RECKLINGHAUSEN. No. 27.—206. RIGOLLET. *De la phlébite paludéenne*, Thèse, Bordeaux, 1891.

Cardiac Incompetency.—207. BALDWIN, HELEN. *Journ. Amer. Med. Assoc.*, Chicago, 1897, xxix. 371.—208. COHN. *Klinik d. embol. Gefässkrankh.*, Berlin, 1860.—209. CHEADLE and LEES. *Lancet*, 1898, ii. 206 (reported by Poynton).—210. DESQUIENS. *Thèse de Paris*, 1906.—211. DÉVÉ. *Bull. Soc. de méd. de Rouen*, 1907, xlvi. 20.—212. HIRSCHLAFF. Inaug. Diss., Berlin, 1893.—213. HUCHARD. *Rev. gén. de clin. et de thérap.*, 1897, xi. 787.—214. KAHN (and HANOT). *Arch. gén. de méd.*, 1896, ii. 469. —215. MADER. *Jahrb. d. Wien. k.-k. Krankenanst.*, 1895, 1897, iv. 252.—216. WELCH. *Festschrift in Honour of A. Jacobi*, New York, 1900, 463.

Chlorosis.—217. BALL. *Trans. Assoc. Amer. Physicians*, 1889, iv. 52.—218.

VON NOORDEN, in Nothnagel's *Spec. Path. u. Therap.*, Bd. viii. Th. ii., Wien, 1897.—219. SCHWEITZER. *Virchows Arch.*, 1898, cli. 337. (The three preceding articles contain the principal references to chlorotic thrombosis. The following Nos. 220 to 232 are the references to cases not found in them.)—220. AUDRY. *Lyon méd.*, 1892 and 1893.—221. DICKINSON, LEE. *Trans. Clin. Soc.*, Lond., 1896, xxix. 63.—222, DUCKWORTH and BUZZARD. *Brit. Med. Journ.*, 1896, i. 149.—223. GAGNONI. *Riforma med.*, 1897, xiii. 472.—224. GUINON. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1896 xii. 297.—225. GUTHEIL. Inaug. Diss., Freiburg, 1892.—226. HAW-THORNE. *Lancet*, Lond., 1908, ii. 857.—227. HAYEM. *Bull. méd.*, 1896, 261.—228. POWELL, DOUGLAS. *Lancet*, 1888, ii. 1124.—229. Dr. THAYER's case was of a chlorotic young woman with thrombosis of left femoral, iliac, and uterine veins. Death from pulmonary embolism.—230. VAQUEZ. See No. 102.—231. VERGELY. *Bull. méd.*, 1889, 1175.—232. VILLARD. *Assoc. franç. pour l'avancement des sciences*, 1891, ii. 791. Paris, 1892.—233. BUTTERSACK. *Ztschr. f. klin. Med.*, 1897, xxxiii. 456.—234. SANKEY, W. *Ed. Med. and S. Journ.*, 1814, 401.

Gouty, Idiopathic, and Primary Infective Thrombosis.—235. BARBE. *La France méd.*, 1898. (Syphilis.)—236. BRIGGS. "Recurring Phlebitis of Obscure Origin," *Johns Hopkins Hosp. Bull.*, Balt., 1905, xvi. 228.—237. DAGUILLON. *Contrib. à l'étude clin. de la phlébite primitive*, Thèse, Paris, 1894. — 238. DOWSE. *Lancet*, 1879, ii. 268.—239. OSLER. *Trans. Assoc. Amer. Physicians*, 1887, x. 135.—240. PAGET. No. 89.—241. TUCKWELL. *St. Barth. Hosp. Rep.*, 1874, x. 23.

Post-Operative.—242. CORDIER. *Journ. Amer. Med. Assoc.*, 1905, xlv. 1792.

Pregnancy. — 243. GOLDSBOROUGH. *Johns Hopkins Hosp. Bull.*, Balt., 1904, xv. 193.

Effects and Symptoms : *Cardiac, Arterial, and Capillary Thrombosis.* —244. BARTH. *Deutsch. med. Wchnschr.*, 1896, xxii. 269.—245. BLACHEZ. *Gaz. des hôp.*, 1866, No. 13.—246. Box. "Pulmonary Embolism due to displacement of a Thrombus in the Main Trunk of the Pulmonary Artery," *Trans. Clin. Soc.*, Lond., 1906, xxxix. 189.—247. BRADFORD and LAWRENCE. "Necrosis of the Cortex of both Kidneys," *Journ. Path. and Bacteriol.*, Edin. and Lond., 1898, v. 195.—248. BRISTOWE. *Trans. Path. Soc.*, Lond., 1870, xxi. 143.—249. CHARCOT. *Compt. rend. Soc. biol.*, Paris, 1858, 1859, 2. s., v. 225.—250. CHIARI. *Prager med. Wchnschr.*, 1897, Nos. 6, 7.—251. GERHARDT. *Würzburg. med. Ztschr.*, Bd. iv. and v.—252. HALPERIN. "Clinical Manifestations of Renal Infarct," *Arch. Int. Med.*, Chicago, 1908, i. 320.—253. HEIDENHAIN. *Deutsch. med. Wchnschr.*, 1891, xvii. 1087.—254. HERRINGHAM and GRIFFITH. "Necrosis of the Cortex of both Kidneys," *Brit. Med. Journ.*, 1905, ii. 1043.—255. KIDD, PERCY. *Trans. Path. Soc.*, Lond., 1886, xxxvii. 197.—256. LLOYD. *Lancet*, Lond., 1906, i. 156.—257. M'PHEDRAN and MACKENZIE. *Trans. Assoc. Amer. Phys.*, Phila., 1903, xviii. 337.—258. NAUNYN, in Nothnagel's *Spec. Path. u. Ther.*, Bd. vii. Th. vi. 216, Wien, 1898. — 259. PITT, NEWTON. *Trans. Path. Soc.*, Lond., 1893, xliv. 52.—260. PORTER. *Journ. Exp. Med.*, 1896, i. 46. — 261. WEBER, PARKES. "Anuria with Necrosis of the Renal Convoluted Tubules," *Lancet*, Lond., 1909, i. 601.—262. WELCH, in *Pepper's System of Medicine*, ii. 505.—263. VON ZIEMSSEN. *Vide* No. 145.

Effects of Venous Thrombosis : *Oedema.*—264. BOLTON, C. *Proc. Roy. Soc.*, 1907, B. lxxix. 267.—265. COHNHEIM. *Vorles. über allg. Path.*, Bd. i. S. 150, 492.—266. COHNSTEIN, in Lubarsch u. Ostertag's *Ergebnisse*, 1896, ii. 563 ; Wiesbaden, 1897 (Literature of Theories of Oedema).—267. HAMBURGER. *Virchows Arch.*, 1895, cxli. 398.—268. LAZARUS-BARLOW. *Phil. Trans. Roy. Soc.*, 1894, clxxxv. B. 779.—269. *Idem.* *A Manual of General Pathology*, London, 1904.—270. LOEB. *Pflügers Arch.*, 1898, lxxi. 457.—271. STARLING, E. H. *Lancet*, Lond., 1896, i. 1407.—272. *Idem.* *The Fluids of the Body*, Lond., 1909, 158.

Thrombosis of Veins of Limbs. —273. BUERGER. "Veins in Thrombo-angiitis," *Journ. Am. Med. Assoc.*, Chicago, 1909, lii. 1319.—274. GALLIARD. *Méd. mod.*, Paris, 1894, v. 861.—275. ROLLESTON. *Lancet*, London, 1906, i. 29.—276. SINGER. *Arch. f. Gynäk.*, 1898, lvi. 218.—277. TROUSSEAU. *Clinical Medicine*, v., Transl. New. Syd. Soc., 1872.

Inferior Vena Cava.—281. BOSANQUET. *Edin. Med. Journ.*, 1902, N.S. xii. 250.—282. BRESLER. *Deutsch. med. Wchnschr.*, 1897, xxiii. 179.—283. GRIFFON. *Bull. Soc. anat.*, Paris, 1898, lxxii. 753.—284. KRAUSE. *Ueber Verschluss der Vena Cava Superior und der Vena Cava Inferior*, Tübingen, 1894 (102 cases).—285. LÖSCHNER. *Prag. med. Wchnschr.*, 1888.—286. MACCALLUM. *Johns Hopkins Hosp. Bull.*, Balt.,

1903, xiv. 88.—287. Phillips, S. *Trans. Clin. Soc.*, Lond., 1901, xxxiv. 24.—288. Schlesinger. *Deutsch. med. Wchnschr.*, 1896, xxii. 460.—289. Stillman and Carey. *Amer. Journ. Med. Sc.*, Phila., 1909, cxxxvii. 333.—290. Thomas. "Beitr. z. Differentialdiagnostik zwischen Verschluss d. Pfortaders. u. d. unteren Hohlvenen," *Bibliotheca Med.*, Cassel, 1895.—291. Vimont. *Contribution a l'étude des obliterations de la veine cave infér.*, Thèse de Paris, 1890.—292. Unruh. *Deutsch. med. Wchnschr.*, Leipzig, 1896, xxii. 746.

Mesenteric Veins.—293. Bradford, J. R. *Trans. Clin. Soc.*, Lond., 1898, xxxi. 203.—294. Corner. *Erasmus Wilson Lectures*, 1904, Lond., 25.—295. Fitz. *Trans. Assoc. Amer. Phys.*, 1887, ii. 140.—296. Jackson, Porter, and Quinby. *Journ. Amer. Med. Assoc.*, Chicago, 1904, xlii. 1469 ; xliii. 25, 113, 183.—297. Johnson, R. *Trans. Clin. Soc.*, Lond., 1898, xxxi. 212.—298. Köster. *Deutsch. med. Wchnschr.*, 1898, xxiv. 325.—299. Rolleston. *Trans. Path. Soc.*, Lond., 1892, xliii. 49.

Splenic Vein.—300. Brown, W. Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii. 155.—301. Dévé. *Semaine méd.*, Paris, 1908, xxviii. 212.—302. Köster. *Deutsch. med. Wchnschr.*, 1898, xxiv. 325.—303. Rolleston. *Trans. Path. Soc.*, Lond., 1892, xliii. 49.

Obliteration of Superior Vena Cava.—304. Banti. *Sperimentale*, 1891, xlv. 405. —305. Duchek. *Vierteljahrsschr. f. d. prakt. Heilk.*, Prag, 1854, xli. 109.—306. Duckworth and Garrod. *St. Barth. Hosp. Rep.*, 1896, xxxii. 71.—307. Osler. *Johns Hopkins Hosp. Bull.*, Balt., 1903, xiv. 169.—308. Oulmont. *Soc. méd. obser.*, Paris, 1856, iii. 361, 468.—309. Poynton. *Lancet*, 1898, ii. 206.

Innominate, Subclavian, and Jugular.—310. Baldwin. *Journ. Amer. Med. Assoc.*, 1897, xxxix. 371.—311. Hirschlaff. Inaug. Diss., Berlin, 1893.—312. Pohl. Inaug. Diss., Göttingen, 1887.

Pulmonary Veins.—313. Schmale. Inaug. Diss., Würtz., 1889.

Corpora Cavernosa Penis.—314. Duckworth, Sir D. "A Case of Gout of the Penis," *Trans. Clin. Soc.*, Lond., 1892, xxv. 97.—315. Weber, F. P. "Persistent Priapism, from Thrombosis of Corpora Cavernosa," *Edin. Med. Journ.*, 1898, N.S. iv. 267.

Treatment : 316. Buerger. *Journ. Am. Med. Assoc.*, Chicago, 1909, lii. 1319.—317. Lecène. *Arch. mal. d. cœur*, Paris, 1909, ii. 138.—318. Wright, A. E., and Knapp. *Med.-Chir. Trans.*, Lond., 1903, lxxxvi. 11.

W. H. W.   1899.
H. D. R.   1909.

EMBOLISM[1]

By Prof. William H. Welch, M.D.

Definition.—Embolism is the impaction in some part of the vascular system of any undissolved material brought there by the blood-current. The transported material is an embolus. Embolism may occur likewise in lymphatic vessels.

Historical Note.—Rudolf Virchow was the creator of the doctrine of embolism. There is scarcely another pathological doctrine, of equal magnitude, the establishment of which is so largely the work of a single man. Not but that there were foreshadowings of this conception before Virchow, notably by Bonetus and van Swieten in the seventeenth and eighteenth centuries, and by Allibert and François in the early part of

---

[1] As Prof. Welch was unable to revise his standard article at the last moment, some additions have been made by one of the editors (H. D. R.).

the last century. A few observers and experimenters, indeed, anticipated some of Virchow's results. The wonder is that until Virchow's time the idea of embolism remained so foreign to medical thought ; so obvious and necessary a corollary does it seem to be of the discovery of the circulation of the blood. Between the years 1846 and 1856 Virchow constructed the whole doctrine of embolism upon the basis of anatomical, experimental, and clinical investigations, which for completeness, accuracy, and just discernment of the truth must always remain a model of scientific research in medicine. These discoveries introduced new chapters and necessitated a recasting of many old ones in pathology. A number of important morbid conditions, among which pulmonary embolism and cerebral embolism may be especially mentioned, were now for the first time clearly recognised. Virchow's studies of thrombosis and his demonstration that not all intravascular ante-mortem clots are formed at the place where they are found, and that infarcts are not the result of inflammation and capillary phlebitis, put an end to the false and to us at present almost incomprehensible ideas then prevailing as to the overshadowing importance of phlebitis in pathological processes. Especially was the doctrine of metastasis, which in old days was one of the most mystical in medicine, greatly expanded and at the same time placed upon an intelligible and firm foundation.

The new fields opened by Virchow have been industriously cultivated by a multitude of workers. The additions to our knowledge have been many and valuable, but they have related mainly to details, and can scarcely be said to have led to new points of view. The works of Bernhard Cohn and of Cohnheim may be signalised as among the most important of the contributions since Virchow's early publications. Cohn's remarkable book, published in 1860, is extraordinarily rich in anatomical, experimental, and clinical facts, and it is well for any one who believes that he has a new observation or opinion concerning embolism to consult it before venturing on publication ; a precaution which has evidently been often neglected by writers on the subject.

Varieties of Emboli.—Substances of the most varied character, solid, liquid or gaseous, may enter the circulation and be conveyed as emboli. Unless some special epithet be used, an embolus is generally understood to be a detached thrombus, or part of it, including under this designation endocarditic vegetations. Other possible sorts of emboli are fragments of diseased heart-valves, calcified masses, bits of tissue, tumour-cells, parenchymatous cells, animal or vegetable parasites, fat, air, pigment-granules, and foreign bodies. Emboli of air, of fat, and of parenchymatous cells will be considered separately. An important classification, as regards their effects, is into bland or aseptic emboli and toxic or septic emboli.

Sources of Emboli.—Emboli in the lungs come from the systemic veins, the right heart, or the pulmonary artery ; those in branches of the portal vein come from the radicles or trunk of this vein ; those in systemic arteries from the pulmonary veins, the left heart, or some artery between the heart and the location of the embolus. Sources of aberrant emboli.

resulting from unusual modes of transportation, will be considered subsequently (p. 765).

Various features in the structure and disposition of thrombi bearing upon the detachment of emboli have been described in the preceding article.   Here may be especially recalled the continuation of an occluding venous thrombus in the form of a partly obstructing thrombus beyond the entrance of an important branch, and the occurrence of softening in the interior of older thrombi ; phenomena evidently favourable to the detachment of fragments.   Globular thrombi in the right heart, particularly in the auricular appendix, are a fruitful source of the emboli which cause pulmonary infarction in heart disease.   Vegetations of the aortic and mitral valves, particularly of the latter, furnish the great majority of emboli in the aortic system.   Thrombosis or embolism of an arterial trunk—as of the internal carotid, splenic, femoral—is often followed by the conveyance of fragments of the plug into branches of the artery.   When the plug in the main trunk is an embolus, this secondary embolism is described by Cohnheim as " recurrent "—an epithet which has also been applied to retrograde embolism, and, therefore, to avoid confusion, had better not be used in either sense.

The detection of the source of an embolus is often unattended by any difficulty ; but sometimes it requires prolonged and painstaking search, and occasionally even such a search is unrewarded.   The greatest difficulties are encountered when the source is in some peripheral venous thrombus which has caused no symptoms and is unattended by lesions suggestive of its location.   An entire thrombus may be dislocated and transported as an embolus.

Site of Deposit.—Emboli are carried along by the blood-current until they are caught on some obstruction, or become lodged in a channel too narrow to permit their further passage.   It is evident that embolism can scarcely occur except in the arterial system, pulmonary and systemic, and in branches of the portal vein.   The rare instances of embolism of systemic veins will be considered under aberrant embolism (p. 765).   An extremely rare occurrence, of which several instances are recorded, is the blocking of the tricuspid or mitral orifice by an embolus.   The result is, of course, sudden death.   Very often an embolus is caught at an arterial bifurcation, which it rides with a prolongation extending into each branch (riding embolus).   This may happen where the diameter of each branch is greater than that of the embolus.   It is not uncommon for several emboli to enter successively the same branch of the pulmonary artery.

Any artery open to the circulating blood may receive an embolus of appropriate size.   The course followed by an embolus in its travels is determined by purely mechanical factors, of which the most important are the size, form, and weight of the plug ; the direction, volume, and energy of the carrying blood-stream ; the size of branches and the angles at which they are given off ; and the position of the body and its members. In accord with these principles we find emboli in the lower lobes of the lungs oftener than in the upper ; and in the right lung oftener than in the

left, the right pulmonary artery being larger than the left. Emboli from the left heart are more frequently carried into the abdominal aorta and its branches than into the carotid or subclavian arteries. The left carotid, arising directly from the aortic arch at its highest point, is in more direct line with the aortic blood-stream than is the right carotid, and is therefore a commoner recipient of emboli. The left common iliac artery is also somewhat more directly in the line of the current in the abdominal aorta, and, therefore, receives emboli somewhat more frequently than the right.

The order of frequency in which emboli are found in the different arteries may be given about as follows :—pulmonary, renal, splenic, cerebral, iliac and the lower extremities, axillary and upper extremities, coeliac axis with its hepatic and gastric branches, central artery of the retina, superior mesenteric, inferior mesenteric, abdominal aorta, coronary of the heart. There is, however, considerable difference of statement on this point. As a matter of fact this list, like similar ones, does not inform us of the frequency with which the different arteries of the body receive emboli ; for it is evident that it is based almost entirely upon embolic manifestations, and not upon the mere presence of emboli. If estimates of frequency be based only on infective emboli, the order would be in several respects different, the hepatic artery, for example, standing higher in the list, and the cerebral lower—sufficient evidence that the customary data for determining the frequency of embolism in different arteries relate only to such emboli as leave behind some record of their presence. Infective emboli, however, do not inform us of the incidence of embolism in different arteries ; for these produce abscesses or other lesions in certain special situations, and not in every place where they may lodge ; a fact which is brought out clearly in the experimental injections of bacteria into the circulation of animals. It seems to me very probable that, of the systemic arteries, those going to the lower extremities must be more frequent receptacles of emboli than either the splenic or the renal ; but the smaller plugs in the former usually leave no readily demonstrable record of their presence, whereas in the latter they always do.

Aberrant Embolism.—Certain exceptions to the general rules already stated concerning the sources and direction of transportation of emboli may be grouped under the heading of aberrant or atypical embolism, the latter epithet being the one employed by Scheven to designate paradoxical embolism, and retrograde embolism.

Zahn gave the name "paradoxical embolism," and his assistant Rostan the name "crossed embolism," to the transportation of emboli derived from veins into the systemic arteries without passing through the pulmonary circulation. Cohnheim was the first to note the passage of venous emboli through an open foramen ovale into the aortic system ; and since then there have been enough observations of this so-called paradoxical embolism to prove that, although not frequent, it is really of practical importance, and not merely a curiosity. Zahn and Rostan found an open foramen ovale in about one-fifth of their necropsies, which

is a considerably smaller percentage than most pathologists, who have investigated the subject, have found. An opening in the form of an oblique slit is certainly very often present in the oval fossa (in 34 per cent of all cases according to Firket), and it has been demonstrated by actual observation that, under certain conditions, this form of opening suffices for the transit of emboli. In three cases an embolus was found by Zahn and Rostan actually engaged in the opening, and two or three similar observations have been made by others.

I have found records of twenty-eight cases of paradoxical embolism, and there is no reason to suppose that this list is complete. The evidence upon which the diagnosis is usually based is an open foramen ovale and the presence in the systemic arteries of coarse emboli, for which the only source to be found is on the venous side or in the right auricle. Whilst in some of the cases there may be room for scepticism as to the venous origin of the arterial embolism, there can be none for Schmorl's observation, in a case of traumatic laceration of the liver, of plugs of hepatic tissue in the left auricle and the main trunk of the renal artery, with an open foramen ovale admitting a finger. Conditions favouring the occurrence of paradoxical embolism are, according to Zahn, increased pressure in the right auricle and lowered pressure in the left. In these circumstances the opening in the oval foramen is widened, and its walls bulge toward the left auricle. Rostan and Hauser have seen thrombi extending from the right auricle through the oval foramen into the left.

The best explanation of certain tumour metastases without pulmonary implication is by paradoxical embolism. Here, however, there is sometimes another possibility ; for, as Zahn has demonstrated, tumour-cells not of large size may pass through the pulmonary capillaries. This is well shewn in the occurrence of secondary melanotic sarcoma of the liver after removal of a primary tumour from the eye, the lungs being commonly free from metastasis. Although the lungs are an excellent filter, their capillaries are certainly so wide that they may permit the transit of emboli too large to pass through capillaries elsewhere in the body.

The first conclusive observation of retrograde transport of an embolus in a human being was made by Heller, in 1870, who found, in a case of primary cancer of the caecum and ileum, a loose plug of cancerous tissue in a branch of an hepatic vein. The only metastatic growths were in the mesenteric, retroperitoneal, and mediastinal lymphatic glands. Long before Heller, however, the conception of retrograde transport of venous emboli was familiar to pathologists ; especially in the discussions of the explanation of metastatic hepatic abscesses in cases in which the lungs are not involved and the atrium of infection does not communicate with the portal system. The experimental side of the subject was diligently cultivated. The general trend of opinion among pathologists, however, was opposed to the acceptance of the doctrine of retrograde transport, under conditions occurring in human beings, until the publication of von Recklinghausen's article on the subject in 1885. He reported a convincing observation of embolism of the renal veins with masses of sarcoma,

derived from a primary growth of the tibia, and also of retrograde embolism from the left auricle into the pulmonary veins. Since this publication there have been a number of equally conclusive demonstrations of the retrograde transport of venous emboli, and the subject has been taken up again on the experimental side. Retrograde venous embolism is an interesting, but, so far as at present known, a rare occurrence.

The difficulty of making sure that a suspected thrombotic embolus in a systemic vein is not an autochthonous thrombus is doubtless the reason why most of the reports of retrograde transport relate to emboli of tumour-cells or parenchymatous cells. In addition to Heller's and von Recklinghausen's cases already mentioned, reference may be made to Arnold's observation of masses, from a primary mammary carcinoma, filling the superior longitudinal sinus, with invasion of the wall of the sinus from within by the new growth, but without any intracranial tumour outside of this wall ; or indeed any metastasis elsewhere in the body except in the axillary and cervical lymph-glands : and also to Ernst's case of primary angio-sarcoma of the left kidney, growing into the renal vein, with a loose plug of sarcomatous tissue distending a branch of a coronary vein of the heart without connexion with a metastatic growth. Bonome's observation of cancer of the thyroid with metastatic nodules in the liver, developing from plugs in the hepatic veins, should probably also be included in the list, as well as two cases of Bonome, reported by Lui, in one of which a cancerous embolus secondary to cancer of the rectum was found in a branch of the superior mesenteric vein ; and in the other a similar retrograde embolus, secondary to adeno-carcinoma of the liver, was met with in the right pampiniform plexus.

To Schmorl's and Lubarsch's cases of emboli of liver-cells in the cerebral and the renal veins may be added two observations from my laboratory, of which one has been reported by Flexner, of clumps of liver-cells in branches of the renal vein in cases with extensive hepatic necroses.

That retrograde transport of ordinary venous thrombi may occur, is demonstrated by Arnold's discovery in a large branch of an hepatic vein of a riding embolus identical in appearance with a thrombus which occupied the right ovarian vein and extended some distance into the inferior vena cava. Cohn accepted, for a limited class of cases, backward conveyance of venous emboli ; and in this sense interprets an observation of thrombosis of the superior longitudinal sinus, with a plug in the right axillary vein identical in appearance with an undoubted embolus in the pulmonary artery. Von Recklinghausen has furnished evidence of the retrograde transport of infective emboli into the renal veins.

From these cases it is seen that retrograde embolism of particles of tumours, of tumour-cells, of parenchymatous cells, and of ordinary bland and infective thrombotic fragments has been observed. Experiments have demonstrated that, under certain conditions, light as well as heavy particles may be transported in the veins in a direction contrary to that of the normal blood-current. The veins in which retrograde embolism in

human beings has been found are the hepatic, the renal, the mesenteric, the pampiniform plexus, the coronary of the heart, the cerebral veins and sinuses, the axillary, and the pulmonary. Experimental retrograde embolism has been produced in many other veins, including those of the lower extremities. While venous valves, when intact, are undoubtedly a protection against this occurrence, they are often imperfectly developed or insufficient. Emboli have been repeatedly observed in the cerebral veins and sinuses which should be protected by valves in the jugular veins.

Retrograde embolism is usually explained by a temporary reflux of the venous current in consequence of some sudden obstacle to the return flow to the right heart, as may occur with forced expiration and coughing. Whatever increases the pressure in the veins near the heart, and impairs the assistance to the venous stream afforded by the respiratory movements and the suction of the right heart, favours this backward movement. Increased intrathoracic pressure, stenosis of the respiratory passages, spasm of respiratory muscles, distension of the right heart, tricuspid insufficiency, slowing of the heart's beats from vagus-irritation, are among the conditions believed to dispose to retrograde transport.

Ribbert does not accept the reflux theory of retrograde embolism ; partly for lack of any positive observation of such backward flow beyond the immediate neighbourhood of the right heart, and partly on account of the difficulty in explaining what becomes of all the blood which would be momentarily pressed back toward the capillaries. His explanation is that in conditions of high venous stasis, emboli, sticking loosely to the venous wall, are not moved forward by the feeble current, but are slowly pressed backward, step by step, by pulse-waves in the veins. For this view he finds support in experiments which he has made. Observations, partly experimental, of Arnold and of Ernst, cannot readily be reconciled with Ribbert's explanation ; so that, notwithstanding difficulties needing further elucidation, the reflux theory seems at present the more probable for most cases.

Of a different nature from the preceding form of retrograde transport is the conveyance of emboli by a blood-current reversed from its normal direction in consequence of obstruction of veins by compression or other causes. This kind of retrograde transport from more or less permanent reversal of the normal current is far more frequent in lymphatic vessels than in veins, and plays an important part in the metastases of tumours by means of the lymphatics.

Anatomical Characters.—The appearances observed in embolised vessels vary with the shape, size, consistence, and nature of the embolus, and the duration of its impaction. Approximately spherical emboli, as a rule, completely close the lumen of the artery in which they lodge. Cylindrical, elongated, or flat emboli are usually caught as riders at an arterial bifurcation ; and often at first leave more or less of the channels by their side open. Thrombi several inches long may be washed out of the femoral or other peripheral vein. Such a transported thrombus may be found in the trunk or a primary division of the pulmonary artery,

folded two, three, or even four times upon itself, and pressed at different points into several of the main arterial branches at the hilum of the lung, as in an interesting case described by Fagge. In this way an embolus may completely plug a vessel three or four times its diameter. Irregularly-shaped emboli, if of soft consistence, may be pressed into an artery so as to block the lumen completely; but if of firmer consistence they leave at first some space for the blood to flow. Emboli may be of such consistence as to be shattered by impact with the arterial wall, the fragments blocking many or all of the small branches, and producing the same effect as if the plug had been arrested in the main trunk.

An embolus is the starting-point of a secondary thrombus which usually, although not always, completes the closure of the vessel, if this was not effected by the embolus itself, and extends on each side to the nearest branch. The same metamorphoses and process of organisation, with consecutive changes in the vascular wall, occur with emboli and encapsulating thrombi, as described in the previous article for primary thrombi. Non-absorbable emboli or parts of emboli, like foreign bodies, are encapsulated by cells and tissue.

In cases of recent embolism, the plug can generally be recognised as an embolus without much difficulty; but, in those of long standing, the anatomical diagnosis between embolism and thrombosis may be difficult, or even impossible. The criteria for the recognition of a fresh embolus are for the most part sufficiently self-evident. Such a plug lies loosely or is but slightly adherent to the vessel-wall. It often presents a broken or fractured surface which, in fortunate cases, may be made to fit on the corresponding surface of the thrombus from which it was originally broken off. It may be bent or folded, or shew the marks of venous valves, or present ramifications which do not correspond to those of the artery in which it lies. It is, of course, of the first importance to find, if possible, the source of the embolus; and, when this is done, to make a careful comparison between the thrombus and the embolic fragment as to resemblances in structure and appearance.

After the embolus has become adherent and surrounded by a secondary thrombus, some of these differential criteria may still remain for a while; but, as time passes, the anatomical diagnosis becomes increasingly difficult. The embolus may perhaps still be distinguished from the surrounding thrombus by marked differences in its age and general appearance and structure, possibly by the presence of lime salts. An adherent plug which rides an arterial bifurcation is much more likely to be an embolus than a primary thrombus. In reaching a conclusion, weight must be given to the condition of the arterial wall; whether there be any local cause for thrombosis,—such as compression, aneurysm, arteriosclerosis; and whether the microscope shews such secondary changes in the arterial wall as generally correspond to the apparent age and character of the adherent plug. The detection of a source for an embolus will be an important consideration. The clinical history may aid in the anatomical diagnosis; and all attendant circumstances, especially the existence else-

where of undoubted emboli, should be taken into consideration. In some situations, as in branches of the renal or splenic arteries, primary thrombosis is so uncommon that the chances are all in favour of embolism.

It is evident from what has been said that in the older cases the anatomical diagnosis must often be based upon a weighing of probabilities, and that sometimes a positive conclusion cannot be reached.

Effects.—Bland or aseptic emboli produce chiefly mechanical effects referable to the obstruction to the circulation ; toxic or infective emboli cause also other changes which may be described as chemical or infective. We shall consider first the mechanical effects.

The direct injury which may be inflicted upon the vessel-wall by sharp calcareous emboli is, according to Ponfick, a rare cause of aneurysm. Embolic aneurysms, however, stand in much more definite relation to chemical properties of the embolus, as will be shewn subsequently (p. 785).

Necrosis ; Infarction.—The fate of a part supplied by an artery closed by a bland embolus depends altogether upon whether it is fed within a certain time after the obstruction with enough arterial blood to preserve its function and integrity. An embolus which does not completely plug the vessel may not cause any appreciable interference with the circulation ; but the closure of the lumen is usually soon effected by a secondary thrombus. The occlusion by a bland embolus of an artery with abundant anastomoses, such as those possessed by the arteries supplying bone, the voluntary muscles, the skin, the thyroid, the uterus, usually causes no circulatory disturbance of any consequence. Even in these situations extensive multiple embolism, or embolism with extensive secondary thrombosis, may cause local anaemia with its consequences.

Sudden death may be the result of embolism of the trunk or a main division of the pulmonary artery, of one of the coronary arteries of the heart, or of the bulbar arteries.

If an adequate collateral circulation be not established within the proper time, the inevitable fate of a part, supplied by an embolised artery, is degeneration or death. Local death is the regular result of embolism of branches of the splenic artery, the renal artery, the basal arteries of the brain, the central artery of the retina, and the main trunk of the superior mesenteric artery. It is the usual result of embolism of one of the coronary arteries of the heart, if the patient survive long enough ; and it is the inconstant result, depending generally upon accessory circumstances, of embolism of the medium-sized and smaller branches of the pulmonary arteries, of cerebral arteries other than the basal, of the abdominal aorta, iliacs, main arteries of the extremities, and some other arteries. A collateral circulation may be established sufficiently to preserve the life of a part, but not to maintain its full nutrition ; in these circumstances it undergoes fatty degeneration or simple atrophy.

When the dead part is so surrounded with living tissue that it can be permeated with lymph, as is usually the case in the viscera, the mode of death is that described by Weigert, and named by Cohnheim, "coagulative necrosis." Here the dead protoplasm, and to some extent inter-

cellular substances, undergo chemical changes, believed to be in part coagulative ; and actual fibrillated fibrin may appear. If there be enough coagulable material present, the necrotic part becomes hard, dry, opaque, and somewhat swollen. For a time its general architecture, both gross and microscopic, is preserved ; but the nuclei and specific granulations disappear early, the former largely by caryorrhexis.

An area of coagulative necrosis resulting from shutting off of the blood-supply is an infarct. Its shape corresponds to that of the arterial tree supplying it, and is, therefore, as a rule, approximately conical, or that of a wedge, the base being toward the periphery of the organ. The wedge-shape is most marked in smaller infarcts ; large ones may be roundish or irregular in shape. The size depends upon that of the occluded artery. The colour is opaque, white, or yellowish, unless haemorrhage is added to the necrosis. We thus distinguish anaemic, pale, or white infarcts, and red or haemorrhagic infarcts ; but, in the latter no less than in the former, the essential thing is the coagulative necrosis, the haemorrhage being merely something added to the necrosis. This was not always clearly recognised, it being supposed at one time that the haemorrhage was the characteristic feature of infarcts, and that pale infarcts were simply decolorised haemorrhagic infarcts. The name "infarct" (from *infarcire*, to stuff), like many other old medical terms, is therefore now used in a sense at variance with its etymological meaning. In some situations, as the kidney and the retina, the infarct is nearly always pale ; in others, as the lungs and the intestine, it is as constantly haemorrhagic ; and in yet others, as the spleen and the heart, it may be either white or red.

Where there is not a sufficient quantity of coagulable substance the area of coagulative necrosis does not become hard ; and it may be of much softer consistence than normal, as is the case with the ischaemic necroses of the brain and spinal cord. Necrosis of peripheral parts, as the toes, foot, leg, hand, is not of the coagulative variety ; for the dead part is not surrounded by living tissue to furnish the lymph which brings one of the factors essential for coagulation. This peripheral necrosis is called gangrene or mortification, and may be either dry or moist.

Collateral Circulation ; Local Anaemia.—As the state of the collateral circulation is the decisive factor in bland embolism, it becomes important to learn the conditions under which establishment or failure of this circulation occurs. This subject is one eminently open to experimental study ; but more attention has been given to the anatomical than to the physiological side. In fact many writers seem to assume that the physiological factors can be so readily deduced from the laws of hydro-dynamics that it is only necessary to investigate the size, arrangement, and distribution of the vascular tubes. Nevertheless experience has shewn abundantly the danger of accepting anything in the physics of the circulation which has not been put to an experimental test on the living body. The experimental study of the physiological conditions which determine the development of a collateral circulation has demon-

strated that this problem is by no means so simple as has been often represented; whilst some old errors have been corrected and new facts have been added, we are still far from an entirely satisfactory solution or any definite agreement of opinion. It is impossible here to do more than touch upon certain points bearing directly upon the subject in hand.

If an artery with slender anastomoses to its area of distribution, such as the femoral or the lingual in a frog's tongue, be tied, the immediate effect is stoppage of the circulation and anaemia of the part supplied by the occluded vessel, accompanied by contraction of the artery below the obstruction. Almost immediately, or within a short time, the blood begins to flow with greatly increased velocity through arteries arising above the point of ligation, but more rapidly only through those which send blood by anastomosing channels to the anaemic part. At the same time these arteries with quickened flow dilate. Formerly this vascular dilatation and increased flow were attributed to rise of blood-pressure above the ligature, but experiments have shewn that in most situations this is a factor of relatively little moment. The rise of pressure cannot of course remain localised, and after ligation of the femoral artery amounts at most to only a few millimetres of mercury. Evidence of the relatively slight importance of this increased pressure is that the ligated artery actually contracts from the point of ligation to the first branch arising above the ligature (Thoma, Goldenblum); and that the phenomena of dilatation and increased velocity occur only in arteries which send blood to the anaemic area, although others which carry blood elsewhere may arise nearer to the point of obstruction (Nothnagel). Moreover, it is hardly conceivable that increased pressure above the ligature can persist for the days and weeks which may be necessary for the full development of the collateral circulation.

As the increased flow cannot be due to any change in the viscosity of the blood, it must be due to increase of the pressure-gradient. Therefore, if it is not the result in any marked degree of rise of pressure above the obstruction, it must be caused by lowered resistance to the stream in the anastomosing vessels. A moment's reflection will shew that this is a far more purposeful and better mode of compensation than one brought about exclusively by a rise of pressure which must act upon arteries in no way concerned in the collateral circulation. The difficulty is an entirely satisfactory and complete explanation of the lowered resistance. It seems impossible that it can be due to anything but a widening of the bed of the stream. Von Recklinghausen has pointed out that the stream-bed for the anastomosing arteries is enlarged, inasmuch as after occlusion of the main artery the blood can flow from these collaterals not only in its original bed, but also, with diminished resistance, into the stream-bed belonging to the closed artery. The pressure-gradient is thus increased, and consequently the velocity of the current is quickened in the anastomosing arteries. The cause of the dilatation of these arteries is not so clear. Thoma states as his first

histo-mechanical principle that increased velocity of the blood-current leads to widening of the lumen, and eventually, if the increase continues, to growth of the vessel-wall in superficies. Admitting this to be true, it can hardly be considered an explanation. As the collateral circulation develops perfectly, and with the same phenomena, after severance of all connexion of the part with the central nervous system, it is evident that vasomotor influences which are under central control are not essential to the process.

Satisfactory as von Recklinghausen's explanation is, as far as it goes, there is evidence that it does not cover all of the facts, and that there is also some mechanism by which the vessels of an ischaemic part are opened wide for the reception of the needed arterial blood. The existence of such a mechanism has been recognised by Lister, Cohnheim, Bier, and others. I must refer especially to Bier's papers for a full presentation of the evidence on this point, and shall merely mention, as a familiar illustration, the extreme arterial hyperaemia which follows the removal of an Esmarch bandage. This flushing of a previously ischaemic part with arterial blood has been usually attributed to paralysis of vaso-constrictor or stimulation of vaso-dilator nerves, but Bier has shewn that it occurs under conditions where this explanation can be probably excluded.

Without following Bier in his somewhat vitalistic conceptions, or speculating regarding the explanation of the phenomenon, we must, I think, admit that deprivation of arterial blood sets up some condition of a part whereby the vessels which feed it are in some way dilated to receive any fresh arterial blood which can reach them. The existence of such an admirably adaptive, self-regulatory capacity must be an important element in the development of a collateral circulation, and it may be remarked that it is a physiological rather than an anatomical factor. Bier believes that this capacity is very unequally developed in different parts of the body; being highest in external parts, and feeble or absent in most of the viscera. He is also of the opinion that the arterioles and capillaries of external parts have the power, by independent contractions, of driving blood into the veins; and that, by contraction of the small veins, the capillaries of these parts are in large measure protected from the reception of venous blood.

A possible, but I think not fully demonstrated, variation in the power to lower the resistance to the collateral stream of arterial blood is not, however, the only physiological property which influences the varying effects following obstruction to the arterial supply of different parts of the body. In some situations there are physiological arrangements which seem calculated to increase the difficulty of establishing an adequate collateral circulation. Mall has shewn that contraction of the intestine exerts a marked influence upon the circulation through this organ. In the light of his results, it is interesting to note that, immediately after closure of the main trunk of the superior mesenteric artery of a dog, the intestine is thrown into violent tonic contractions and remains in an anaemic, contracted condition for two or three hours; after

which the spasm relaxes and the bloodless condition at once gives place to venous hyperaemia and haemorrhagic infarction, which appears in the third to sixth hour after the occlusion of the artery (Mall and Welch). This intestinal contraction, which in these circumstances is equivalent to arterial spasm, is probably one, although not the sole, reason why, in spite of free anastomoses, occlusion of the arteries supplying the intestine is followed by necrosis and haemorrhage. That the explanation is not to be found simply in the great length of intestine supplied by a single artery, is evident from the fact that, if the extra-intestinal arteries supplying a loop much more than 5 centimetres in length be suddenly closed, the loop becomes haemorrhagic and necrotic (Mall and Welch, Bier). That the conditions are essentially identical in man is proven by the experience of surgeons, who have repeatedly observed the same results after separation of the mesentery close to the intestine over about the same length. The blood can enter at each end of the short-loop arteries, whose branches anastomose freely within the walls of the loop with those of the closed arteries; there being a particularly rich arterial plexus in the submucous coat (Heller). But these anastomoses are insufficient to preserve the part; although, with reference to the extent of territory to be supplied, they are large in comparison with some of the trivial anastomoses which in external parts can respond effectively to the call for a collateral circulation to far larger areas. It must be left to future investigations to determine how far the inability of the intestinal vessels to compensate circulatory obstructions of a degree readily compensated in many other situations may be due, as claimed by Bier, to an inherent incapacity to lessen the resistance to the collateral stream, or to contraction of the muscular coats of the intestine, or to other causes. As Panski and Thoma have shewn that slowing and interruption of the circulation in the spleen are followed, for several hours, by contraction of the muscular trabeculae, it is probable that the development of a collateral circulation in this organ meets an obstacle similar to that in the intestine.

The various organs and tissues differ so widely as regards their susceptibility to the injurious effects of lack of arterial blood that local anaemias of equal intensity and duration may in one part of the body produce no appreciable effect, and in another cause the immediate abolition of function and the inevitable death of the part. In general, the more highly differentiated, specific cells of an organ are those which suffer first and most intensely. At one end of the scale are the ganglion-cells of the brain, which, after the withdrawal of arterial blood for half an hour, and probably for a much shorter time, cannot be restored to life; and at the other end may be placed the periosteum, the cells of which may be still capable of producing bone two or three days after all circulation has ceased. So susceptible to local anaemia are the ganglion-cells of the central nervous system, that not only is embolism of the branches of the cerebral arteries with only capillary communications, even of the minute terminal twigs in the cortex, always

followed by necrotic softening, but also embolism of the anastomosing arteries in the pia very often causes softening of at least a part of the area supplied by the plugged artery.   In the well-known Stenson experiment, temporary closure of the rabbit's abdominal aorta, just below the origin of the renal arteries, for an hour, results in the inevitable death of the ganglion-cells in the central grey matter of the lumbar cord ; and this notwithstanding the free anastomoses of the anterior and posterior spinal arteries.   Many of the lesions which pass under the names of myelitis and haemorrhagic encephalitis present the histological characters of ischaemic necrosis, although often no arterial occlusion can be found.

Perhaps, next to elements of the nervous system, the epithelial cells of the cortical tubules of the kidney are most susceptible to ischaemia. Litten has demonstrated that the temporary ligation of the renal artery of the rabbit for one and a half to two hours is followed invariably by necrosis of many of these epithelial cells.   The cells in the walls of the blood-vessels and of connective tissue are relatively insusceptible to temporary slowing or cessation of the circulation.

It is evident from the preceding statements that the nature of the organ or tissue has a very important influence in determining whether local necrosis follows arterial embolism.

I have dwelt in some detail, although within the limited space necessarily inadequately, upon certain physiological characters of the circulation and of different organs and tissues, which appear to me deserving of more consideration than is usually given to them in discussions of the causes of embolic necroses and infarctions.   It is, of course, not to be inferred that the number and size of the anastomoses are not of prime importance in determining the mechanical effects of arterial embolism, but, important as they are, they are not the exclusive determinants of the result.   There is no single anatomical formula applicable to the circulatory conditions under which all embolic enfarcts occur.   The nearest approach to such a formula is that embodied in Cohnheim's doctrine of terminal arteries, a name which he gave to arteries whose branches do not communicate with each other or with those of other arteries, although capillaries are of course everywhere in communication with each other.   Terminal vessels in this sense are the renal, the splenic, the pulmonary, the central artery of the retina, the basal arteries of the brain, and in general all branches of cerebral and spinal arteries after they have penetrated the brain or the spinal cord, the intramuscular branches of the coronary arteries of the heart, and the portal vein.[1]   Cohnheim's teaching was that infarction occurs

[1] There is some confusion as to the sense in which the words "terminal arteries" should be used, and it must be admitted that later investigations have detracted from the precision given to this term by Cohnheim.   Thus some do not recognise the pulmonary artery as terminal, because the lung is supplied likewise by the bronchial and several other arteries whose capillaries communicate with those of the pulmonary artery.   But unless we make the extent of a second arterial supply the decisive point in the definition, we should have, for the same reason, to exclude the renal and the splenic arteries from the class of "terminal arteries."   Then the conception of arteries which are "functionally" but not anatomically terminal, creates still further confusion.

always after embolism of a terminal vessel, except of the pulmonary artery, whose capillaries, under ordinary conditions, are numerous and wide enough, after obliteration of an arterial branch, to maintain a sufficient circulation ; and of the portal vein, whose capillaries communicate freely with those of the hepatic artery. Thoma and Goldenblum have shewn that, contrary to Cohnheim's results, no infarction follows embolism or ligature of the frog's lingual artery, which is or can readily be made a terminal artery, provided the tongue be replaced in the mouth after the operation so as to avoid stretching and drying from exposure to the air. It is, therefore, quite possible in some situations for an adequate circulation to be carried on through merely capillary communications, although the conditions are of course less favourable than when there are arterial anastomoses. On the other hand, as we have seen, embolism of anastomosing arteries, such as the mesenteric and the cerebral, may be followed by necrosis or infarction ; and it cannot be said that the anastomoses in all of these cases are so unimportant that the arteries are virtually terminal.

We may conclude then that, under ordinary conditions, embolism of an artery having abundant and large anastomoses has no important mechanical effect; that embolism of an artery with few and minute anastomoses, especially embolism of an artery with only capillary communications, is in many situations followed by necrosis, this result being favoured by certain physiological conditions, which have been considered ; and that embolism of arteries with fairly well developed anastomoses may in certain situations also cause necrosis. Among the factors influencing the result, other than those relating to the number and size of the anastomoses, are the varying susceptibility of cells to ischaemia, interference with the circulation by contraction of muscular constituents of a part, and perhaps some inherent weakness in the physiological part of the mechanism by which a vigorous collateral circulation is established.

The compensation of sudden occlusion of an artery, by means of the collateral circulation, generally presupposes vessels with fairly normal walls and a certain vigour of the circulation. When the arteries have lost their elasticity, or the general circulation is feeble, or there is some pre-existing obstacle to the circulation such as chronic passive congestion, the development of an adequate collateral circulation is rendered correspondingly difficult, and may be impossible. Hence embolism of arteries of the extremities is often followed by gangrene in the aged, in arteriosclerosis, in heart disease, and in infective, anaemic, and exhausting diseases. There are some observations which suggest that arterial spasm may co-operate with embolism in causing local anaemia.

The agencies by which a sufficient collateral circulation is established may be thrown out of order to such a degree that embolism of arteries having even the most ample anastomoses may be followed by necrosis. Foci of cerebral softening have been observed after occlusion of the internal carotid or of one of the vertebral arteries, although the

circle of Willis, the largest and most perfect anastomosis in the body, was open, and no vascular obstruction could be found beyond it. Here, doubtless, an important factor in this exceptional occurrence is the rapidity with which nerve-cells die when insufficiently fed with arterial blood. Cohn narrates the interesting case of a young woman rendered extremely anaemic by repeated haemorrhages from cancer of the tongue. In order to control the bleeding the right carotid was tied. The patient immediately, to all appearances, lost consciousness; acquired ptosis of the right, then of the left eye, drawing of the angle of the mouth to the right, and relaxation and almost complete paralysis of the left extremities. The pulse almost disappeared and the face became very anaemic. Respiration was unaffected. The ligature was at once removed, and at the same moment the patient awoke "as from a dream," and the symptoms just mentioned quickly disappeared. She said that she had not completely lost consciousness but was unable to speak, and that her will had lost control over the organs. She had lost so much blood that she died three hours later without again losing consciousness before death. At the necropsy the carotids and all of the cerebral vessels were found open, and there was no change in the brain except anaemia. In this case, the general anaemia was evidently so great that after closure of one carotid, which probably lasted not more than a minute or two, a sufficient supply of blood could not reach the brain through the circle of Willis.

Haemorrhagic Infarction.—The explanation of the accumulation and extravasation of blood in haemorrhagic infarcts has been the subject of much speculation and experimental study. It is only in certain situations that infarcts are haemorrhagic throughout; and, as already mentioned, these are no less necrotic than are the white infarcts. The necrosis and the haemorrhage are co-ordinate effects of the disturbance of the circulation, neither being caused by the other. Virchow, in his early writings, suggested as possibilities, without definitely adopting any of them, most of the explanations which have since been advanced to account for the apparently paradoxical phenomenon that the occlusion of an artery may be followed by hyperaemia and haemorrhage in the area of its distribution. Cohnheim, on the basis of experimental investigations published in 1872, came to the conclusion that the hyperaemia which may follow arterial embolism is the result of regurgitant flow from the veins, that the haemorrhage occurs by diapedesis, and that this diapedesis is the result of some molecular change in the vascular walls deprived of their normal supply of nutriment. Although Cohn, in 1860, had shewn conclusively, by numerous experiments on various organs, that the hyperaemia and haemorrhage are not the result of regurgitant flow from the veins, Cohnheim's views were widely accepted until Litten, in 1880, in apparent ignorance of Cohn's work, repeated the experiments of the latter upon this point with the same results. The experiments of Dr. Mall and myself upon haemorrhagic infarction of the intestine in 1887 convinced us that the blood which causes the infarct is not regurgitated

from the veins. Cohnheim's results upon the frog as to the source of the blood in infarcts have not been confirmed by subsequent experimenters (Zielonko, Kossuchin, Küttner, Goldenblum, Thoma).

In situations where closure of an artery is followed by haemorrhagic infarction, tying the veins also, so as to shut off all opportunity for reflux of venous blood, increases the hyperaemia and the haemorrhage ; and it may render haemorrhagic an infarct which would otherwise be anaemic. On the other hand, if all vascular communication of a part be cut off except that with the veins, the part undergoes simple necrosis without haemorrhagic infarction ; and the result is the same even if the artery be cut open, so as to afford apparently the most favourable opportunity for backward flow from the veins. Or, expressed differently, if after closure of an artery all possibility of access of blood to the obstructed area through anastomosing arteries and capillaries be prevented, the veins remaining open, the part dies without haemorrhagic infarction. Cohnheim was in error in supposing that haemorrhagic infarction cannot occur where the veins are provided with valves, for it has been shewn by Bryant, Köppe, and Mall that the small intestinal veins of the dog have effective valves ; yet nowhere can haemorrhagic infarction be more readily produced experimentally by arterial obstruction than in the intestine of this animal. It is, then, quite certain that the blood which accumulates in the capillaries and small veins, and is extravasated in haemorrhagic infarction, comes in through the capillary, and, if they exist, the arterial anastomoses, and is not regurgitated from the veins.

It cannot be doubted that the red corpuscles escape by diapedesis, not by rhexis ; but our experiments are in entire accord with those of Litten in failing to furnish any support to the prevalent doctrine that the haemorrhage is the result of changes in the walls of the vessels caused by insufficient supply of arterial blood ; in fact they seem to us more conclusive upon this point. If a loop of intestine be completely shut off from the circulation for three or four hours (by which time, after ligation of the superior mesenteric artery, haemorrhagic infarction begins to appear), and the obstruction be then removed, the blood at once shoots in from the arteries with great rapidity, and distends the vessels.[1] If, as usually happens, the blood has not coagulated in the vessels, no haemorrhagic infarction subsequently appears. If, immediately after the circulation has been fully re-established in the loop, the superior mesenteric artery be ligated, the intestine from the lower part of the duodenum into the colon becomes the seat of haemorrhagic infarction in the usual time ; but the infarction does not appear earlier and is not more intense in the part which had been previously deprived of its circulation for three or four hours than in the rest of the small intestine. It is true, as Cohnheim has shewn, that re-establishment of

---

[1] Bier's experimental results concerning the absence of hyperaemia after temporary ischaemia of the intestine do not, according to our experience, apply to prolonged ischaemia, which we found to be followed by intense hyperaemia.

a local circulation, after its stoppage for many hours or days, may be followed by haemorrhages in the previously ischaemic area; but haemorrhagic infarction after arterial occlusion begins long before it is possible to demonstrate this change in the vascular wall caused by lack of blood-supply.

In the part undergoing haemorrhagic infarction the circulation is greatly retarded in consequence of the small difference between the arterial and the venous pressures. This result may be brought about by rise of the venous or lowering of the arterial pressure. If the veins are obstructed sufficiently to render the outflow nil, or very small, and the arteries are open, the infarction is intense, and occurs with high intracapillary pressure. In consequence of the free anastomoses of veins this mode of production of an infarct is rare, but it may occur after thrombosis of the mesenteric, the splenic, and the central retinal veins. Its explanation offers no special difficulties. If the veins are open the arterial pressure must be reduced in order to furnish the conditions necessary for the production of haemorrhagic infarction. This latter case is the one present in arterial embolism with haemorrhagic infarction, and is the one especially needing explanation. The intracapillary pressure in this case may vary, but will generally be low. The arterial pressure is so low that the lateral pulse-waves nearly or entirely disappear, so that the force which drives the blood into the capillaries is no longer the normal intermittent one, which experiment has shewn to be essential for the long-continued circulation of the blood through the capillaries and veins. This reduction, or absence of lateral pulsation, to which, so far as I know, other experimenters have not called attention, I believe to be the factor of first importance in the causation of haemorrhagic infarction following arterial embolism.

We are not sufficiently informed concerning the physical and vital properties of the blood and of the blood-vessels to be able to predict positively what would happen under such abnormal circulatory conditions as those named, and actual observation only can furnish a solution. The difficulties in making such observations under the requisite conditions are considerable. Dr. Mall and I, in examining microscopically, in a specially constructed apparatus, the mesenteric circulation of the dog after ligation of the superior mesenteric artery, observed that immediately after the occlusion the circulation ceases in the arteries, capillaries, and veins. In a short time the circulation returns, but with altered characters. The arteries are contracted, but may subsequently dilate somewhat; and the blood from the collaterals flows through them with diminished rapidity, and without distinct lateral pulsation. The direction of the current is reversed in some of the arteries. The movement of the blood in the capillaries and veins is sluggish and irregular. The direction of the current in some of the veins may be temporarily reversed, but we were unable to trace a regurgitant venous flow into the capillaries. The distinction between axial and plasmatic current is obliterated. Gradually the smaller and then

the larger veins become more and more distended with red corpuscles, and all of the phenomena of an intense venous hyperaemia appear, so that one instinctively searches for some obstruction to the venous outflow. The red corpuscles in the veins tend to accumulate in clumps, and may be moved forward, or forward and backward, in clumps or solid columns. Stasis appears in the veins. This is at first observed only here and there, and is readily broken up by an advancing column of blood ; but it gradually involves more and more of the veins, and in some becomes permanent, producing an evident obstacle to the forward movement of the blood. The same phenomena of distension with red corpuscles, clumping, to-and-fro movement, and stasis appear gradually in the capillaries. An interesting appearance, sometimes observed in capillaries and veins, is that of interrupted columns of compacted red corpuscles with intervening clear spaces which are sometimes clumps of white corpuscles, sometimes of platelets, sometimes only clear plasma. With the partial blocking of the veins and capillaries, red corpuscles begin to pass through the walls of these vessels by diapedesis ; and after a time the haemorrhage becomes so great that it is difficult to observe the condition within the vessels. The venous outflow is diminished immediately or shortly after the closure of the superior mesenteric artery : it then rises, but later it continuously falls to a minimum.

An experiment which we made shews that the blood for haemorrhagic infarction need not necessarily enter from the collaterals, and it sheds some light upon the condition of the circulation during the production of the infarct. We ligated all of the vascular communications of the intestine, with the exception of the main artery and vein, and then tied the intestine above and below, so that the included intestine was supplied only by the main artery and the blood returned by the main vein. In these circumstances no infarction results. We then by a special device gradually constricted the main artery. In repeated experiments we found that not until the artery is sufficiently compressed to stop the lateral pulsation in its branches—the pressure in these being then about one-fifth of the normal—does haemorrhagic infarction appear. Precautions were taken to make sure that the flow through the constricted main artery and its branches continued, and that the vein remained open. We have often measured the blood-pressure in branches of the superior mesenteric artery after ligation of this artery and during the progress of an infarction, and have found it to be generally one-fourth to one-fifth of the normal pressure. If the pressure on the arterial side falls below a certain minimum no haemorrhage occurs in the infarction.

It is evident from the preceding description that the phenomena observed under these peculiar circulatory conditions are in large part dependent upon the physical properties of the blood, especially upon its viscosity and the presence of suspended particles which readily stick together ; and differ in important respects from those which would occur under similar conditions with a thin, homogeneous fluid. The pressure-gradient from arteries to veins of the ischaemic area is so low that the

red corpuscles cannot fully overcome the resistance in the veins and capillaries. They accumulate in these situations, and probably undergo some physical change by which they become adherent to each other and to the vascular wall. The absence of the normal pulse-waves prevents the breaking up of these masses of corpuscles, the longitudinal pulse-waves sometimes observed having little or no effect in disintegrating the masses. In this way numerous small veins and capillaries become blocked, with a resulting rise of intracapillary pressure and diminution of outflow of blood through the veins. Von Frey has shewn by interesting experiments that an intermittent pulsating force is necessary to prevent the speedy blocking of veins and capillaries with red corpuscles in carrying on artificial circulation with defibrinated blood through living organs. Kronecker has also demonstrated the influence of a pulsating force in increasing the venous outflow.

The diapedesis is due to the slowing and stagnation of the blood, and to the blood-pressure. Without a certain height of pressure there is no diapedesis; and, with a given retardation and stasis of the blood-current, the higher the intracapillary and intravenous pressure the greater the amount of diapedesis. The matter which needs explanation is that the diapedesis may occur with lower than the normal pressure, and through vessel-walls apparently unaltered. This I attribute to the fact that the red corpuscles, in consequence of the slow circulation, have opportunity to become engaged in the narrow paths followed by the lymph as it passes out between the endothelial cells. Diapedesis is a slow process, and the channels for it are much smaller than the thickness of a red corpuscle. Unless the red corpuscles can get started on the path between the endothelial cells, they cannot traverse it; and unless the circulation is very much slowed, and the outer plasmatic current obliterated, there is no opportunity for the corpuscles to become engaged between the endothelial cells, provided, that is, the vascular wall be normal. With greatly retarded circulation there is opportunity, and when the way in front is blocked by compact masses of red corpuscles, and sometimes by actual thrombi, the only path open to the corpuscles is that followed by the lymph between the endothelial cells. This then becomes the direction of least resistance for their movement.

The reason why infarctions are haemorrhagic in some situations and not in others offers difficulties chiefly in consequence of our ignorance of the exact circulatory conditions which lead to the production of infarction in different parts of the body. It is generally assumed that these circulatory conditions are everywhere essentially the same; but this is by no means proven. As we have already seen, the physiological conditions which influence the result are various. It may be, therefore, that the requisite intracapillary and intravenous pressure, or some other condition of the circulation essential for the production of haemorrhagic infarction, is lacking when the infarction is anaemic. In general a high venous pressure favours haemorrhage in an infarction, and a low arterial pressure opposes it. The pressure in the superior mesenteric and portal

veins is higher than in any other veins of the body. Haemorrhagic infarction of the lung occurs especially with high degrees of chronic passive congestion in which the venous pressure is elevated. Thrombosis of veins seems to be the cause of at least some of the haemorrhagic infarcts of the spleen. Haemorrhagic infarction of the kidney may be produced experimentally by ligating the renal veins.

The studies of recent years upon the formation of lymph have demonstrated that the blood-vessels in different regions differ markedly in their permeability, those of the liver being probably the most permeable. It may be that this difference in the constitution of the vessels is an important factor in determining the extent of diapedesis under similar circulatory conditions. As pointed out by Weigert, however, the greatest influence appears to be exercised by the resistance offered by the tissues to the escape of red corpuscles from the vessels. Haemorrhagic infarction occurs especially where this tissue-resistance is low, as in the loose, spongy texture of the lungs, and in the soft mucosa and lax submucosa of the intestine. The haemorrhage is far less in the dense muscular coats of the intestine. The considerable resistance offered by the naturally firm consistence of the kidney is increased by the swelling and hardness resulting from coagulative necrosis of the epithelial and other cells of this organ; so that infarcts in this situation are nearly always anaemic in the greater part of their extent, although often haemorrhagic in the periphery. The spleen is of softer consistence than the kidney; and here both white and red infarcts may occur, the latter especially with increased venous pressure. Although infarcts of the brain are soft, they are much swollen in the fresh state from infiltration with serum, so as to displace surrounding parts (Marchand). Here also there must be considerable resistance to the passage of red corpuscles through the vascular walls; but it is not uncommon for these softened areas to present scattered foci of haemorrhage, and sometimes they are markedly haemorrhagic. The intra-ocular pressure is probably a factor in making embolic infarcts of the retina anaemic. Embolism of arteries of the extremities with insufficient collateral circulation is often associated with extravasations of blood in the ischaemic areas.

Metamorphoses of Infarcts.—A bland infarct is a foreign body most of the constituents of which are capable of absorption and replacement by connective tissue. The red corpuscles lose their colouring matter, some of which is transformed into amorphous or crystalline haematoidin. Polynuclear leucocytes, through chemiotactic influences, wander in from the periphery, the advance guard being usually the seat of marked nuclear fragmentation. This nuclear detritus mingles with that derived from the dead cells of the part. The necrotic tissue undergoes digestion by intra-cellular enzymes (proteases) which have not been destroyed when the cells underwent necrosis. In sterile infarcts the digestion is mainly carried on by the intracellular enzymes of the necrotic tissues (autolysis), whereas in infective infarcts the digestion is largely effected by the enzymes of leucocytes which have wandered into the infarcted area (heterolysis,

Jacoby). The autolysis of bland infarcts is, according to Wells, due to enzymes derived from the cytoplasm and not from the nuclei of the necrosed cells, and is a much slower process than the heterolysis due to the leucocytic invasion seen in infective infarcts. The removal of the necrotic tissue is carried out by invading leucocytes. Young mesoblastic cells wander in. Granulation tissue develops from the living tissue around the infarct. At the margin of the infarct there is usually more or less fat (Fischler). This is probably due to persistence of the cell lipase which acts upon the fatty acid and glycerin diffusing into the necrotic area with the plasma (Wells). In the course of time new vessels and new connective tissue grow in; and finally a scar, more or less pigmented according to the previous content of blood, marks the site of the infarct. In chronic endocarditis, depressed, wedge-shaped scars are often found in the spleen and the kidneys. They are rare in the lungs, not because haemorrhagic infarcts in this situation usually undergo resolution like pneumonia or simple haemorrhages, but because pulmonary infarcts generally occur under conditions not compatible with the prolonged survival of the patient. Partly organised infarcts are not uncommon in the lungs. In the brain, ischaemic softening may remain for a long time with apparently little change; but the common ultimate result is a cyst-like structure, which may be more or less pigmented, and is characterised by a meshwork of delicate neuroglia and connective-tissue fibres, infiltrated with milky or clear serum. Into the finer histological details of the process of substitution of an infarct by scar-tissue it is not necessary here to enter.

Metastases. Chemical Effects.—Embolism and metastasis are sometimes employed as practically synonymous terms; but, in ordinary usage, by metastasis is understood any local, morbid condition produced by the transportation of pathological material by the lymphatic or blood-current from one part of the body to another.

We have already considered the coarser bland emboli in respect of their mechanical effects. Similar emboli, so small as to become lodged only in arterioles or capillaries, produce no mechanical effects unless, as rarely happens, numerous arterioles or capillaries are obstructed. The subject of transportation of pigment granules, and that of metallic and carboniferous dust, producing the various conioses, does not fall within the scope of this article. On account of certain special features, emboli of air, of fat, and of parenchyma-cells are most conveniently considered separately (p. 789 *et seq.*). There remain, in contrast to the dead and inert emboli to which our attention has been especially directed, those containing tumour-cells and parasitic organisms, or their products.

Masses of a tumour growing into a blood-vessel may be broken off and transported as coarse emboli, producing all of the mechanical effects which we have described. There have been instances of sudden death from blocking of the pulmonary artery by cancerous or sarcomatous emboli, as in a case reported by Feltz. It is, however, as a cause of metastatic growths that emboli of tumour-cells have their chief significance.

In individual cases it is oftener a matter of faith than of demonstration that the metastasis is due to such emboli, for opportunities to bring absolutely conclusive proof of this mode of origin of secondary tumours are not common. There have, however, been enough instances in which the demonstration has been sufficient to establish firmly the doctrine of the embolic origin of metastatic tumours. The evidence is that tumour-metastases are far more frequently due to capillary emboli than to those of larger size. Cancers and sarcomas furnish the great majority of emboli of this class ; but in rare instances even benign tumours may penetrate blood-vessels and give rise to emboli, which exceptionally are the starting-points of secondary growths of the same nature as the primary. Mention has already been made of paradoxical and retrograde transport of tumour-emboli, as well as of the possibility of emboli of tumour-cells being so small as to traverse the pulmonary capillaries (p. 765 *et seq.*).

Certain animal parasites, as the *Filaria sanguinis*, *Schistosomum haemato-bium*, and the malarial parasite, are inhabitants of the blood, or, in certain stages of their existence within the human body, are frequently found there. According to observations of Cerfontaine and Askanazy, the usual mode of transportation of the embryos of *Trichinella spiralis* from the intestine is by the lymphatic and blood currents. Echinococci have been known to pass from the liver through the vena cava ; or primarily from the right heart into the pulmonary artery ; and emboli from echinococci present in the wall of the left heart may be transported to distant organs (Davaine). The *Entamoeba histolytica* has been found in the intestinal veins ; and may reach the liver through the portal vein.

On account of their frequency and serious consequences, infective emboli containing pathogenetic bacteria are of especial significance. Such emboli constitute an important means of distribution of infective agents from primary foci of infection to distant parts of the body, where the pathogenetic micro-organisms, by their multiplication and their chemical products, can continue to manifest their specific activities. These emboli are often derived from infective venous thrombi connected with some primary area of infection. The portal of infection may be through the integument, the alimentary canal, the respiratory tract, the genito-urinary passages, the middle ear, or the eye, with corresponding infective thrombophlebitis in these various situations. Or there may be no demonstrable atrium of infection, as in many cases of infective endocarditis, which constitutes an important source of infective emboli. Emboli may of course come from secondary and subsequent foci of infection.

Coarse emboli are by no means essential for the causation of infective metastases, nor is it necessary that there should be any thrombosis to afford opportunity for the distribution of micro-organisms from a primary focus. Bacteria may gain access to the circulation, singly or in clumps; and such bacteria, without being enclosed in plugs of even capillary size, may become attached to the walls of capillaries and small vessels and produce local metastases. In this way infective material coming from the systemic veins may pass through the pulmonary capillaries

without damage to the lungs, and become localised in various organs of the body.

We cannot explain the various localisations of infective processes in internal organs of the body exclusively by the mechanical distribution of pathogenetic micro-organisms by the circulation. We must reckon with the vital resistance of the tissues, which varies in different parts of the body, in different species and individuals, and with reference to different organisms. Even the pyogenetic micrococci, which are capable of causing abscesses anywhere in the body, do not generally produce their pathogenetic effects in every place where they may chance to lodge. They have their seats of preference, which vary in different species of animals and probably in different individuals.

The mere presence of pathogenetic bacteria in an embolus does not necessarily impart to it infective properties. This is true even of emboli containing pyogenetic cocci. I have in several instances observed in the spleen and kidney the mechanical, bland effects only of emboli derived from the vegetations of acute infective endocarditis, and have been able to demonstrate streptococci or other pathogenetic organisms in the original vegetations and in the emboli. As has already been remarked concerning thrombi, the line cannot be sharply drawn between bland emboli and septic emboli, simply on the basis of the presence of bacteria; although of course the septic properties must be derived from micro-organisms.

Infective emboli are capable of producing all of the mechanical effects of bland emboli; to these are added the specific effects of the micro-organisms or their products. These latter effects are essentially chemical in nature, and may occur wherever the emboli lodge, being thus independent of the particular circulatory conditions essential for the production of mechanical effects. The most important of these chemical effects are haemorrhages, usually of small size, and of an entirely different causation from those of haemorrhagic infarction; necroses; inflammation, often suppurative, and, in case of putrefactive bacteria, gangrenous putrefaction. The most important function of infective embolism is in the causation of pyaemia. This subject has been most competently presented by Sir Watson Cheyne in Vol. I. p. 881, who has left nothing which requires further consideration here.

Embolic Aneurysms.—Both the first recognition and the correct explanation of embolic aneurysms, at least of the great majority of cases, belong to British physicians and surgeons. Tufnell, in 1853, called attention to the influence of emboli in causing aneurysmal dilatation. There followed observations by Ogle, Wilks, Holmes, Church, and R. W. Smith, before the appearance, in 1873, of Ponfick's important paper on embolic aneurysms. Ponfick explained their formation by direct injury to the vessel-wall, inflicted usually by calcareous, spinous emboli; a view which has since been confirmed only by Thoma. In 1887, Goodhart, in reporting a case, gave the first satisfactory explanation of the mode of production of most of these aneurysms. He pointed out their

association with acute infective endocarditis, and referred them to acute softening of the arterial wall, caused by toxic emboli. Other observations followed; and in 1888 Osler reported a case which, although not embolic, belongs etiologically to the same general category. This was a case of multiple mycotic aneurysms of the aorta due to infective endaortitis associated with infective endocarditis. In 1886 and 1887 appeared the contribution of Langton and Bowlby, the most valuable in English literature, who fully confirmed and expanded in detail the views first briefly announced by Goodhart. Eppinger, in his extensive monograph on aneurysms published in 1887, presented the results of a minute and careful study of this class of aneurysm, which he calls aneurysma mycotico-embolicum, and reported seven personal observations. Of later papers on the subject may be mentioned those of Pel, and Spronck, Duckworth, Buday, Clarke, and Libman.

The evidence is conclusive that aneurysms may be caused by the destructive action of bacteria contained in emboli or directly implanted on the inner vascular wall. The usual source for such emboli in relation to aneurysm is furnished by acute infective endocarditis; but as there is every transition from ordinary warty endocarditis to the most malignant forms, and as the same species of micro-organisms may be found in the relatively benign as in the malignant cases, no single type of endocarditis is exclusively associated with these aneurysms. As is demonstrated by Osler's case, the same result may follow a mycotic endarteritis not secondary to embolism.

Eppinger has shewn that at least the intima and the internal elastic lamina, and usually a part, sometimes the whole, of the media, are destroyed by the action of the bacteria, when an aneurysm is produced. The site of the aneurysm corresponds to this circumscribed area of destruction, and therefore to the seat of the embolus, and is not above it, as some have supposed. The aneurysm is usually formed acutely, sometimes slowly. It may remain small or attain a large size, and an arterio-venous aneurysm has been known to result (Libman). Multiplicity and location at or just above an arterial branching are common characteristics of embolic aneurysms. Favourite situations are the cerebral and mesenteric arteries and arteries of the extremities; but these aneurysms may occur in almost any artery. Arteries without firm support from the surrounding tissues offer the most favourable conditions for the production of embolic aneurysms.

Eppinger totally rejects direct mechanical injury from an embolus as a cause of aneurysm in the manner alleged by Ponfick; and Langton and Bowlby are likewise sceptical as to the validity of Ponfick's explanation beyond its possible application to some of his own cases. Libman, however, in 1907 urged that some embolic aneurysms are non-mycotic; and suggested the following classification of aneurysms, embolic or mycotic in origin: (i.) non-mycotic embolic aneurysms; (ii.) mycotic embolic aneurysms; (iii.) aneurysms due to infection of the wall of the vessels by bacteria in the lumen. Certainly the great majority of

embolic aneurysms are caused by pathogenetic organisms, and belong, therefore, to the class of parasitic aneurysms rather than to that of traumatic aneurysms. The responsible micro - organisms are usually staphylococci or streptococci (Libman). The affection is not common.

In this connexion mere mention may be made of the interesting and very common verminous aneurysms of the anterior mesenteric artery of horses, caused by the *Strongylus armatus.*

General Symptoms.—The symptoms of bland embolism are dependent mainly upon the degree and extent of the local anaemia produced by the arterial obstruction, and upon the specific functions of the part involved. In infective embolism there are additional symptoms referable to local and general infection. Here the constitutional symptoms usually overshadow those referable to the embolic obstruction and the local lesions.

It is not known that any symptoms attend the act of transportation of an embolus, even through the heart. In some situations there is sudden pain at the moment of impaction of the embolus (embolic ictus). This is more marked in large arteries, especially those supplying the extremities, than in smaller and visceral arteries. This pain has been attributed to various causes ; but the most probable explanation seems to me to be irritation, by the impact of the embolus and by the sudden distension of the artery, of sensory nerves and nerve - endings in the vascular wall, present especially in the outer coat. It may be that the Pacinian corpuscles, which are particularly abundant in and around the adventitia of the abdominal aorta, the mesenteric arteries, the iliac and the femoral arteries, are susceptible to painful impressions. Embolism of the arteries named is characterised especially by the intensity of the pain, described sometimes as the sensation of a painful blow, at the moment of impaction of the embolus. Surgeons are familiar with the pain which attends the act of ligation of larger blood-vessels.

Of the pain which follows arterial embolism there are other causes, such as irritation of sensory nerves by local anaemia, altered tension of the part, presence of waste and abnormal metabolic products, structural changes in nerves, inflammation of serous membranes covering infarcts, and so forth.

Some writers have spoken of the occasional occurrence of a nervous or reflex chill at the time of the embolic act ; but, without denying the possibility of such an occurrence, I think that chills associated with embolism have been due usually to infection rather than to vascular plugging.

Although Stricker has constructed a hypothesis of fever based largely upon experiments interpreted by him as demonstrating that the commotion mechanically set up by emboli causes fever, I am not aware of any conclusive observations which shew that fever may be produced in this way in human beings. Independently of the intervention of pathogenetic micro-organisms, arterial embolism may, however, be accompanied by elevation of temperature. Direct invasion of nervous thermic centres

is, of course, only a special case in certain localisations of cerebral embolism. Gangolphe and Courmont attribute the fever sometimes observed after arterial occlusion to the absorption of pyretogenetic substances which they find produced in tissues undergoing necrobiosis. Other possible causes of fever may be the reactive and secondary inflammations consecutive to embolism.

Only in external parts, or parts open to inspection, can the phenomena of mortification, or "local cadaverisation," as Cruveilhier designated the results of shutting off arterial blood, be directly observed. Here are manifest the pallor accompanied by patches of lividity, the cessation of pulsation, the loss of turgidity, the coldness, the annihilation of function, the local death. The haemorrhages which result from arterial obstruction may, however, be evident, not in external parts only, but also by the discharge of blood from the respiratory passages, the intestine, and the urinary tract ; as the result of pulmonary, intestinal, and renal infarction respectively. The phenomena following retinal embolism are open to direct inspection by the ophthalmoscope. In parts not accessible to physical exploration the symptoms are referable mainly to the disturbance or abolition of function, and, therefore, vary with the special functions of the part. They will be considered in connexion with embolism of special arteries (p. 796).

Diagnosis.—The main reliance in the differential diagnosis of embolism from thrombosis, or from other forms of arterial obstruction, is the discovery of a source for emboli, the sudden onset and the intensity of symptoms referable to local arterial anaemia, occasionally the disappearance or marked improvement of symptoms in consequence of complete or partial re-establishment of the circulation, and to some extent the absence of arteriosclerosis or other causes of primary arterial thrombosis.

Valuable as these characters are for diagnosis, they are neither always present nor infallible. For pulmonary embolism the source is to be sought in peripheral venous thrombosis or cardiac disease with thrombi in the right heart ; for embolism in the aortic system, the usual source is the left heart, the great majority of cases being associated with disease of the aortic or mitral valve. It may, however, be impossible to detect the source, and its existence does not exclude the occurrence of thrombosis or other forms of arterial occlusion.

Nor are the symptoms consecutive to embolism always sudden in onset. An embolus may at first only partly obstruct the lumen of the vessel, which is later closed by a secondary thrombus ; or it may be so situated that a thrombus springing from it is the real cause of the local anaemia. For example, an embolus lodged in the internal carotid artery usually causes no definite symptoms, but a secondary thrombus may extend from the embolus into the middle cerebral artery, in which case cerebral softening is sure to follow. On the other hand, the complete closure of an artery may be effected by a thrombus with such rapidity as to suggest embolism.

Whilst the sudden occlusion of an artery by an embolus often causes

temporary ischaemia of greater intensity and over a larger area than the more gradual closure of the same artery by a thrombus, so that when the collateral circulation is fully established the disappearance or reduction of the symptoms may be more marked in the former case than the latter, there may be even in thrombosis very decided improvement in the symptoms with the development of the collateral circulation.

The existence of arteriosclerosis, of course, does not exclude embolism ; but in case of doubt the chances are strongly in favour of embolism in children and young adults with healthy arteries, especially if cardiac disease be present ; the most common association in the latter cases being with mitral affections.

Notwithstanding all of these uncertainties, the diagnosis of embolism, when it produces definite symptoms, can be correctly made in the majority of cases.

Air-Embolism. — The majority of cases in which death has been attributed to the entrance of air into the circulation have been surgical operations and wounds about the neck, shoulder, upper part of the thorax and skull, where air has been sucked into gaping veins and sinuses by thoracic aspiration ; and cases in which air has entered the uterine veins, chiefly from the puerperal uterus, either spontaneously, as after abortions or detachment of placenta praevia, or after injections into the uterine cavity. Jürgensen has reported cases in which he believes death was caused by the entrance of gas into open veins connected with diseased areas in the stomach and intestine. Gaseous embolism has been assigned as the cause of symptoms and of death in caisson-disease and in divers ; and it has been observed in connexion with the development of gas-producing bacilli in the body.

A large number of experiments have been made to determine the effects of air introduced into the circulation. These have demonstrated that when the air is introduced slowly and at intervals, enormous quantities can sometimes be injected in a comparatively short time without manifest injury. Thus Laborde and Muron injected into the external jugular vein of a dog 1120 c.c. in the space of an hour and a half without causing death ; and Jürgensen injected into the left femoral artery of a dog, weighing 43·5 kilo, 3550 c.c. in the space of two hours and a half with only slight disturbance of the respiration and of the action of the heart. In these circumstances the air-bubbles circulate with the blood, pass through the capillaries, and are speedily eliminated. Small amounts of air introduced directly into the carotids, the left heart or thoracic aorta, are often quickly fatal from embolism of the cerebral or coronary arteries.

The sudden introduction of large amounts of air into the veins is quickly fatal. Rabbits are much more susceptible to air-embolism than dogs or horses. 50 c.c. of air, and even more, can often be injected at once into the external jugular vein of a medium-sized dog without causing death ; nor can a dog be killed by simple aspiration of air into the

veins, even when an open glass tube is inserted into the axillary or jugular vein and pushed into the thorax (Feltz). Barthélemy says that as much as 4000 c.c. of air must be introduced into the veins of horses in order to cause death.

After death from entrance of air into the veins, the right cavities of the heart are found distended with frothy blood, and blood containing air-bubbles is found in the veins—especially those near the heart—and in the pulmonary artery and its branches. It is exceptional in these circumstances for air to pass through the pulmonary capillaries into the left heart and aortic system.

There are two principal explanations of the cause of death in these cases. According to one, associated especially with Couty's name, the air is churned up with the blood into a frothy fluid in the right heart, and on account of its compressibility this mixture cannot be propelled by the right ventricle, which thus becomes over-distended and paralysed. According to another hypothesis, supported by experiments of Passet and of Hauer, blood mixed with air-bubbles is propelled into the pulmonary artery and its branches, but the frothy mixture cannot be driven through the pulmonary capillaries, so that death results from pulmonary embolism; Wolf considers that this is by far the most frequent cause of death. The paralysing influence upon the heart of obstruction to the coronary circulation from accumulation of air in the right heart and in the coronary veins must also be an important factor, as well as the cerebral anaemia. Probably all of these factors—over-distension of the right heart, embolism of the pulmonary artery and its branches and of the coronary veins, and cerebral anaemia—may be concerned in causing death, although not necessarily all in equal degree in every case.

We have no information as to the amount of air required to cause death by intravenous aspiration or injection in human beings. It seems certain that man is relatively more susceptible in this respect than the dog or the horse; but it is probable that the fatal quantity of air must be at least several cubic centimetres, and that the entrance of a few bubbles of air into the veins is of no consequence. Many authors have entertained very exaggerated ideas of the danger of entrance of a small quantity of air into the veins.

A large proportion of the cases reported in medical records as deaths due to air-embolism will not stand rigid criticism. I have had occasion to look through the records of a large number of these cases, and have been amazed at the frequently unsatisfactory and meagre character of the evidence upon which was based the assumption that death was due to the entrance of air into the circulation.

So far as I am aware, the first attempt to make a bacteriological examination and to determine the nature of the gas-bubbles found in the blood in circumstances suggestive of death from entrance of air into the vessels, was made by me in 1891. A patient with an aortic aneurysm, which had perforated externally and given rise to repeated losses of blood, died suddenly without renewed haemorrhage. At the

necropsy made in cool weather eight hours after death, there was abundant odourless gas in the heart and vessels without a trace of cadaveric decomposition anywhere in the body. It was proven that the gas was generated by an anaerobic bacillus, which was studied by Dr. Nuttall and myself, and named by us *Bacillus aerogenes capsulatus*. This bacillus is identical with one subsequently found by E. Fraenkel in gaseous phlegmons, and with that found by Ernst and others in livers which are the seat of post-mortem emphysema (*Schaumleber*). In 1896 Dr. Flexner and I reported twenty-three personal observations in which this gas-bacillus was found. The only points concerning these cases which here concern us are, that this bacillus not only may produce gas in cadavers, but may invade the living body, and cause a variety of affections characterised by the presence of gas. There is evidence that the bacilli may be widely distributed by the circulation before death, and that gas generated by them may be present in the vessels during life. In most cases, however, in which this bacillus was present, the gas found in the heart and blood-vessels was generated after death.

Our observations have demonstrated that the finding of gas-bubbles in the heart and vessels a few hours after death without any evidence of cadaveric decomposition is no proof that the gas is atmospheric air, or is not generated by a micro-organism. In all such cases a bacteriological examination is necessary to determine the origin of the gas. In many cases reported as death from entrance of air into the veins, the evidence for this conclusion has been nothing more than finding gas-bubbles in the heart and vessels after sudden or otherwise unexplained death. In the absence of a bacteriological examination, the only cases which can be accepted as conclusive are those in which death has occurred immediately or shortly after the actually observed entrance of a considerable amount of air into the veins. There have been a number of carefully observed and indisputable instances in which during a surgical operation in the " dangerous region " life was imperilled or extinguished by the demonstrated entrance of air into wounded veins. After the audible sound of the suction of air into the vein, death was sometimes instantaneous ; or it occurred in a few minutes after great dyspnoea, syncope, dilatation of the pupils, pallor or cyanosis, occasionally convulsions, sometimes the detection by auscultation over the heart of a churning sound synchronous with the cardiac systole, and the exit from the wounded vein of blood containing air-bubbles. These very alarming symptoms may disappear and the patient recover.

The evidence for this mode of death would seem to be almost as conclusive for a certain number of the sudden deaths following injections into the uterus, especially for the purpose of effecting criminal abortion, and after the separation of placenta praevia. But I am sceptical as to this explanation of many of the deaths which have been reported as due to the entrance of air into the uterine veins. In the reports of Dr. Flexner and myself will be found the description of several cases of invasion of the *B. aerogenes capsulatus*, which without

bacteriological examination would have the same claim to be regarded as deaths from entrance of air into the uterine veins as many of those so recorded. I have had the opportunity to examine the museum specimen of a uterus of a much-quoted case so reported, and I found in its walls bacilli morphologically identical with our gas-bacillus. Certainly all cases of this kind should hereafter be reported only after a bacteriological examination. I do not deny the possibility of the occurrence of fatal air-embolism from the uterus; it is possible, though I do not know of any proof of it, that in some of the cases of sudden death during or immediately after some manipulation or operation on the pregnant uterus and attributed to air-embolism, gas, generated by bacteria, may have existed under pressure within the uterine cavity and have entered wounded veins in sufficient quantity and so suddenly as to cause death. Jürgensen's cases of supposed entrance of gas into the general circulation through the gastric and the intestinal veins are undoubtedly instances of invasion, either before or after death, of gas-forming bacilli.

Since Paul Bert's researches, the symptoms and death which occasionally follow the rapid reduction of previously heightened atmospheric pressure upon exit from a caisson or diver's apparatus, have been attributed to the liberation of bubbles of gas (nitrogen 82 per cent, carbon dioxide 16 per cent, oxygen 2 per cent) in the circulating blood, and air-embolism in the lungs, central nervous system, and other parts. The bubbles are chiefly liberated in the venous blood, and if sufficiently numerous may block the pulmonary artery; this pulmonary embolism is the chief cause of death; there is very little evidence to suggest that cardiac embarrassment is the important factor (Boycott). In caisson-disease the gas is in a fine froth, and thus differs from the large bullae of air seen in cases of air-embolism due to wounds of the veins. Experiments on goats shew that rapid decompression is followed by infarction chiefly in fat and in the white matter of the spinal cord (Boycott and Damant). (See also art. "Caisson-Disease" in Vol. VII.)

Ewald and Kobert have made the curious observation that the lungs are not air-tight under an increase of intrapulmonary pressure which may temporarily occur in human beings. They found in experiments on animals that small air-bubbles may appear in these circumstances in the pulmonary veins and left heart without any demonstrable rupture of the pulmonary tissue; and they argue that this may occur under similar conditions in human beings. The entrance into the circulation of a few minute air-bubbles in this way would doubtless produce no effects. Ewald and Kobert cite two or three not at all convincing published cases in support of the possibility of death resulting from the entrance of air through unruptured pulmonary veins. Very plausible is Janeway's hypothesis that the transitory hemiplegia and other cerebral symptoms, which have occasionally been observed to follow washing-out the pleural cavity with peroxide of hydrogen, or some other procedure by which air or gas may accumulate in this cavity under high

pressure, are due to air-embolism or gaseous embolism of the cerebral vessels.

Not less remarkable are the experimental observations of Lewin and Goldschmidt concerning air-embolism following injections of air into the bladder and its passage into the ureters and renal pelves. It has not been demonstrated that the same phenomenon can occur under similar conditions in human beings.

Fat-embolism.—Fat-embolism, first observed in human beings by Zenker and by Wagner in 1862, is a common form of embolism; in 1905 Connell estimated that 250 cases had been reported; but its practical importance does not correspond to its frequency. It is of greater surgical than medical interest, inasmuch as the severer forms are nearly always the result of trauma. The usual conditions for its occurrence are (i.) rupture of the wall of a vessel, (ii.) proximity of liquid fat, and (iii.) some force sufficient to propel the fat into the vessel.

Fat-embolism probably occurs in every case of fracture of bone containing fat-marrow. When the bone is rarefied, and contains an unusual quantity of fat-marrow, embolism resulting from its injury may be very extensive; as is illustrated by several fatal cases of fat-embolism following the forcible rupture of adhesions in an ankylosed joint. Ribbert has shewn that fat-embolism may result from simple concussion of bone, as from falls or a blow. Inflammations, haemorrhages, and degenerations of the osseous marrow may cause it. It may likewise result from traumatic lesions, necroses, haemorrhages, inflammation of adipose tissue in any part of the body,—of the brain, of a fatty liver, in a word of any organ or part containing fat. Injury to the pelvic fat during childbirth leads to fat-embolism. Oil-globules in the blood may come from fatty metamorphoses of thrombi, of endothelial cells, and of atheromatous plaques. It has also followed subcutaneous injections of oil given to keep up the patient's nutrition. The lipaemia of digestion and of diabetes mellitus has not been generally supposed to lead to fat-embolism, but Sanders and Hamilton observed capillaries filled with oil-globules after death from diabetic coma, and they attribute in certain cases dyspnoea and coma in diabetes to this cause. It is probable that fat-embolism is not of much importance in producing diabetic coma; for lipaemia is rare in diabetes, and conversely it may be considerable without symptoms of fat-embolism. The air-hunger of diabetes is in most cases due to acid-intoxication.

In the great majority of cases, fat-embolism is entirely innocuous, and, unless it is searched for, its existence is not revealed at necropsy, and then only by microscopical examination. Plugging of capillaries and small arteries with oil may, however, be so extensive and so situated as to cause grave symptoms and even death. More moderate plugging may aid in causing death in those greatly weakened by shock, haemorrhage, or other causes. The detection of fat-embolism in the pulmonary vessels may be of medico-legal value in determining whether injuries have been inflicted before or after death.

The deposition of fat-emboli is most abundant in the small arteries and capillaries of the lung, where in extreme cases the appearances of microscopic sections may indicate that considerably over one-half of the pulmonary capillaries are filled with cylinders and drops of oil. In rare instances of extensive injury the amount of fat in the blood may be enormous, so that post-mortem clots in the heart and pulmonary artery may be enveloped in layers of solidified fat. Some of the oil passes through the pulmonary capillaries and blocks the capillaries and arterioles of various organs ; those which suffer most being the brain, the kidneys, and the heart. The extent of the embolism in the aortic system varies much in different cases, being sometimes slight, at other times extensive. Probably the force of the circulation determines the amount of fat which passes through the pulmonary capillaries. Oil once deposited may be again mobilised and transferred to other capillaries.

As already stated, it is only in the comparatively rare instances of extensive fat-embolism that effects of any consequence are produced. The fat itself is perfectly bland and unirritating, although it may be accidentally associated with toxic or infective material. The lesions and symptoms, when present, are referable mainly to the lungs, the brain, the heart, and the kidneys. These lesions are multiple ecchymoses (which in the lungs and the brain may be very numerous and extensive), pulmonary oedema, and patchy fatty degeneration of the cardiac muscle and of the epithelium of the convoluted tubules of the kidney. Pulmonary oedema, referable probably to paralysis of the left heart, is common with extensive fat-embolism of the lungs. Death may undoubtedly be caused by fat-embolism of the cerebral vessels, possibly also by that of the coronary vessels. There may be a remarkable interval, during which the patient feels well, between the accident and the onset of symptoms.

The *symptoms* in the extreme cases are quickened respiration, rapid prostration, reddish frothy expectoration, the crepitations of pulmonary oedema, small frequent pulse, cyanosis, and—with cerebral invasion— coma, vomiting, convulsions, and occasionally focal cerebral symptoms. The temperature may either fall or rise. We may, therefore, follow Brenzinger in recognising two classical groups, pulmonary and cerebral, the latter being much the rarer. Oil-globules are often found in the urine, but it is still an open question whether these are eliminated through the glomerular capillaries, many of which are often filled with oil.

From the investigations of Beneke it appears that the oil is readily disposed of, in small part by saponification, possibly oxidation, and emulsion by means of the blood-plasma ; but in larger part through the metabolic and phagocytic activities of wandering cells which form a layer around the fat. The saponifying ferment—lipase—which Hanriot has discovered in blood-serum is probably one of the agents concerned in disposing of the fat.

Embolism by Parenchymatous Cells.—This is in general of more pathologico-anatomical than clinical interest, and therefore need not be considered here in detail. As has been shewn by Lubarsch, Aschoff, and Maximow, bone-marrow cells, with large budding nuclei, usually undergoing degeneration, may often be found lodged in the pulmonary capillaries after injury to bone, in toxic and infective diseases, in leukaemia, and in association with emboli of other parenchymatous cells. I have seen them in large number in the capillaries of the liver in a case of myeloid leukaemia.

Emboli of liver-cells are found chiefly in the pulmonary capillaries, but may pass through an open foramen ovale so as to reach capillaries of the brain, kidneys, and other organs. F. C. Turner in 1884 first observed liver-cells within hepatic vessels; and later Jürgens, Klebs, Schmorl, Lubarsch, Flexner, and others noted their transportation as emboli after injury, haemorrhages, and necroses of the liver, and with especial frequency in puerperal eclampsia. Secondary platelet-thrombi are usually formed about the cells.

Especial significance was attached by Schmorl to the presence of emboli of placental giant-cells (syncytia) in the pulmonary capillaries in cases of puerperal eclampsia (94); but these emboli, although frequent, are not constant in this affection, and they may occur in pregnant women without eclampsia (Lubarsch, Leusden, Kassjanow).

To the group of parenchymatous emboli may be added the transport of large cells from the spleen to the liver through the splenic and portal veins. I have seen large splenic cells containing pigment and parasites blocking the capillaries of the liver in cases of malaria; and also the well-known large splenic cells containing red blood-corpuscles in cases of malaria and of enteric fever. The crescentic endothelial cells of the spleen may enter the circulation.

After trauma fragments of osseous and medullary tissue may be carried to the pulmonary vessels as emboli (Lubarsch, Maximow). Emboli of large masses of hepatic tissue have been found in branches of the pulmonary artery by Schmorl, Zenker, Hess, and Gaylord as a result of traumatic laceration of the liver. Chorion-villi may be detached and conveyed as emboli to the lungs (Schmorl), or by retrograde transport to veins in the vaginal wall (Neumann, Pick).

So far as is known, emboli of marrow-cells, of liver-cells, of normal syncytial cells, and of splenic cells undergo only regressive metamorphoses, which lead to their eventual disappearance. That without the presence of any syncytial tumour in the uterus or tubes, emboli of syncytial cells may give rise to primary chorion-epithelioma in distant parts of the body is now generally accepted; but it can hardly be supposed that when this occurs the displaced syncytial cells are normal. Indeed, Schmorl's investigations support the view that emboli of normal placental cells disappear; among 150 women dying in various stages of gestation and child-bed he found emboli of placental cells in the lungs to be extremely frequent, more so in 83 cases with eclampsia than in the

others. In two cases only was there undoubted proliferation of the embolic cells, and in both of these the placentae were abnormal (96). Emboli of liver-cells manifest a distinct coagulative influence (Hanau, Lubarsch); and in two instances Lubarsch attributed infarcts in the kidney and the liver to thrombi formed around these cells. Marrow-cells and syncytial cells may likewise cause, in less degree, secondary platelet and hyaline thrombi; but it does not appear that these thrombi have the importance in the etiology of puerperal eclampsia which is attached to them by Schmorl. With a few exceptions, no important lesion of the tissues or definite symptoms have been conclusively referred to emboli of these parenchymatous cells.

Although widely different in results, the transportation of tumour-cells by the blood-current is a process similar to that of parenchymatous embolism, for which indeed cellular embolism seems to me a preferable designation. Benno Schmidt has found small branches of the pulmonary artery plugged with cancer-cells derived from gastric cancer or its meta-stases, both with and without growth of the cells into the walls of the plugged arteries. Such cells may reach the lungs by conveyance through the thoracic duct and innominate vein.

Emboli composed of Foreign Substances.—Since the introduction of subcutaneous intramuscular injections of insoluble substances, such as mercurial preparations for syphilis and paraffin in the repair of a sunken nose and for rectal prolapse, a new form of embolism has appeared. The sudden appearance of pain in the chest, cough, and elevation of temperature, immediately after the hypodermic injection of undissolved preparations of mercury, is attributed to pulmonary embolism. The symptoms disappear in a few days without serious consequences. This complication has been rare in the experience of most of those who have employed this treatment of syphilis, but has led some to abandon the method. In 1902 Mr. S. Paget collected three cases of embolism after injection of paraffin.

Embolism of Special Arteries.—I shall present the salient character-istics of the more important special localisations of embolism, so far as these have not been sufficiently considered in the preceding pages, or do not pertain to other articles in this work. Embolism of the central nervous system will be discussed under Diseases of the Brain and Spinal Cord. The pyaemic manifestations of infective embolism have been described in the articles on "Pyaemia" (Vol. I. p. 880) and on "Infective Endocarditis" (Vol. I. p. 905).

Pulmonary Embolism.—The effects of pulmonary embolism vary with the size of the plugged vessel, the rapidity and completeness of the closure, the nature of the embolus, and associated conditions. Embolism of large, of medium-sized and small arteries, and of capillaries may be distinguished.

The most frequent source of large emboli is peripheral venous throm-bosis, although they may come from the right heart. Sudden or rapid death follows embolism of the trunk or of both main divisions of the

pulmonary artery. It may occur also from embolism of only one of the
main divisions or from plugging of a large number of branches at the
hilum of the lung. Embolism due to particles of growths, to parenchy-
matous cells, and to hydatid cysts, of which Garnier and Jomier have
collected twelve examples, is referred to elsewhere.

Death may be instantaneous from syncope. More frequently the
patient cries out, is seized with extreme precordial distress and violent
suffocation, and dies in a few seconds or minutes. Or, when there is
still some passage for the blood, the symptoms may be prolonged for
hours or even days before the fatal termination. The symptoms of
large pulmonary embolism are the sudden appearance of a painful
sense of oppression in the chest, rapid respiration, intense dyspnoea,
pallor followed by cyanosis, turgidity of the cervical veins, exophthalmos,
dilatation of the pupils, tumultuous or weak and irregular heart's action,
small, empty radial pulse, great restlessness, cold sweat, chills, syncope,
opisthotonos, and convulsions. The intelligence may be preserved, or
there may be delirium, coma, and other cerebral symptoms. Particularly
striking is the contrast between the violence of the dyspnoea and the
freedom with which the air enters the lungs and the absence of pul-
monary physical signs ; unless in the more prolonged cases it be the sign
of oedema of the lungs. Litten found in two cases systolic or systolic
and diastolic stenotic murmurs in the first and second intercostal spaces
on the right or left side of the sternum. In prolonged cases the symptoms
may be paroxysmal with marked remissions. Recovery may follow after
the appearance of grave symptoms. There has been much and rather
profitless discussion as to the degrees in which the symptoms are refer-
able to asphyxia, to cerebral anaemia, or to interference with the coronary
circulation. Doubtless all three factors are concerned, but the exact
apportionment to each of its due share in the result is not easy, nor very
important.

The diagnosis is based upon the sudden appearance of the symptoms,
with a recognised source for an embolus. It is surprising to find in the
larger statistics, as those of Bang and of Bünger, how often the throm-
bosis leading to fatal pulmonary embolism has been latent. Here the
diagnosis cannot always be made ; but in many cases it may be suspected,
or be reasonably certain : as when the above-mentioned symptoms appear
in puerperal women ; during convalescence from infective fevers, as enteric
fever, influenza, pneumonia ; in marasmic and anaemic conditions, as
pulmonary tuberculosis, cancer, chlorosis ; after surgical operations,
especially those on the appendix and the pelvic organs ; and in persons
with varicose veins. As surgical technique has so enormously diminished
the risks of infection and haemorrhage, the dangers of post-operative
pulmonary embolism have become more prominent. In Lichtenberg's
collection of 23,600 operations, including 16,000 laparotomies, there
were 2 per cent of pulmonary complications, and among the laparotomies
5·5 per cent. Among 1000 operations on the vermiform appendix
Mühsam reported 37 cases, 6 of which were fatal. Mauclaire has collected

50 cases after the radical cure of inguinal hernia, 12 out of 25 fully reported cases being fatal.   The prognosis of post-operative pulmonary embolism is extremely grave ; in 233 cases collected by Lenormant death occurred in 106 or 45·5 per cent.

Even at necropsies the source for the embolus has sometimes been missed, but this has been due generally to inability or failure to make the necessary dissection of the peripheral veins, or to dislocation of the entire thrombus.   Serre has published a series of cases of pulmonary embolism with latent thrombosis, shewing the difficulties which may attend the discovery of the source, and the frequency with which patient search reveals the primary thrombus.   The majority of plugs in the trunk or main divisions of the pulmonary artery, found in cases of sudden death, present the anatomical characters of emboli, associated perhaps with secondary thrombi ; but there remain a certain number of cases of sudden or gradual death from primary thrombosis of the pulmonary artery, or from thrombosis extending into a main division from an embolus in a smaller branch (see "Thrombosis," p. 744).

Bland embolism of medium-sized and small branches of the pulmonary artery in normal lungs, and without serious impairment of the pulmonary circulation, usually causes no symptoms and no changes in the parenchyma of the lungs.   Even in lungs structurally altered, and with serious disturbances of the circulation, such embolism may be without effects. The explanation of the harmlessness of the majority of medium-sized and small pulmonary emboli is that the collateral circulation through the numerous and wide pulmonary capillaries is, under ordinary conditions, quite capable of supplying sufficient blood to an area whose artery is obstructed, to preserve its function and integrity ; and that the pulmonary tissue, in contrast to the brain and the kidney, is relatively insusceptible to partial local anaemia.

Often enough, however, medium-sized and smaller branches of the pulmonary artery are occluded by emboli or thrombi under conditions in which the pulmonary circulation is incapable of compensating the obstruction, and then the result is haemorrhagic infarction of the lung. The most common and important of the conditions thus favouring the production of haemorrhagic infarction is chronic passive congestion of the lungs from valvular or other disease of the left heart.   It is especially during broken compensation of cardiac disease that haemorrhagic infarction of the lungs occurs, sometimes indeed almost as a terminal event.   Other favouring conditions are weakness of the right heart, fatty degeneration of the heart, general feebleness of the circulation, pulmonary emphysema, infective diseases.

The source of the embolus causing pulmonary haemorrhagic infarction is oftener the right heart than a peripheral thrombus.   Globular thrombi are often formed in the right auricular appendix and ventricular apex in uncompensated disease of the left heart, particularly of the mitral valve (see "Thrombosis," p. 720).   Wagener records fatal pulmonary embolism in a child two months old, due to detachment of a thrombus

in a patent ductus arteriosus. The infarction may be caused also by thrombosis of branches of the pulmonary artery, which are not infrequently the seat of fatty degeneration of the intima and of sclerosis in cardiac disease and in emphysema. Thrombi in larger branches often give rise to emboli in smaller ones. Dr. Box has described a series of cases to shew that a primary thrombus formed in the trunk of the pulmonary artery may become detached and carried towards the bifurcation of the artery, where becoming coiled upon itself it occludes the orifices of the main branches of the vessel.

Pulmonary infarcts are usually multiple, more frequent in the lower than the upper lobes, and occur on the right side somewhat oftener than on the left, corresponding thus with the distribution of emboli. It has been estimated that in half the cases the infarcts are in the lower lobe of the right lung. Infarction may, however, be confined to the middle lobe of the right lung. Their size varies generally from that of a hazel-nut to a pigeon's egg ; but it may be smaller or much larger, up' to half or even an entire lobe. They are conical or of a wedge-shape, the base being at the pleura. Infarcts are rarely buried in the substance of the lung so as to be invisible from the pleural surface. Typical fresh infarcts are strikingly hard, sharply circumscribed, swollen, upon section dark red, almost black, smooth or slightly granular, and much drier than ordinary haemorrhages. Examined microscopically, the air-cells, bronchi, and any loose connective-tissue which may be included in the infarct are stuffed full of red corpuscles. The capillaries are distended, and in all but the freshest infarcts usually contain, in larger or smaller amount, hyaline thrombi, to which von Recklinghausen attaches much importance in the production of the infarct. Fibrin may be scanty in very recent infarcts, but in older ones it is abundant. The walls of the alveoli in the central part of the infarct are the seat of typical coagulative necrosis with fragmentation and solution of the nuclear chromatin. It is probable that the red corpuscles also undergo some kind of coagulative change, for otherwise it is difficult to explain the extremely hard consistence of the fresh infarct. It is possible that small pulmonary infarcts and very recent ones may occur without necrosis ; but the ordinary ones are necrotic, and cannot therefore be removed by resolution ; but, if the patient lives long enough, and suppuration or gangrene of the infarct does not ensue, are substituted by cicatricial tissue (Willgerodt).

Ever since the first admirable description of haemorrhagic infarcts of the lung by Laennec there has been considerable difference of opinion as to their explanation. The doctrine that they are usually caused by emboli, however, gradually gained general acceptance. This explanation has always had opponents, chiefly on the grounds that emboli often occur in the pulmonary arteries without infarction ; that infarction is not always associated with obstruction of the corresponding artery ; that some have believed that simple haemorrhages may produce the same appearances, and that until recently attempts to produce pulmonary infarction experimentally have been without positive or at least suffi-

ciently satisfactory results.   Hamilton strongly opposed the embolic explanation, and attributed haemorrhagic infarction of the lung to a simple apoplexy, resulting usually from rupture of the alveolar capillaries in chronic passive congestion.   Grawitz, likewise, considers that embolism has nothing to do with the causation of pulmonary infarction, which he explains by haemorrhage from newly-formed, richly-vascularised, peribronchial, subpleural, and interlobular connective-tissue, consecutive to the chronic bronchitis of cardiac and other diseases.   He emphasises structural changes in the lung as an essential pre-requisite for infarction. Grawitz's attack especially has stimulated investigation which, in my opinion, has strengthened the supports of the embolic doctrine of haemorrhagic infarction.

The evidence seems to me conclusive that pulmonary infarcts are caused by embolism and thrombosis of branches of the pulmonary artery.   In the great majority of cases the arteries supplying the areas of infarction are plugged.   Upon this point my experience is in accord with that of von Recklinghausen, Orth, Hanau, Oestreich, and many others.   That these arterial plugs are secondary to the infarction is improbable, as haemorrhages elsewhere, as well as undoubted ones in the lungs, often as they cause secondary venous thrombosis, rarely cause arterial thrombosis.   Moreover, there is sometimes an interval of open artery between the plug and the infarct, a relation not observed with the undoubtedly secondary thrombosis of veins connected with the infarct, and not explicable on the assumption that the arterial thrombosis is secondary.   The plug often has the characters of a riding embolus. Not a few of the plugs, however, are primary thrombi.   The occasional occurrence of pulmonary infarction without obstruction in the arteries has as much, but no more, weight against the embolic explanation as the similar, and I believe quite as frequent, occurrence of splenic infarcts without embolism or thrombosis of the splenic arteries.   Both the haemorrhage and the necrosis of infarcts are essentially capillary phenomena, each being independent of the other ; and undoubtedly can occur, in ways little understood, in various regions, without plugging of the arteries.

The anatomical characters of pulmonary infarcts are essentially the same as those of haemorrhagic infarcts of the spleen and other parts. The conical shape, the hard consistence, the peripheral situation, the coagulative necrosis are distinctive characters of pulmonary as of splenic infarcts.   The necrosis cannot well be attributed to compression of the alveolar walls by the extravasated blood, for the capillaries in these are usually distended widely with blood.   It has the general characters of the ischaemic necrosis of infarcts, except that it apparently occurs somewhat later in the formation of the infarct and does not usually reach the periphery,—phenomena which may be explained by the relative tolerance of the pulmonary tissue of partial ischaemia, and by a better peripheral circulation than is present in infarcts elsewhere.

Inasmuch as emboli do not ordinarily cause infarction in normal

human lungs with vigorous circulation, it is not surprising to find that similar emboli under similar conditions do not cause infarction in the lungs of animals. It is not easy to reproduce experimentally in animals the conditions under which pulmonary infarcts occur in man ; yet there have been several valuable contributions in recent years to the experimental production of pulmonary infarction : these have furnished an experimental basis, which, if not all that is to be desired, still marks a distinct advance for the embolic doctrine of haemorrhagic infarction of the lung. Pulmonary infarcts, in all essential respects identical with those in human lungs, have been produced by experimental embolism or arterial occlusion by Cohnheim and Littén, Perl, Küttner, Mögling, Grawitz, Klebs, Gsell, Sgambati, Orth, Zahn, and Fujinami. Most of these experimental infarcts have been produced under conditions not very analogous to those of human infarcts ; but the essential fact that typical haemorrhagic infarction of the lung may be caused by arterial plugging has been experimentally established. Into the details of these experiments it is impossible here to enter.

Whether genuine haemorrhagic infarcts of the lung may ever be caused by simple haemorrhage from rupture of blood-vessels is perhaps an open question. At present this mode of their production seems to me undemonstrated and improbable, so that I hold that simple pulmonary apoplexies and genuine infarcts should be clearly distinguished from each other. Neither the results of experimental introduction of blood into the trachea (Perl and Lippmann, Sommerbrodt, Nothnagel, Gluziński), nor the appearances of the lungs after undoubted bronchorrhagias, pneumorrhagias, and suicidal cutting of the trachea, support the opinion that aspiration of blood from the trachea and bronchi causes genuine haemorrhagic infarction. In only one of Sommerbrodt's numerous experiments was such infarction observed, and this he regards as accidental. The explanation of this exceptional result is probably the same as in Perl's experiment with thrombosis after venesection and anaemia.

I have seen, in two or three instances, nearly white or pale-red fresh anaemic infarcts in densely consolidated lungs. In very rare instances pulmonary infarcts are anaemic in consequence of extreme weakness of the circulation, or because the infarct occurred very shortly before death (Freyberger). Even when caused by bland emboli pulmonary infarcts are exposed to the invasion of bacteria from the air-passages ; and such bacterial invasion may lead to suppuration or gangrene. Completely cicatrised pulmonary infarcts occur, but they are not common— life being usually cut short by the associated cardiac disease before the infarct is healed.

Haemorrhagic infarction of the lungs may be entirely latent ; often, however, the diagnosis can be made during life. The affection may be ushered in by a chill or chilly sensation, increase of a usually existing dyspnoea, and localised pain in the side. These symptoms are far from constant. The characteristic symptom, although by no means patho-

gnomonic, is bloody expectoration. Profuse haemoptysis was noted by Laennec, but is very rare. The sputum contains dots and streaks of blood, or small dark coagula ; or, more frequently, the blood is intimately mixed with the expectoration, which is in small masses and usually less viscid and darker red than that of pneumonia, although it may resemble the latter. Blood may be present in the sputum for one or two weeks, or even longer, after the onset of the infarction. It acquires after a time a brownish-red tint, and generally contains the pigmented epithelial cells usually seen in the sputum of chronic passive congestion. Circumscribed sero-fibrinous pleurisy is usually associated with pulmonary infarction, and a very considerable pleural effusion may follow. Even with infarcts not more than four or five centimetres in diameter the physical signs of consolidation and subcrepitant rales can sometimes be detected, usually in the posterior lower parts of the lungs. These signs are referable not only to the infarct, but also to the surrounding localised oedema and perhaps reactive pneumonia. There may be moderate elevation of temperature. When the characteristic bloody expectoration, together with signs of circumscribed consolidation, appears in the later stages of cardiac disease, or with peripheral venous thrombosis, there is generally little doubt of the diagnosis. Yet similar expectoration may occur from simple bronchial haemorrhages in intense passive congestion of the lungs without infarction. The expectoration in malignant disease of the lungs may resemble that of pulmonary infarction. The embolic pneumonias and abscesses caused by infective emboli are pyaemic manifestations, and have been considered in the article on "Pyaemia" (Vol. I. p. 887).

Splenic Infarction.—Anaemic infarcts of the spleen, which are commoner than the haemorrhagic variety, are not usually in the recent state so pale and bloodless as those of the kidney ; for the spleen is much richer in blood than the kidney, and in chronic passive congestion, during which the larger number of infarcts occur, the red pulp contains much blood outside of the vessels. Many of these infarcts can be appropriately described as mixed red and white infarcts. Splenic infarcts vary greatly in size, but in general they are much larger than those occurring under the same conditions in the kidney, as comparatively large arteries in the spleen break up into numerous small terminal twigs. Averaging perhaps two to six centimetres in diameter, a single infarct may occupy one-half or more of the spleen. The recent infarcts are hard, swollen, and more or less wedge-shaped, with the base at the capsule, which is often coated with fibrin, or in older cases thickened and adherent by fibrous tissue. The great majority are caused by emboli from the left heart or the aorta ; but both haemorrhagic and pale splenic infarcts occur without arterial occlusion, especially in certain acute infective diseases, oftenest in relapsing fever, but also in typhus, enteric fever, cholera, and septicaemia. The causation of the latter is unknown. Ponfick attributes them to venous thrombosis, which may be the cause of the haemorrhagic infarcts ; but it is difficult to understand

how it can produce the pale anaemic infarcts. Bland infarcts are mostly absorbed and substituted by pigmented, occasionally calcified, scars, which when numerous may cause a lobular deformity of the spleen. Splenic calculi have been described as a late result of infarction (Lemaire).

Splenic infarction is often entirely latent. Of the symptoms attributed to it chills and elevation of temperature belong usually to the accompanying acute or chronic endocarditis. Swelling of the spleen is produced by infarcts. The most diagnostic value attaches to the sudden appearance of pain in the region of the spleen, perhaps increased by lying on the left side, by deep inspiration, and by pressure; and to a perisplenitic friction-rub, which can sometimes be detected. The condition may thus simulate left-sided pleurisy. Vomiting may also occur. These symptoms are not very certain diagnostic points; but when they occur with some manifest source for a splenic embolus, and perhaps with recognised embolism in other organs, they justify a strong suspicion of splenic infarction.

Renal Infarction.—There have been a few instances, especially after trauma, of nearly total necrosis of a kidney from thrombosis of the renal artery, combined usually with thrombosis of the vein. Halperin records a case of thrombosis of the left renal artery due to endarteritis obliterans with an enormous infarct occupying more than two-thirds of that kidney. Usually plugging of the main artery leads to multiple infarction with intervening intact areas. The capsular arteries suffice for the preservation of at least a narrow outer rim of renal tissue.

Renal infarcts are nearly always anaemic, in the recent state somewhat swollen, and of an opaque pale yellowish colour, with the base of the wedge just beneath the capsule and the apex towards the hilum, most frequently near the boundary between the pyramid and cortex. Three zones can often be distinguished: the main central yellowish-white mass of necrotic tissue; next to this a narrow yellow zone of fatty cells, nuclear fragments, and disintegrating leucocytes; and an outer, irregular, variable rim of hyperaemia and haemorrhage which belongs partly to the infarct and partly to the surrounding tissue. The haemorrhage may extend a variable distance into the infarct, and in very rare instances genuine haemorrhagic infarcts occur in the kidney. Numerous scars from old infarcts may produce a form of atrophic kidney to which the epithet embolic is applicable. Thorel finds that a limited regeneration of the epithelium and even of uriniferous tubules may occur in healing renal infarcts.

Very large infarcts may so stretch the renal capsule as to induce severe pain, which may suggest Dietl's crises or renal colic, but it is constant and not intermittent. In a case diagnosed by Traube an infarct two inches in diameter, projecting well above the surface, caused intense pain and tenderness in the region of the infarcted kidney, with extension of the pain into the corresponding thigh. With the ordinary small infarcts pain is not usually a prominent symptom. The chief sign

of diagnostic value is the sudden appearance of blood in the urine in association with disease of the left heart, aortic aneurysm, or other recognised source for a renal embolus.   The amount of blood is usually only moderate or evident by microscopical examination of the urine.   It is to be remembered that chronic passive congestion of the kidney is itself one of the many causes of haematuria.   Haematuria may be absent, and is less common than albuminuria.   The general symptoms are vomiting, collapse, headache, constipation, and fever.   When haematuria is present, the condition must be diagnosed from acute nephritis without dropsy (*vide* Vol. IV. Part I. p. 604).

Infective emboli, which are often capillary in size, cause multiple, often miliary, abscesses in the kidney.   This is the haematogenous variety of acute suppurative nephritis which occurs often in acute infective endocarditis and other forms of pyaemia.   Here the pyuria and other renal symptoms are usually of less consequence than those of general infection.

Embolism and Thrombosis of the Mesenteric and Intestinal Arteries.— Thrombosis of the mesenteric veins, which causes lesions and symptoms identical with those following embolism of the mesenteric arteries, has been referred to in the preceding article (p. 752).   Since Virchow's first description of embolism of the superior mesenteric artery, in 1847, a large number of cases have been reported of embolism or thrombosis of the mesenteric arteries.   The affection, although not common, occurs often enough and is of such gravity as to be of considerable clinical interest.   In Watson's collection of cases there are eight which occurred within a single year in Boston.   The casuistic literature upon the subject is fairly extensive.   The articles of Litten and of Faber contain reports of most of the cases published up to 1875.   The principal clinical features were carefully studied by Gerhardt and by Kussmaul in 1863-64.   The papers of Watson and of Elliot in 1894-95 refer to about 50 reported cases, and in 1904 Jackson, Porter, and Quinby analysed 121 cases.   The effects of occlusion of the mesenteric arteries have been experimentally studied by Beckmann, Cohn, Litten, Faber, Welch and Mall, and Tangl and Harley.

The principal conclusions drawn by Mall and myself from our experiments have been stated already in the discussion of the collateral circulation, and of haemorrhagic infarction following embolism (pp. 779 and 780).   It may here be repeated that, according to our experiments, the blood which produces the haemorrhagic infarction enters by the anastomosing arteries and not by reflux from the veins ; that the haemorrhage cannot be explained by any demonstrable change in the vascular walls, but is the result of retardation and stasis of the circulation and clumping of red corpuscles in the veins and capillaries, attributable in large part in cases of arterial obstruction to reduction or loss of lateral pulsation of the blood-current ; that the ischaemia is increased by the tonic contraction of the intestinal muscle which follows, for two or three hours, closure of the superior mesenteric artery ; and that the

sudden and complete shutting off of the direct arterial supply to a loop of intestine 5 to 10 cm. in length is followed by haemorrhage and necrosis of the loop, even when the vessels at each end of the loop are open. These results we obtained by experimentation upon dogs, but there is no reason to suppose that they do not apply to human beings. With the exception of Cohn, the other experimenters explain the infarction by regurgitant flow from the veins and alterations in the vascular walls.

The majority of the cases of haemorrhagic infarction of the intestine have been due to embolism of the mesenteric arteries, the source of the embolus being usually the left heart, sometimes an atheromatous aorta or aortic aneurysm, and in one instance a thrombus in the pulmonary veins caused by gangrene of the lungs (Virchow). Several cases have been caused by autochthonous thrombosis resulting from arteriosclerosis, aneurysm, pressure, or the extension of a thrombus from the adjacent aorta. It is probable that a certain number of the cases reported as embolic were referable to primary thrombosis of the mesenteric arteries, as no source for an embolus could be discovered, and the plugs in some of these instances were fresh adherent thrombi. As has been shewn in the preceding article, primary thrombi may form in arteries which are free from atheroma or other chronic disease.[1]

In the great majority of the cases the obstruction was in the superior mesenteric artery. The few scattered instances of embolism or thrombosis of the inferior mesenteric artery indicate that this also may, very rarely, cause incomplete haemorrhagic infarction of the corresponding part of the intestine, but that the collateral circulation here is better, and the lesions likely to consist only in small haemorrhages in the intestinal mucosa. The inferior mesenteric artery may be obliterated without any manifest disturbance in the structure or function of the part of the intestine supplied by it.

The obstruction may be situated in the main stem or in any of the branches of the superior mesenteric artery. Intestinal infarction has been associated with embolism of the larger branches oftener than with that of the main stem. As the anastomoses through the arterial arches are so free, obstruction of single small branches is without mechanical effects. There have, however, been several instances of intestinal infarction caused by multiple emboli or extensive thrombosis of small branches of the superior mesenteric artery.

Intestinal infarction is not the imperative result of occlusion of the superior mesenteric artery, as infarction is of occlusion of branches of

[1] Litten has reported two cases of haemorrhagic infarction of the intestine from thrombosis caused by what he calls "latticed endarteritis" (gitterförmige Endarteritis) of the mesenteric arteries. So far as I can learn he has not furnished the fuller description which he promised in his article in 1889. Without such description there is room for the suspicion that Litten has mistaken the latticework markings sometimes seen after detachment of an adherent thrombus for a special form of endarteritis. It does not appear from his article that he has observed this "latticed endarteritis" except after removing adherent thrombi.

the splenic and renal arteries, and of the basal cerebral. Both the trunk and the principal branches of this artery may be gradually closed without serious effects. Tiedemann and Virchow have found the superior mesenteric artery completely obliterated by old, firm thrombi or connective tissue without any lesions in the jejunum or ileum. The most remarkable case is that of Chiene, who found in a woman sixty-five years old, with aneurysm of the abdominal aorta, complete obliteration of the coeliac axis and both mesenteric arteries, with an adequate collateral circulation through the greatly distended extra-peritoneal anastomosing arteries. In a number of instances plugging of large branches of the superior mesenteric artery has caused no more than hyperaemia and superficial ecchymoses, without genuine infarction of the intestine.

The rapid and complete closure of the superior mesenteric artery, however, is followed with great regularity, probably constantly, by haemorrhagic infarction of the intestine. There have been several instances in which embolism or thrombosis of the trunk of this artery has caused haemorrhagic infarction extending from the lower part of the duodenum into the transverse colon (Oppolzer, Pieper, Faber, Kaufmann), as in the experimental cases. More frequently the infarction is in the lower part of the jejunum and the ileum, corresponding to the occlusion of a principal branch or of several branches supplying this region. The infarction corresponds in general to the area of distribution of the plugged arteries, but it may occupy only a part of this area. In several instances a single small loop or several loops with intervening normal intestine have been infarcted.

As already intimated, the infarction may be complete or only partial. When completely infarcted, the wall of the affected intestine is thickened, oedematous, of a dark-red colour from infiltration with blood and covered with lustreless peritoneum. The margins of the infarct are often sharply marked, but may pass gradually into the normal bowel. The mucous membrane is necrotic, often defective, and may be coated with a diphtheritic exudate. In a few instances the intestine has been gangrenous over considerable areas, without typical haemorrhagic infarction, or with the haemorrhagic appearance adjacent to the gangrene. The lumen of the intestine contains black tarry blood. There is bloody fluid in the peritoneal cavity, and usually a fibrinous, sometimes a fibrino-purulent exudate on the peritoneum covering the infarction; and there may be general peritonitis. The mesentery is succulent and haemorrhagic, usually in patches, exceptionally in the form of large flat masses of extravasated blood. Areas of fat-necrosis may be present in the mesentery. The mesenteric veins are distended and the mesenteric glands often swollen and haemorrhagic. Various intestinal bacteria, most commonly the colon bacillus, may make their way into the peritoneal cavity through the necrotic wall. Flexner and I have reported an instance of haemorrhagic infarction of the jejunum in which evidences of pneumo-peritonitis, supposed to be due to perforation, existed during life. At the necropsy, made six hours after death, a large

amount of gas was found in the peritoneal cavity without perforation. The *B. aerogenes capsulatus* was present in large numbers in the peritoneal exudate. This case demonstrates the generation of gas in the closed peritoneal cavity. In the intestinal mucosa were gas-blebs which were observed also in one of Faber's cases and in Jürgens' case of intestinal infarction.

The haemorrhagic infarction is by no means always so completely formed as that just described. There may be no haemorrhages in the mesentery. The extravasation of blood may be limited to the mucosa, or even to the submucosa, as in one of Ponfick's cases. An instance of nearly complete thrombosis of the trunk of the superior mesenteric artery, reported by Councilman, shewed paralysis, great distension and ecchymoses of the small intestine, but no infarction. Between mere venous hyperaemia with scattered superficial haemorrhages, and complete necrosis and infarction, there are all gradations, the controlling factors being doubtless the rapidity and extent of the arterial occlusion and the vigour of the general circulation.

There have been two or three instances in which the anatomical picture of haemorrhagic infarction of the intestine has been present without the discovery of any obstruction in the corresponding arteries or veins. Lycett reports an observation of haemorrhagic infarction of the small intestine in an infant one month old without discoverable cause.

Symptoms.—Cases of haemorrhagic infarction of the bowel have been divided into two groups : (i.) with a gradual onset and chronic course, and (ii.) with a sudden onset and acute symptoms often regarded as those of intestinal obstruction ; most of the cases belong to this category. In the majority of cases, severe colicky pain and abdominal tenderness, either without distinct localisation or most marked near the umbilicus, are prominent and usually the first symptoms. The pain at the beginning is perhaps attributable to the violent, tonic spasm of the intestine which follows sudden occlusion of the superior mesenteric artery. After a few hours this spasm gives place to complete paralysis of the affected part of the bowel, and then the pain may be referable to peritonitis. The local anaemia, haemorrhage, and necrosis seem, however, quite sufficient to account for the pain. Vomiting, which often becomes bloody and occasionally faecal, is also usually an early and persistent symptom. By far the most characteristic symptom is the passage of tarry blood in the stools, which are frequently diarrhoeal, and sometimes have the odour of carrion. In nearly all cases there is haemorrhage into the bowel, but the blood is not always voided. Osswald states that in two-thirds of the cases obvious intestinal haemorrhage is absent. Symptoms of intestinal obstruction—tympanitic distension of the abdomen, feculent vomiting, and obstipation—are in some cases prominent, and are readily explained by the complete paralysis of the infarcted bowel. The sub-normal temperature, pallor, cold sweats, and collapse, which appear in most cases, are explicable in part by the intestinal haemorrhage, and in part by the shock of the destructive lesion. Attention has been drawn

to a peculiar excitability of the nervous system suggesting hysteria (Mayland). The sensation of a palpable tumour, referable to a collection of blood in the mesentery or to the infarcted bowel, has been noted in only three or four cases.

The chief emphasis for purposes of diagnosis is to be laid upon the occurrence of intestinal haemorrhage, not explicable by independent disease of the intestine or by portal obstruction, in combination with other symptoms mentioned, and with the recognition of some source for an embolus, perhaps of embolic manifestations elsewhere. In the majority of cases the diagnosis has been intestinal obstruction or acute peritonitis. The symptoms closely resemble those of intussusception, in which haemorrhage from the bowel, although generally less abundant than with embolism of the superior mesenteric artery, is common. Fortunately the distinction of haemorrhagic infarction from intestinal obstruction is not of much practical importance ; for if the symptoms and condition of the patient warrant it, an exploratory laparotomy is indicated in both conditions.

The prognosis is grave ; and with complete infarction and necrosis of the intestine it is almost necessarily fatal, unless surgical relief be available. Watson estimates that in about one-sixth of the cases the location and extent of the infarction are suitable for resection of the bowel. Operation in 47 cases of thrombosis or embolism of the mesenteric vessels gave a mortality of 92 per cent (Jackson, Porter, Quinby).

When the infarction is incomplete, and is limited chiefly to the inner coats of the intestine, recovery may doubtless take place. Cohn, Moos, Lereboullet, and Finlayson have reported instances of recovery after symptoms indicative of haemorrhagic infarction. Packard attributed cicatricial areas found in the mesentery of an old man dead of rupture of the ascending aorta to healed infarction ; but no previous history was obtained, and Packard's interpretation does not seem to me to be free from doubt. Death may occur within 20 to 48 hours after the onset, or the duration may be protracted over several days. Karcher has reported the survival of a patient with mitral stenosis for two months after the complete occlusion of the superior mesenteric artery by an embolus, the symptoms being sufficiently characteristic to have permitted a probable diagnosis during life.

Intestinal ulcers due to embolism or thrombosis constitute a distinct class, which has been studied especially by Ponfick, Parenski, and Nothnagel. Parenski relates an instance of operation for intestinal stricture, which at the necropsy was found to be caused by cicatrisation of an ulcer due to embolism of a branch of the superior mesenteric artery. Much more common are ulcers caused by infective emboli lodging in the small arteries and capillaries in the intestinal wall ; they are observed especially in acute ulcerative endocarditis and pyaemia. These emboli cause haemorrhages, necroses, and miliary abscesses with resulting ulceration. The ulcers are usually multiple, sometimes numerous, and situated in the small intestine and caecum. The intestinal ulcers occasionally associated

with degenerative multiple neuritis are referred by Minkowski and Lorenz to thrombosis caused by disease of the small arteries, which has been repeatedly observed in this form of neuritis.

Embolism and Thrombosis of the Thoracic Aorta.—Unless there be some abnormal narrowing or obstruction of the aorta, it is hardly possible for an embolus to lodge in this vessel, except at the ostium or the bifurcation. An exception to this rule may result from the detachment of a large aneurysmal clot, which, as in three cases of abdominal aneurysm reported by Bristowe, may block the aorta at or just below the mouth of the aneurysm.

I know of but three instances of embolism of the mouth of the aorta —two reported by Cohn with instantaneous death, and one by Reid in which the patient lived an hour and a half after the first symptoms of partial obstruction.

In a very few instances the lumen of an atheromatous thoracic aorta has been seriously encroached upon, or even obliterated, by thrombotic masses. Such cases have been reported by Trost, Tewat, Carville, Armet, Chvostek, Jaurand, and Pitt. The thrombus may occupy the ascending, the transverse, or the descending aorta, and may occlude the mouths of the left carotid and subclavian arteries. If there remain a sufficient channel for the blood, as in Pitt's case, there is no resulting circulatory disturbance ; otherwise there may be paralysis, oedema, gangrene of the lower extremities, and if the left subclavian is obliterated, of the corresponding upper extremity.

Bochdalek and Lüttich have each described an instance of occlusion of the aorta in infants by the extension of an obliterating thrombus from a dilated ductus Botalli. Wagener figures a thrombus issuing from the ductus Botalli hanging freely into the aorta. Far more frequent is stenosis or atresia of the aorta at or near the attachment of this duct, due usually to persistence of the isthmus aortae, as was first shewn by Rokitansky. (*Vide* Coarctation of the Aorta, p. 286.)

Embolism and Thrombosis of the Abdominal Aorta.—Graham, in 1814, referred to a museum specimen in Glasgow, which had belonged to Allan Burns, of occlusion of the abdominal aorta just above the bifurcation by old laminated coagulum extending into the iliacs. In 1898, I found 59 subsequent reports of occlusion of the abdominal aorta by embolism or thrombosis.[1] I did not include in this list the detachment of clots from abdominal aneurysms, although Bristowe's 3 cases demonstrate that this may occasion the same symptoms. The monographs and articles of Meynard, Cammareri, Selter, Roussel, Charrier and Apert, and Heiligenthal contain references to or reports of 47 cases ; to these I added 12 published cases not mentioned by them. Since the first edition of this work a number of cases have been published ; in 1903

---

[1] I have not included von Weismayr's case (*Wien. med. Presse*, 1894, xxxv. 1774) as it was reported while the patient was living, and in the discussion some doubt was expressed as to the diagnosis ; nor the brief mention made by Teleky, at the same time, of a similar observation.

Barié and Halbron collected 37 cases of embolism of the abdominal aorta including 6 since 1898, and others have been reported by Rénon, Claisse and Abrami, Vigouroux and Charpentier, Mazoux, F. H. Hawkins, Fawcett, Daggett, and others. The statistics, however, have been retained in the same form as in the first edition.

Of the 59 cases 3 patients were living at the time of the reports, and in 2 fatal cases there was no necropsy. In the remaining 54 the plug occupied the lower end of the aorta and extended a variable distance into the arteries below. In 31 the plug did not reach higher than the inferior mesenteric artery; in 10 the upper extremity lay between the inferior mesenteric and the renals; in 3 between the renals and the superior mesenteric; in 2 between the latter and the coeliac axis; in 1 just below the pillars of the diaphragm; and in 7 the length of the plug is not stated. The upper part was often conical; so that, when the plug extended higher than the inferior mesenteric, it was often not obliterating until at or below this artery. In the great majority of cases only the last, or the last two, lumbar arteries were blocked by the thrombus. In several instances a thrombus, either independent or continuous with that in the aorta, occupied the lumbar, the mesenteric, the renal, or other branches of the aorta. In all instances the thrombus extended into the common iliacs, and in many into arteries lower down, sometimes even as far as the posterior tibial, the end being usually lower on one side than on the other.

It is difficult, indeed impossible, from the published descriptions, which are only too often incomplete and unsatisfactory, to determine accurately how many of the cases were referable to embolism and how many to thrombosis. Essentially similar cases have been interpreted differently in this respect by different observers. .The plug was usually adherent, and only in relatively few cases were its anatomical characters such (or at least so described) as to indicate positively its nature as embolus or primary thrombus. The majority of cases with sudden or rapid invasion of characteristic symptoms were associated with cardiac disease, or disease of the upper part of the aorta; and would, therefore, naturally be interpreted as embolic. Still, in many of these no satisfactory source for a large embolus was demonstrated. Some cases not less abrupt in onset were without any affection of the heart or of the aorta above the plug. The sudden appearance of symptoms of obstruction of the aorta, although strongly indicative of embolism, are not decisive upon this point. Barth, in 1848, described a case of obstruction of the aorta by a cylindrical thrombus extending from the superior mesenteric artery to the bifurcation, and leaving only a narrow channel for the circulation of the blood. There were no circulatory disturbances. If this narrow channel had been suddenly closed at one point, as might readily happen, the symptoms would probably have been those of embolism. It is evident that aortic thromboses secondary to only partly obliterative emboli riding the bifurcation of the aorta, or to emboli or thrombi in the iliacs or lower arteries, may occasion

symptoms like those of primary thrombosis of the aorta. There are several instances of such secondary thrombosis of the aorta in my collection of cases.

Without much confidence in the accuracy of the classification in several instances, I have divided the 59 cases into 45 referable to embolism of the bifurcation of the aorta, and 14 due to thrombosis ; of the latter, 7 were primary, 6 secondary to embolism of the iliacs, or possibly the femoral, and 1 to thrombosis of the arteries of the extremities. The source of the aortic embolus is believed to have been the heart in 35 cases ; aneurysm of the ascending aorta in 1 ; pressure of a tumour on the aorta in 2 ; atheroma of the thoracic aorta in 1 ; in 6 it was undetermined. The heart was found to be normal at the necropsy in 11 of the 53 cases ; and in 7 both the heart and the aorta above the plug were normal.

Mitral stenosis existed in 20 cases (2 of these being caused by thrombi extending from the left auricle into the ventricle) ; acute mitral endocarditis in 3 ; mitral endocarditis, not further defined, in 4 ; mitral insufficiency without stenosis in 1 ; thrombus in the left auricle without valvular disease in 1 ; thrombi in the left ventricle, mostly without valvular disease, in 8 ; and large aortic vegetations in 1.

The most interesting point in the etiology of plugging of the abdominal aorta, so far as it is permissible to draw conclusions from so few instances, is that nearly 34 per cent of the cases were associated with mitral stenosis. In many of these the stenosis was extreme. The question at once arises of the source of the embolus in these cases, for it cannot be supposed that an embolus large enough to occlude the lower end of the aorta could pass through the contracted mitral orifice. Some of the cases may be explained by a smaller embolus caught at the aortic bifurcation, or in an artery lower down, with secondary thrombosis of the aorta ; but the sudden onset of motor and sensory paraplegia, and of cessation of pulsation in both femoral arteries in a large number of cases, seems to demand abrupt stoppage of the circulation through both common iliacs. A few observers who have realised the difficulty here presented have assumed that a large thrombus had formed in the left ventricle and been detached without leaving any trace behind ; for only in two or three of the cases with mitral stenosis was there any evidence of a thrombus in the left ventricle or the aorta above the plug. This explanation must be regarded as purely hypothetical. The coexistence in a number of the cases of infarctions of the spleen, kidney, or brain has seemed to some writers strong evidence in favour of the embolic nature of the aortic plug. It is possible that the explanation even of the cases with acute bilateral symptoms referable to aortic obstruction, and associated with marked mitral stenosis, may be the lodgment of a small embolus followed by thrombosis of the aorta. Although in the classification above given I have placed nearly all the cases with mitral stenosis under embolism, I am nevertheless not disinclined, in spite of the rapid onset of the symptoms and frequently

coexistent infarctions, to interpret many of them as primary thromboses of the aorta. The circulatory conditions in extreme uncompensated mitral stenosis seem favourable to the occurrence of arterial thrombosis ; and, if this view be accepted for the plugging of the abdominal aorta, the question arises whether thrombi frequently present in smaller arteries in association with this form of valvular disease may not oftener be primary than is generally supposed ?

In a few cases congenital narrowing of the aorta was noted. In three instances plugging of the abdominal aorta was associated with embolism or thrombosis of arteries of an upper extremity. Coincident thrombosis of the vena cava, iliac, or femoral veins was observed in a few cases. In Jürgens' patient there was haemorrhagic infarction of the intestine. In several instances haemorrhages were found at necropsy in the mucous membrane of the bladder and uterus. Herter, in his experiments in my laboratory with ligation of the abdominal aorta in rabbits, found haemorrhagic infarction of the uterus to be such a common result of this operation that, when it was desired to keep the animals alive for any length of time, we abandoned the use of female rabbits for Stenson's experiment. It does not appear, however, that in human beings haemorrhage of the uterus is a common sequel of occlusion of the abdominal aorta.[1] It is probable that if search were made in suitable cases in human beings who have died of aortic thrombosis or embolism, the interesting muscular changes described by Herter in the experimental cases would be found, as similar changes had been previously discovered by Litten in an instance of occlusion of the right iliac and femoral arteries. The most important of these muscular alterations are vacuolisation, proliferation of the sarcolemma nuclei, atrophy, and fatty and pseudo-waxy degenerations.

Plugging of the abdominal aorta has occurred most frequently in the course of chronic cardiac or arterial disease ; but in some instances it took place during or after an acute infective disease, as rheumatic fever, puerperal infection, erysipelas, during convalescence from enteric fever (Forgues), and after pneumonia (Leyden).

Of the 59 cases 30 were females, 27 males, and in 2 the sex is not stated : 17 were between twenty and thirty years of age, 12 between thirty and forty, 8 between forty and fifty, 13 between fifty and sixty, 1 was nineteen, 1 sixty-one, and the ages of 7 are not given.[2] Marked atheromatous changes in the arteries were noted in 14 cases. Occlusion of the abdominal aorta by embolism or thrombosis, therefore, is not especially a senile affection.

---

[1] It may here be mentioned that Herxheimer, Popoff, and Chiari have each described an instance of haemorrhagic infarction of the uterus after extensive bilateral plugging of the vessels supplying this organ.

[2] In Lüttich's case already mentioned (p. 809) of thrombosis of the aorta in an infant fourteen days old, a thrombus beginning 4 cm. below the insertion of the ductus Botalli extended into the iliac arteries. Charrier and Apert include in their collection of reports of thrombosis of the abdominal aorta 2 cases from Allibert's thesis of 1828, one three and the other three and a half years old, with gangrene of one leg. I have not counted these 3 cases in my list.

When one considers the manifold conditions under which the abdominal aorta may become partly or completely plugged by embolism or by primary or secondary thrombosis, it is evident that there can be no general uniformity of symptoms. The plug may be so situated as to interfere with the circulation in one leg more than in the other. Diversities arise from variations in the collateral circulation in different cases. Still the majority of patients have presented a well-characterised group of symptoms. In the larger number of cases the onset has been acute, in the minority insidious and gradual. The symptoms have often appeared simultaneously in both legs, but there may be a short or a long interval between the invasion of one and that of the other leg. In the more acute cases the leading symptoms are pain in the legs, sometimes in the loins and abdomen, sudden or rapidly manifested paraplegia, anaesthesia of the legs, absence of femoral pulsation, and phenomena of mortification extending from the feet upward. In several instances the patients, while walking, have been seized with excruciating pain in the legs, and have fallen paralysed to the ground. The pain is often atrocious and more or less paroxysmal. There may be tenderness on pressure over the occluded aorta. In a few cases pain has not been a prominent symptom.

Although the paraplegia has been repeatedly described as instantaneous in its appearance, it is to be inferred from the histories of carefully observed patients that at least a short interval of time and sometimes several hours and even days elapse before it is complete. In 44 cases in which there are definite statements about the motor power, there was complete or nearly complete paraplegia in 24; incomplete paralysis of both lower extremities, described in some instances merely as weakness, in 10; paralysis of only one leg in 5; and no paralysis in 5. The paralysis seems to be usually of the flaccid variety, but in some cases the paralysed muscles are stiff. In Barié's patient the paralysed legs were completely rigid, and it may be inferred that a condition analogous to rigor mortis had set in. With complete paralysis the reflexes and electrical excitability are abolished. Paralysis of the bladder and rectum, with retention of urine and involuntary evacuations, was observed in several cases, but not in the majority.

Of the cases with satisfactory histories in only 2 was there no disturbance of sensation. In some there was only numbness or some reduction of sensation, but in most there was definite anaesthesia, extending in some instances no higher than the knee,—more frequently to the middle or upper third of the thigh, and in 2 cases as high as Poupart's ligament. There was sometimes complete analgesia, which, however, did not exclude sensations of spontaneous pain in the legs. In many cases, however, there was hyperalgesia, either in the anaesthetic area or above it.

The symptom of greatest diagnostic value is absence of pulsation in the arteries of the lower extremities; but it is not pathognomonic, for it may occur in coarctation of the aorta and sometimes in abdominal aneurysm.

Wilbur observed excessive aortic pulsation above the obstruction. The legs become cold, and their surface temperature may even fall below that of the room (Browne, Manz). Absence of bleeding upon incision and of reactive hyperaemia after application of heat have been noted. The skin, at first pale, soon acquires a livid mottling, and the superficial veins may be dilated. Oedema of one or both legs and cutaneous haemorrhages are recorded in some of the histories. If the patient lives long enough gangrene usually ensues, and it may be manifest within twenty-seven to forty-eight hours. Gangrene was bilateral in at least 24 cases, and unilateral in 17. The extent of the gangrene varied greatly in different cases, being sometimes limited to the foot, sometimes reaching the middle of the thigh, and, in Bell's patient, involving the scrotum. Tympanites, diarrhoea, and albuminuria are common. Exceptional symptoms are the appearance of blood in the urine or stools, haematemesis, and priapism. Bed-sores appeared in many cases, and may appear within a few days from the onset.

Death may occur within twenty-four hours from the beginning of the attack. Fourteen patients died within the first four days, with collapse and rapid, weak, usually irregular pulse. There may be marked improvement in the initial symptoms either in one or in both legs. The larger number of patients die after a variable interval, which may extend over several weeks or even months, from gangrene, decubitus, and sepsis.

Of the deviations from the type may be especially mentioned incomplete manifestations of symptoms on one or both sides, transitory affection of one leg, limitation of the symptoms to one lower extremity only (4 cases), and affection of one leg followed after days, weeks, or months by that of the other (6 cases). The two cases reported by Barth and by Jean are considered particularly characteristic of slowly forming thrombosis. Here the first symptoms were chiefly numbness and intermittent claudication, which, after a long interval, deepened into paraplegia without gangrene.

All but 3 cases terminated fatally, more frequently from the remote effects than from the immediate shock of occlusion of the aorta. The three instances of survival with marked amelioration of all the symptoms are reported by Gull, Chvostek, and Nuñez. These cases began acutely with severe pains, paraplegia, disturbances of sensation, coldness and lividity of the lower extremities. The femoral pulse disappeared completely in Gull's and Nuñez's cases, but in Chvostek's it could still be felt, although it was feeble. In Chvostek's patients patches of superficial gangrene appeared; but in the other 2 cases there was no gangrene. Nuñez reports that after a year and a half there was no return of the femoral pulse on either side.

Since the demonstration by Schiffer and Weil, confirmed by Ehrlich and Brieger, Spronck, Herter, and others, that the paraplegia which follows immediately or very shortly after ligation of the abdominal aorta just below the renal arteries in rabbits (Stenson's experiment) is due to

ischaemia of the lumbar cord, many have assumed that the same explanation applies to the paraplegia in human beings after occlusion of the abdominal aorta. If the rabbit's aorta be tied for an hour, and the ligature be then removed, the paraplegia and paralysis of the bladder and rectum are permanent, the grey matter of the lumbar cord undergoes necrosis, and a genuine myelitis affecting chiefly the grey but also the white matter ensues. The same experiment gives negative results with the cat and usually with the dog. In view of the great interest of the subject, it is, to say the least, remarkable how few of the reports of necropsies on persons dead of embolism or thrombosis of the aorta have anything to say about the condition of the spinal cord. Roussel and Heiligenthal observed no macroscopic changes in the spinal cord. In Bell's and Barié and du Castel's cases the cord was microscopically normal, save congestion in the latter. Broca, Legroux, and Malbranc noted with the naked eye changes in colour, from which no definite conclusions can be drawn. The only detailed report of a microscopical examination of the cord is that of Helbing, who found, in the lumbar region of a man who lived thirty-nine days after embolism of the abdominal aorta, degeneration of the anterior and posterior nerve-roots more marked on one side than the other ; and degenerations in the cord for the most part explicable by the changes in the nerve-roots. The lesions of the cord were quite unlike those found in experimental cases, and are interpreted by Helbing as essentially analogous to those after amputation, and not referable to ischaemia of the cord.

As the matter now stands, there are no direct observations to support the opinion that the paraplegia following embolism or thrombosis of the abdominal aorta in human beings is caused by ischaemia of the cord, so that the old explanation which refers it to ischaemia of the peripheral nerves and muscles has the most in its favour. The question of the possibility of this mode of production of paraplegia, however, seems to me still open, and it is to be hoped that hereafter fatal cases of this rare condition will not be reported without satisfactory microscopical examination of the spinal cord. The anatomical investigations of Kadyi and of Williamson at least do not exclude the possibility that the lumbar cord in human beings is dependent to a considerable extent for its blood-supply upon the lumbar arteries.

The diagnosis of ischaemic paraplegia from spinal paraplegia can generally be made without difficulty by the absence of femoral pulsation, by the coldness and lividity of the extremities, and by the occurrence of gangrene in the former.

Embolism of Arteries of the Extremities.—Of the arteries of the extremities the popliteal and the femoral are the most frequent recipients of emboli. The results of embolism of arteries supplying the extremities are essentially similar to those of arterial thrombosis, which have already been considered (p. 743). The modifications resulting from the sudden advent of embolism are sufficiently self-evident. There may be severe pain at the moment of impaction and at the site of lodgment of the

embolus. The general principles involved in the differentiation of embolism from thrombosis have been presented under Diagnosis (p. 788).

Hepatic Infarction.—Although the intrahepatic branches of the hepatic artery and of the portal vein are terminal vessels, their capillary communications are so abundant that, as a rule, embolism or thrombosis of the hepatic vessels causes no interference with the circulation in the liver. Experiments of Cohnheim and Litten and of Doyon and Dufourt have demonstrated that complete interruption of the circulation through the hepatic arteries of the rabbit and the dog is followed by necrosis of the liver. Necrosis of the entire liver in man, as the result of embolism of the hepatic artery, has been recorded in rare instances (Chiari, Lancereaux).

Infarcts of the liver cannot be correlated with any clinical manifestations, but are of considerable pathological interest. They occur in various conditions, and are usually associated with some gross obstruction of the hepatic blood-vessels, but in extremely rare instances they are found in infective conditions without any occlusion of the larger blood-vessels. Hepatic infarcts may be haemorrhagic or anaemic, the former being the commoner. In 40 cases, including Chiari's 17 examples and most of Dr. Lazarus-Barlow's 32 cases, 29 were haemorrhagic, 10 anaemic, and in one of Dr. Pitt's cases both haemorrhagic and anaemic infarcts were present. An anaemic infarct of the liver was described as long ago as 1864 by Murchison. These two forms differ very considerably ; the white or anaemic infarcts shew complete necrosis of the liver-cells (Baldwin, Longcope), whilst the haemorrhagic infarcts do not. Anaemic infarcts may be associated with embolism of the hepatic artery (Baldwin, Ogle), with obstruction of the hepatic veins (Longcope), with obstruction of the portal vein, and may be produced by injuries which rupture the liver and cut off the blood-supply to parts of the organ (Klebs, Lubarsch, Lazarus-Barlow, Heile).

Haemorrhagic infarcts are wedge-shaped, rectangular, or irregular, and from their dark-red colour may, to the naked eye, resemble cavernomas ("naevi") of the liver. They are most often seen in connexion with occlusion of the portal vein by emboli or thrombosis ; out of Chiari's 17 cases there were 15 due to embolism. Köhler and Chiari found that the red colour is due mainly to dilatation of the intralobular capillaries, with atrophy of the liver-cells. Genuine coagulative necrosis is not present in the haemorrhagic infarcts. The affected areas are patches of circumscribed red atrophy rather than typical haemorrhagic infarcts. Zahn reproduced the same condition experimentally by emboli of sterilised mercury injected into the mesenteric veins ; the change in the liver did not begin until the eighth day, and was distinct after thirty-five days. It is probable that the areas do not undergo cicatrisation.

Rattone's theory, based upon experiments, that occlusion of branches of both the hepatic artery and portal vein is essential for the production of infarction of the liver, is not supported by the observations in human

beings. Klebs attributes the infarction to extensive capillary thrombosis. Köhler considers that the essential factor is the combination of occlusion of branches of the portal vein with obstruction to the return flow from the hepatic veins. Chiari and Steinhaus believe that the second factor, to be added to the plugging of portal branches, is feeble flow through the hepatic artery, from weakness of the general circulation. Wooldridge, by injecting coagulative tissue-extracts into the jugular vein of the dog, caused extensive clotting of blood in the portal vein and its branches, followed by numerous haemorrhages and necroses in the liver ; but the interpretation of these results as actual infarctions does not seem to me certain, inasmuch as these extracts in toxic doses produce a haemorrhagic diathesis, and may cause necroses in various situations independently of thrombosis. The focal necroses so often met with in the liver in various infective and toxic states do not usually stand in any definite relation to closure of the vessels (Welch and Flexner).

Embolism of the coronary arteries of the heart has already been considered (p. 120).

Embolism and Thrombosis of the Retinal Vessels.—Plugging of the retinal vessels is of general pathological as well as special ophthalmological interest, for it is possible to observe with the ophthalmoscope the circulatory disturbances in the retina. Ischaemia and stasis follow immediately closure of the central artery of the retina by an embolus. Vision is lost with characteristic suddenness. Both the arteries and the veins are narrowed, the latter being often unequally contracted. Subsequently the veins may dilate to some extent, especially in the periphery of the retina, and present ampulliform swellings. An interesting phenomenon is the appearance in the veins of an intermittent, sluggish stream of broken cylinders of red corpuscles, separated by clear spaces ; and by pressure on the eye-ball a similarly interrupted current may often be made to flow through arteries and veins. This appearance of interrupted columns of blood is evidently similar to that observed by Mall and myself after closure of the superior mesenteric artery and previously described (p. 779). After a short time the optic papilla becomes pale and grey, and the retina, especially in the neighbourhood of the papilla and macula, assumes an opaque, greyish-white, oedematous aspect. Haemorrhages are exceptional. A characteristic ophthalmoscopic appearance is the cherry-red spot in the centre of the macula, caused by the red colour of the choroid shining through. There may be more or less return of the circulation with improvement and even complete restoration of vision ; but the prognosis as regards sight is in general unfavourable, as atrophy of the retina and of the optic nerve is likely to ensue. The prognosis is more favourable with embolism of branches of the retinal artery. Here multiple haemorrhages usually occur.

Thrombosis of the central retinal vein is distinguished from plugging of the artery especially by the abundant haemorrhages. With occlusion of the central artery the condition is anaemic infarction, and with plugging of the vein haemorrhagic infarction.

There is some difference of opinion as to the relative frequency of embolism and of thrombosis of the central retinal artery. Of 129 cases collected by Fischer, 91 had heart disease; whereas Kern reports that of 12 cases in Haab's clinic only 2 had demonstrable cardiac disease; and of 83 cases, collected from the records, in 66 per cent there was no demonstrable source for an embolus. The latter author, therefore, regards the majority of plugs in the central artery of the retina as primary thrombi. The generally accepted opinion, however, is that embolism is more common than thrombosis of the retinal arteries.

Treatment.—In the preceding pages mention has been made of the surgical treatment of haemorrhagic infarction of the intestine and of gangrene of the extremities; and under "Thrombosis" the importance of preventing so far as may be the separation of emboli has been emphasised. The general indications in the treatment of embolism are essentially similar to those already considered for thrombosis (p. 756).

Surgical Treatment.—The brilliant experimental results obtained by Carrel in the suture of arteries has opened the field for the surgical treatment of embolism—namely, incision of the artery and removal of the embolus. This has been done by Trendelenburg, Sievers, and Ranzi in cases of pulmonary embolism, but so far without a satisfactory result. Arteriotomy with removal of the embolus has been carried out in the case of more accessible arteries by others (Handley, Moynihan).

<div style="text-align: right">WM. H. WELCH.   1899.</div>

<div style="text-align: right">H. D. ROLLESTON.   1909.</div>

REFERENCES[1]

Historical: 1. COHN, B. *Klinik d. embol. Gefässkrankh.*, Berlin, 1860.—2. B. COHNHEIM. *Untersuch. üb. d. embol. Processe*, Berlin, 1872.—3. *Idem. Vorles. üb. allg. Pathol.*, Berlin, 1892.—4. VIRCHOW. *Gesammelte Abhandl.*, Frankf., 1856.

Aberrant Embolism: 5. ARNOLD. *Virchows Arch.*, 1891, cxxiv. 385. — 6. BONOME. *Arch. per le sc. med.*, 1889, xiii. 267.—7. ERNST. *Virchows Arch.*, 1898, cli. 69.—8. FIRKET. *Acad. roy. de méd. de Belge*, 1890.—9. FLEXNER. *Bull. Johns Hopkins Hosp.*, 1896, vii. 173.—10. HAUSER. *München. med. Wchnschr.*, 1888, xxxv. 583.—11. HELLER. *Deutsch. Arch. f. klin. Med.*, 1870, vii. 127.—12. LUBARSCH. *Fortschr. d. Med.*, 1893, xi. 805.—13. LUI. *Arch. per le sc. med.*, 1894, xviii. 99.—14. RIBBERT. *Centralbl. f. allg. Path.*, 1897, viii. 433.—15. v. RECKLINGHAUSEN. *Virchows Arch.*, 1885, c. 503.—16. ROSTAN. Thèse, Genève, 1884.—17. SCHEVEN. Inaug.-Diss., Rostock, 1894.—18. SCHMORL. *Deutsch. Arch. f. klin. Med.*, 1888, xlii. 499.—19. *Idem. Path.-anat. Untersuch. üb. Puerp.-Eklampsie*, Leipz., 1893.—20. ZAHN. *Virchows Arch.*, 1889, cxv. 71, and cxvii. 1.

Anatomical Characters: 21. FAGGE. *Trans. Path. Soc.*, London, 1876, xxvii. 70.

Effects: 22. ASKANAZY. *Virchows Arch.*, 1895, cxli. 42.—23. BIER. *Ibid.* 1897, cxlvii. 256 and 444; 1898, cliii. 306, 434.—24. BRYANT. *Boston Med. and Surg. Journ.*, 1888, cxix. 400.—25. CERFONTAINE. *Arch. de biol.*, 1894, xiii. 125.—26. DAVAINE. *Traité des entozoaires*, Paris, 1877, 406.—27. FELTZ. *Schmidts Jahrb.*, 1870.—28. FISCHLER. *Centralbl. f. allg. Path. u. path. Anat.*, Jena, 1902. xiii. 417.— 29. v. FREY. *Arch. f. Physiol.*, 1885, 533.—30. GOLDENBLUM. *Versuche üb. Collateral-circulation, etc.*, Inaug.-Diss. Dorpat, 1889.—31. JACOBY. *Ztschr. f. physiol. Chem.*, Strassb., 1900, xxx. 149.—32. KÖPPE. *Arch. f. Physiol.*, 1890, Suppl.-Bd. 168.—33.

---

[1] The references are only to authors cited in the text, and are not intended to be a complete bibliography of the subject. The references to authors cited under different headings in the text will usually be found only under the first heading in which the reference appears.

Kossuchin. *Virchows Arch.*, **1876**, lxvii. 449.—34. Küttner. *Ibid.* 1876, lxi. 21 ; 1878, lxxiii. 476.—35. Lister. *Bull. Acad. de méd.*, 1878, 2 s., vii. 640.—36. Litten. *Ztschr. f. kl. Med.*, 1880, i. 131.—37. Mall. *Johns Hopkins Hosp. Rep.*, 1890, i. 37.— 38. Marchand. *Berl. kl. Wchnschr.*, 1894, xxxi. 36.—39. Nothnagel. *Ztschr. f. kl. Med.*, 1889, xv. 42.—40. Panski und Thoma. *Arch. f. exp. Path.*, 1893, xxxi. 303.—41. Ponfick. *Virchows Arch.*, 1873, lviii. 528.—42. v. Recklinghausen. *Handb. d. allg. Path. d. Kreislaufs, etc.*, Stuttg., 1883.—43. Thoma. *Lehrb. d. path. Anat.*, Th. i., Stuttg., 1894.—44. Weigert. *Virchows Arch.*, 1877, lxx. 486 ; 1878, lxxii. 250 ; 1880, lxxix. 104.—45. *Idem. Centralbl. f. allg. Path.*, 1891, ii. 785.—46. Welch. "Haemorrhagic Infarction," *Trans. Assoc. Amer. Physicians*, 1887, ii. 121.—47. Wells, H. G. *Journ. Med. Res.*, Boston, 1906, x. 149.—48. *Idem. Chemical Pathology*, 1907, 273, W. B. Saunders & Co.—49. Zielonko. *Virchows Arch.*, 1873, lvii. 436.

Embolic Aneurysms : 50. Buday. *Zieglers Beitr.*, 1891, x. 187.—51. Clarke. *Trans. Path. Soc.*, London, 1896, xlvii. 24.—52. Duckworth. *Brit. Med. Journ.*, 1890, i. 1355.—53. Eppinger. "Pathogenesis, Histogenesis u. Aetiologie d. Aneurysmen," *Arch. f. klin. Chir.*, Berl., 1887, xxxv., Suppl.-Hft., 1-563.—54. Langton and Bowlby. *Med.-Chir. Trans.*, 1887, lxx. 117. (Consult for references to Ogle, Wilks, Holmes, Church, Smith, Goodhart, and other previous literature.)— 55. Libman. "Cases of Mycotic Aneurisms," *Mount Sinai Hosp. Rep.*, N.Y., 1907, v. 481.—56. *Idem.* "Embolic Aneurism," *Ibid.*, 1907, v. 488.—57. Pel und Spronck. *Ztschr. f. klin. Med.*, xii. 327.—58. Thoma. *Deutsch. med. Wchnschr.*, 1889, xv. 362.—59. Tufnell. *Dubl. Quart. Journ. Med. Sc.*, 1853, xv. 371.

General Symptomatology : 60. Gangolphe et Courmont. *Arch. méd. expér.*, 1891, iii. 504.—61. Stricker. *Vorles. üb. allg. u. exper. Pathologie*, 770, Wien, 1883.

Air-Embolism : 62. Bert. *La pression barométrique, etc.*, Paris, 1878.—63. Boycott. "Caisson Disease," *Quarterly Journ. Med.*, Oxford, 1908, i. 348.—64. Boycott and Damant. "Some Lesions of the Spinal Cord produced by Experimental Caisson Disease," *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii. 507.—65. Couty. *Études expér. sur l'entrée de l'air dans les veines*, Thèse, Paris, 1875 (also for reference to Barthélemy).—66. Feltz. *Compt. rend.*, 1878, lxxxvi. No. 5.—67. Ewald und Kobert. *Pflügers Arch.*, 1883, xxxi. 160.—68. Hauer. *Ztschr. f. Heilk.*, 1890, xi. 159.—69. Heller, Mager, und H. von Schrötter. "Luftdruckerkrankungen," Wien, 1900.—70. Janeway. *Trans. Assoc. Amer. Physicians*, 1898, xiii. 87.—71. Jürgensen. *Deutsch. Arch. f. klin. Med.*, 1882, xxxi. 441.—72. Laborde et Muron. *Compt. rend. Soc. biol.*, 1873, v. 84.—73. Lewin. *Arch. f. exp. Path. u. Pharm.*, 1897, xl. 308.—74. Passet. *Arb. a. d. path. Inst. zu München*, 293, Stuttg., 1886.—75. Welch. *Johns Hopkins Hosp. Bull.*, Balt., 1900, ix. 185.—76. Welch and Flexner. *Journ. Exp. Med.*, 1896, i. 5.—77. Welch and Nuttall. *Johns Hopkins Hosp. Bull.*, 1892, iii. 81.—78. Wolf. *Virchows Arch.*, 1903, clxxiv. 454.

Fat-Embolism : 79. Beneke. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1897, xxii. 343.—80. Brenzinger. *Wien. klin. Rundschau*, 1906.—81. Connell. *Journ. Am. Med. Assoc.*, Chicago, 1905, xliv. 612.—82. Hanriot. *Compt. rend. Acad. d. sc.*, 1896, cxxii. 753 ; cxxiii. 833 ; 1897, cxxiv. 255 and 778.—83. Ribbert. *Correspondenz-Bl. f. schweiz. Aerzte*, 1894, xxiv. 457.—84. Sanders and Hamilton. *Edin. Med. Journ.*, 1879-80, xxv. 47.—85. Wagner. *Arch. d. Heilk.*, 1862, iii. 241 ; 1865, vi.— 86. Zenker. *Beitr. z. norm. u. path. Anat. d. Lunge*, Dresden, 1862.

Embolism by Parenchymatous Cells : 87. Aschoff. *Virchows Arch.*, 1893, cxxxiv. 11.—88. Gaylord. *Proc. Path. Soc.*, Philadelphia, 1898, N.S. i. 184.—89. Hanau. *Fortschr. d. Med.*, 1886, iv. 387.—90. Lubarsch. *Fortschr. d. Med.*, 1893, xi. 805 and 845 (consult for references to Turner, Jürgens, Klebs, Zenker, and Hess). —91. *Idem. Virchows Arch.*, 1898, cli. 546.—92. Maximow. *Virchows Arch.*, 1898, cli. 297 (also for references to Leusden and Kassjanow).—93. Neumann. *Monatschr. f. Geburtsh. u. Gynäkol.*, 1897, vi. 17, 157.—94. Pick. *Berl. klin. Wchnschr.*, 1897, xxxiv. 1069 (also for Schmorl).—95. Schmidt, B. *Centralbl. f. allg. Path.*, 1897, viii. 860.—96. Schmorl. *Zentralbl. f. Gynäk.*, Leipz., 1905, xxix. 129.

Embolism by Paraffin : 97. Paget, S. *Trans. Clin. Soc.*, London, 1903, xxxvi. 128.

Pulmonary Embolism : 98. Bang. *Iagttagelser og Studier over dödelig Embolie og Thrombose i Lungearterierne.* Copenhagen, 1880.—99. Box. *Trans. Clin. Soc.*, London, 1906, xxxix. 189.—100. Bünger. *Ueb. Embolie d. Lungenarterie.* Inaug.-Diss.,

Kiel, 1895.—101. COHNHEIM und LITTEN. *Virchows Arch.*, 1875, lxv. 99.—102. FREYBERGER. *Trans. Path. Soc.*, London, 1898, xlix. 27.—103. FUJINAMI. *Virchows Arch.*, 1898, clii. 61, 193 (also for Oestreich).—104. GARNIER et JOMIER. "Les embolies hydatiques," *Presse méd.*, Paris, 1905, xiii. 369.—105. GLUZINSKI. *Deutsch. Arch. f. kl. Med.*, 1895, liv. 178 (for references to Perl and Lippmann, Sommerbrodt, and Nothnagel).—106. GRAWITZ. *Virchows Festschrift der Assistenten*, Berlin, 1891. —107. GSELL. *Mittheil. a. Klinik. u. med. Inst. d. Schweiz*, iii. R. Hft. 3 (also for Hanau).—108. HAMILTON. *A Textbook of Pathology*, i. 683, London, 1889.— 109. KÜTTNER. *Virchows Arch.*, 1878, lxxiii. 39.—110. KLEBS. *Allg. Path.*, ii. 20. Jena, 1889.—111. LENORMANT. *Arch. gén. de chir.*, Paris, 1909, iii. 234.— 112. LICHTENBERG. *Centralbl. f. d. Grenzgeb. d. Med. u. Chir.*, 1908, xi.—113. LITTEN. *Charité-Ann.*, 1878, iii., 1876, 180.—114. MAUCLAIRE. *Arch. gén. de chir.*, Paris, 1908, ii. 573.—115. MÖGLING. *Zieglers Beitr.*, 1886, i. 133 (see also No. 106). —116. MÜHSAM. Quoted by LENORMANT.—117. ORTH. *Centralbl. f. allg. Path.*, 1897, viii. 589.—118. PERL. *Virchows Arch.*, 1874, lix. 39.—119. SERRE. *De l'origine embolique des thromboses de l'artère pulmon.* Thèse, Lyon, 1895.—120. SGAMBATI. *Arch. ed atti d. Soc. ital. di chir.*, Roma, 1897, xi. 37.—121. WAGENER. *Deutsches Arch. f. klin. Med.*, Leipz., 1907, lxxxix. 626.—122. WILLGERODT. *Arb. a. d. path. Inst. in Göttingen*, Berlin, 1893, p. 100.—123. ZAHN. *Centralbl. f. allg. Path.*, 1897, viii. 860.

Splenic Infarction. Renal Infarction : 124. HALPERIN. "Clinical Manifestations of Renal Infarction," *Arch. Int. Med.*, Chicago, 1908, i. 320.—125. LEMAIRE. *Gaz. hebd. d. sc. méd. de Bordeaux*, 1908.—126. PONFICK. *Virchows Arch.*, 1874, lx. 153.—127. THOREL. *Ibid.* 1896, cxlvi. 297.—128. TRAUBE. *Gesammelte Beitr. z. Path. u. Physiol.*, Berlin, 1871, ii. 347.

Embolism and Thrombosis of the Mesenteric Arteries : 129. BECKMANN. *Virchows Arch.*, 1858, xiii. 501.—130. CHIENE. *Journ. Anat. and Physiol.*, 1869, iii. 65. —131. COUNCILMAN. *Boston Med. and Surg. Journ.*, 1894, cxxx. 410.—132. ELLIOT. *Ann. Surg.*, 1895, xxi. 9.—133. FABER. *Deutsches Arch. f. kl. Med.*, 1875, xvi. 527. —134. FINLAYSON. *Glasgow Med. Journ.*, 1888, xxix. 414.—135. GALLAVARDIN. *Gaz. des hôp. de Paris*, 1901, 929, 957.—136. GERHARDT. *Würzb. med. Ztschr.*, 1863, iv. 141.—137. JACKSON, PORTER, and QUINBY. *Journ. Amer. Med. Assoc.*, Chicago, 1904, xlii. 1469 and xliii. 25, 113, 183.—138. KARCHER. *Correspondenz-Bl. f. schweiz. Aerzte*, 1897, xxvii. 548.—139. KAUFMANN. *Virchows Arch.*, 1889, cxvi. 353.—140. KUSSMAUL. *Würzb. med. Ztschr.*, 1864, v. 210.—141. LEREBOULLET. *Rec. de mém. de méd.*, 1875, xxxi. 417.—142. LITTEN. *Virchows Arch.*, 1875, lxiii. 289.—143. *Idem. Deutsche med. Wchnschr.*, 1889, xv. 145.—144. LORENZ. *Ztschr. f. klin. Med.*, 1891, xviii. 493.—145. LYCETT. *Brit. Med. Journ.*, 1898, ii. 84.—146. MAYLAND. *Brit. Med. Journ.*, 1901, ii. 1454.—147. MAUCLAIRE et JACOULET. *Arch. gén. de chir.*, Paris, 1908, ii. 213.—148. MINKOWSKI. *Mitth. a. d. med. Klin. zu Königsberg*, 1888, 59.—149. MOOS. *Virchows Arch.*, 1867, xli. 58.—150. NOTHNAGEL. *Spec. Path. u. Therap.*, xvii. 156, Wien, 1898.—151. OPPOLZER. *Allg. Wien. med. Ztg.*, 1862, vii.—152. OSSWALD. *Ztschr. f. klin. Med.*, Berlin, 1904, liii. 308.—153. PACKARD. *Proc. Path. Soc. Philadelphia*, 1898, N.S. i. 288.—154. PARENSKI. *Wiener med. Jahrb.*, 1876, 275.—155. PIEPER. *Allg. med. Centr. Ztg.*, 1865, 493.—156. PONFICK. *Virchows Arch.*, 1870, l. 623.—157. TANGL und HARLEY. *Centralbl. f. d. med. Wiss.*, 1895, 673.—158. TIEDEMANN. *Von d. Verengerung u. Schliessung d. Pulsadern in Krankheiten*, Heidelb. u. Leipz., 1843.—159. WATSON. *Boston Med. and Surg. Journ.*, 1894, cxxxi. 552.—160. WELCH and FLEXNER. *Journ. Exp. Med.*, 1896, i. 35.

Embolism and Thrombosis of the Thoracic Aorta : 161. ARMET. Thèse de Paris, 181.—162. CHVOSTEK. *Wiener med. Blätter*, 1881, 1513 (also for references to Trost, Carville, Lüttich, and Tewat).—163. BOCHDALEK. *Vrtljschr. f. d. prakt. Heilk.*, 1845, viii. 160.—164. BRISTOWE. *Lancet*, 1881, i. 131 and 166.—165. JAURAND. *Prog. méd.*, 1882, x. 147.—166. PITT. *Trans. Path. Soc.*, London, 1889, xl. 74.

Embolism and Thrombosis of the Abdominal Aorta : 167. BARIÉ et HALBRON. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1903, xx. 794.—168. CAMMARERI. *Morgagni*, 1885, xxvii. i. 113.—169. CHARRIER et APERT. *Bull. Soc. anat. de Paris*, 1896, 5 s., x. 766.—170. GRAHAM. *Med.-Chir. Trans.*, 1814, v. 297.—171. HAWKINS, F. H. *Trans. Clin. Soc.*, London, 1906, xxxix. 135.—172. HEILIGENTHAL. *Deutsche med. Wchnschr.*, 1898, xxiv. 519.—173. MAZOUX. *Thèse de Paris*, No. 256, 1904-5.—174. MEYNARD. *Étude sur l'oblitération de l'aorte abdom. par embolie ou par thrombose.* Thèse,

Paris, 1883 (Meynard's case is identical with Barié's and du Castel's).—175. RÉNON, CLAISSE, et ABRAMI. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1905, xxii. 349.—176. ROUSSEL. *Études sur les embolies de l'aorte abdom.* Thèse, Lyon, 1893 (the cases of Barié and of Desnos reckoned as separate cases by Selter and by Roussel are identical). —177. SELTER. *Ueb. Embolie d. Aorta abdom.* Inaug.-Diss., Strassburg, 1891.—178. VIGOUROUX et CHARPENTIER. *Bull. Soc. anat.*, Paris, 1903, 6 s., v. 393—179. WAGENER. *Deutsches Arch. f. klin. Med.*, Leipz., 1907, lxxxix. 626. (The references to the additional twelve cases of embolism or thrombosis of the abdominal aorta are Nos. 180 to 191 inclusive.)—180. BALLINGALL. *Trans. Med. and Phys. Soc. Bombay*, 1857, N.S. No. iii. App. p. xxv.—181. BRISTOWE. *Trans. Path. Soc.*, London, 1872, xxiii. 21.—182. CARTER. *Trans. Med. and Phys. Soc. Bombay* (1859), 1860, N.S. No. v. App. p. xxii.—183. GOODWORTH. *Brit. Med. Journ.*, 1896, i. 1501.—184. KIRKMAN. *Lancet*, 1863, ii. 510.—185. MANZ. *Berl. klin. Wchnschr.*, 1889, xxvi. 812.—186. NUÑEZ. *Gac. méd. de la Habana*, 1879-80, ii. 160.—187. OSLER. *Trans. Assoc. Amer. Physicians*, 1887, ii. 135.—188. PETTIT. *New Orleans Med. and Surg. Journ.*, 1880-81, N.S. viii. 1151.—189. SCHILLING. *München. med. Wchnschr.*, 1895, xlii. 227.—190. SCHOLZ. *Ein Fall von Obturation d. Aorta abdom.* Inaug.-Diss., Tübingen, 1850.— 191. WILBUR. *Amer. Journ. Med. Sc.*, Phila., 1857, N.S. xxxiv. 286.—192. BARTH. *Bull. Soc. anat. Paris*, 1848, xxiii. 260.—192a. DAGGETT. *Yale Med. Journ.*, 1909, xv. 418.—192b. FAWCETT. *Guy's Hosp. Gaz.*, 1906, xx. 306.—193. HERTER. *Journ. Nervous and Mental Dis.*, 1889, xvi. 197 (for references to Schiffer, Weil, Ehrlich and Brieger, and Spronck).—194. HERXHEIMER. *Virchows Arch.*, 1886, civ. 20.— 195. KADYI. *Ueb. d. Blutgefässe d. menschl. Rückenmarks*, Lemburg, 1889.—196. LITTEN. *Virchows Arch.*, 1880, lxxx. 281.—197. POPOFF. *Arch. f. Gynäk.*, 1894, xlvii. 12.—198. WILLIAMSON. *On the Relation of Diseases of the Spinal Cord to the Distribution and Lesions of the Spinal Blood-Vessels*, London, 1895.

Hepatic Infarction : 199. ARNOLD. *Virchows Arch.*, 1891, cxxiv. 388.—200. BALDWIN. *Journ. Med. Res.*, Boston, viii. 431.—201. CHIARI. *Centralbl. f. allg. Path.*, 1898, ix. 839.—202. COHNHEIM und LITTEN. *Virchows Arch.*, 1876, lxvii. 153. —203. DOYON et DUFOURT. *Arch. de physiol.*, 1898, 5 s., x. 522.—204. FLEXNER. *Johns Hopkins Hosp. Rep.* 1897, vi. 259.—205. HEILE. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1900, xxviii. 443.—206. KLEBS. *Virchows Festschrift der Assistenten*, 1891, 8.—207. KÖHLER. *Arb. a. d. path. Inst. in Göttingen*, 1893, 121.—208. LANCEREAUX. *Traité des maladies du foie et du pancreas*, 1899, 541.—209. LAZARUS-BARLOW. *Brit. Med. Journ.*, 1899, ii. 1342.—210. LONGCOPE. *Univ. Penna. Med. Bull.*, Phila., 1901, xiv. 223.—211. LUBARSCH. *Fortschr. d. Med.*, 1893, xi. 809.— 212. MURCHISON. *Trans. Path. Soc.*, London, 1864, xv. 132.—213. OGLE. *Trans. Path. Soc.*, London, 1898, xlvi. 73.—214. OSLER. *Trans. Assoc. Amer. Phys.*, 1887, ii. 136.—215. PITT. *Trans. Path. Soc.*, London, 1895, xlvi. 75.—216. RATTONE. *Arch. per le sc. med.*, 1888, xii. 223.—217. STEINHAUS. *Deutsch. Arch. f. klin. Med.*, Leipz., 1904, lxxx. 364.—218. WELCH and FLEXNER. *Johns Hopkins Hosp. Bull.*, 1892, iii. 17.—219. WOOLDRIDGE. *Trans. Path. Soc.*, London, 1888, xxxix. 421.—220. ZAHN. *Centralbl. f. allg. Path.*, 1897, viii. 860.

Embolism and Thrombosis of the Retinal Vessels : 221. FISCHER. *Ueb. d. Embolie d. Art. centr. Retinae.* Leipzig, 1891.—222. KERN. *Zur Embolie d. Art. centr. Retinac.* Inaug.-Diss. Zürich, 1892.

Surgical Treatment : 223. HANDLEY. *Brit. Med. Journ.*, 1907, ii. 712.—224. MOYNIHAN. *Ibid.*, 1907, ii. 826.—225. RANZI. *Arch. f. klin. Chir.*, Berlin, 1908, lxxxvii. 380.—226. SIEVERS. *Deutsche Ztschr. f. Chir.*, Leipz., 1908, xciii.—227. TRENDELENBURG. *Arch. f. klin. Chir.*, Berlin, 1908, lxxxvi. 686.—228. *Idem. Deutsche med. Wchnschr.*, 1908, xxxiv. 1172.

W. H. W.
H. D. R.

DISEASES OF THE LYMPHATIC VESSELS

By H. D. ROLLESTON, M.D., F.R.C.P.

UNDER this heading an account will be given (i.) of the morbid con-
ditions of the thoracic duct, especially of obstruction, and of chylous
and chyliform effusions ; (ii.) of diseases of the other lymphatic vessels.

DISEASES OF THE THORACIC DUCT

Affections of the thoracic duct are of importance from two points
of view : (i.) by producing obstruction to the outflow of lymph into the
general circulation (*vide* p. 823); and (ii.) by conveying lymphatic
infection, and thus leading to haemic infections.

Inflammation.—*Acute inflammation* is rarely recognised or described.
Infection and acute inflammation may spread into the duct from the
abdominal lymphatics, and septicaemia and pyaemia of intestinal origin
may be thus initiated.   Warthin, who gives a full account of this sub-
ject, describes a case of pyaemia secondary to salpingitis, in which
the mesenteric lymphatics, receptaculum chyli, and the thoracic duct
shewed suppurative inflammation.   A case of suppurative inflammation
of the thoracic duct apparently due to food poisoning, and imitating
enteric fever, is described by De Forest.   The receptaculum chyli
resembled a Bologna sausage, and contained offensive pus of a bluish
colour, which could be made to trickle through the valve at the junction
of the left subclavian and jugular veins.   The kidneys were in a con-
dition of toxic nephritis.   Warthin suggests that the condition described
by Nockher as a "gangrenous thoracic duct" in a patient dead of a
"malignant epidemic fever" may have been of the same nature.

Chronic inflammation of the thoracic duct, apart from that accompany-
ing filariasis and tuberculosis, is very seldom described.   It is probable
that the duct may be involved by inflammation spreading from
adjacent strictures, and that some cases of stricture of the duct are
thus explained.   In 1903 a case of chronic obliterative inflammation
of the duct with resulting chylous ascites was described by Comey
and M'Kibben.

Tuberculosis of the thoracic duct occurs much more often than
has been generally recognised, and is important in the dissemina-
tion of tuberculosis (Ponfick, Benda).   Tubercle bacilli pass from
the abdominal lymphatic glands into the thoracic duct, and may
infect its wall and produce infiltration, thickening, and even complete
obstruction, as in Sir W. Whitla's case in which rupture resulted.
Tubercle bacilli more often pass up the thoracic duct without producing

definite morbid changes in its walls, and so enter the general circulation ; examination of smears from the contents of the thoracic duct shews that tubercle bacilli are found in 75 per cent of the cases of generalised miliary tuberculosis (Longcope), and constantly in tuberculosis of the abdominal lymphatic glands (Whipple). From his observations Whipple concludes that the thoracic duct is of more importance in the dissemination of tubercle bacilli in subacute than in acute miliary tuberculosis. The intestinal origin of pulmonary tuberculosis has been fully discussed by Dr. Bulloch in the section on the "Paths of Infection in Pulmonary Tuberculosis" (Vol. V. p. 305).

Haemorrhage into the thoracic duct may occur as the result of bleeding into the gastro-intestinal tract or from injury. Its only importance is that thrombosis may occur and give rise to obstruction.

Malignant Disease.—*Primary malignant disease* of the thoracic duct does not appear to have been described, but a primary endothelioma of the receptaculum chyli might be expected.

Secondary malignant disease of the thoracic duct has been estimated to occur in 12 per cent of the cases of intra-abdominal malignant disease (Nattan-Larrier). Both carcinoma and sarcoma give rise to secondary implication of the duct ; but as infection commonly spreads from the abdominal viscera, secondary carcinoma is more often seen.

The thoracic duct is usually somewhat irregularly enlarged in cases of secondary malignant disease, and presents nodular masses at intervals. It is often occluded, but lymphatic obstruction is usually obviated by the free anastomosis. The spread of intra-abdominal malignant disease to the thoracic duct is of importance, as in this way the glands above the left clavicle may become infected, either by the direct spread of growth along the walls of the thoracic duct, or possibly by emboli of tumour-cells becoming regurgitated into these glands (Stevens). Malignant disease implicating the thoracic duct may, by the accompanying obstruction, lead to chylous ascites (*vide* p. 827).

Secondary malignant disease of the right lymphatic duct may occur in carcinoma of the mamma, and give rise to lymphatic oedema of the arm.

Obstruction of the Thoracic Duct.—The extreme freedom of the anastomotic communications of the lymphatic vessels forming the tributaries of the thoracic duct, and the fact that the lymphatic system is not a closed scheme of vessels but is in continuity with the interstices of the tissues, must be taken into account in considering the subject of obstruction of the thoracic duct. It is remarkable how frequently all signs of it are absent, when, from the presence of tumours, aneurysms, or dense adhesions, it might naturally be expected. In addition to the compensatory efforts on the part of collateral lymphatic anastomoses some complementary absorption is accomplished by the venous channels ; and it is highly probable, therefore, that concomitant venous obstruction plays an important part in the production of dilated lymphatics and oedema. Obstruction may take place in any part of the course of the thoracic

duct; but the effects are more often noticed when the interference with the flow of its contents is near the termination of the duct which is usually in the left internal jugular vein, close to its junction with the subclavian vein. This may be due partly to the greater frequency of tumour and other causes leading to obstruction in this situation; partly to the consolidation of the duct here into a single trunk; and partly, again, to the greater difficulty of a compensatory collateral circulation between the parts of the duct above and below the obstruction, than in the case of obstruction lower down. Tumours or inflammatory formations in the superior mediastinum would be likely to interfere with those lymphatic trunks in the anterior mediastinum which convey lymph from the peritoneal cavity, and eventually open into the thoracic duct or right lymphatic duct.

Causes of Obstruction to the Thoracic Duct.—Mediastinal growths, especially those in the anterior mediastinum, or glands enlarged from tuberculous, lymphadenomatous, or malignant infection, may compress the duct from without. Cicatricial adhesions, the results of past inflammatory processes, may have the same effect, but rarely do.

Aneurysm of the aorta is an extremely rare cause of obstruction of the thoracic duct. The gradual increase of the size of the sac, and the absence of any infiltration, probably allow a collateral circulation to develop. There may, however, be great dilatation of the lymphatics in the abdomen, as was noted by Morgagni (p. 650).

Sir S. Wilks mentions an exceptional case of exophthalmic goitre in which the enlarged thyroid gland passed deeply into the thorax, and was thought, by pressure on the duct, to account for the extreme emaciation.

Thrombosis of the left innominate vein has been recorded as a cause of obstruction of the thoracic duct (Ormerod, S. Martin). This lesion, though by no means excessively rare, necessarily obstructs the outflow of lymph from the duct; and, unless free anastomosis with the right lymphatic duct is established, the effects of backward pressure in the lymphatic system must follow. If the thrombosis extend into the duct itself these results would be rendered more marked. In tricuspid incompetence backward pressure has, in a few isolated cases, led to lymphatic stagnation in the thoracic duct. Here, again, it is possible that thrombosis in the duct might have occurred, and so given rise to obstruction. Obliterative endophlebitis of the subclavian vein has been found to cause obstruction of the thoracic duct. Thrombosis of the thoracic duct may be due to inflammation either starting in its walls or spreading to it by extension from adjacent structures. It may also be due to coagulation of blood effused into the duct from injury, or possibly from regurgitation from the internal jugular vein in extreme backward pressure. As just mentioned, thrombosis may spread into the duct from the large veins; further, it may be due to malignant invasion of the duct, or to parasitic invasion.

Changes in the walls of the duct, such as infiltration with tuber-

culous or inflammatory products, may cause obstruction. Congenital stenosis has been suggested, and, if it occurs at all, is best explained as the result of some inflammation early in life. Secondary malignant disease of the thoracic duct, contrary to what might be expected, appears very rarely to give rise to dilatation of the distal lymphatics; thus, in 13 cases collected by Nattan-Larrier lymphatic stasis occurred in one only. As in aneurysm, there is probably sufficient time to enable a competent collateral circulation to become established.

In filarial disease the duct is probably obstructed by the parent worms (*vide* Vol. II. Part II. p. 944).

The results of obstruction to the flow of lymph through the thoracic duct, like those of the backward pressure in the venous system, are widespread. Lymphatic stagnation leads to dilatation and opening up of a collateral circulation; if for any cause this fail to compensate, transudation, leakage, and, from rupture of its tributaries or even of the thoracic duct or receptaculum, free escape of lymph will follow. The dilatation of the lymphatics, or lymphangiectasis, may be very diffuse; its occurrence in different parts of the body and its relation to lymphangioma will be referred to on p. 837 *et seq.*

Elephantiasis resembling that due to filariasis except in its causation sometimes arises in this country. Its origin is often obscure; it may sometimes be due to recurrent attacks of infective lymphangitis, to inflammation of the retroperitoneal glands, or to chronic inflammatory thickening of the retroperitoneal tissues. In a woman at one time under my care extreme elephantiasis was found to be due to prevertebral fibrosis surrounding the lymphatics about the level of the receptaculum chyli; there was also chronic peritonitis (Bernstein and Price). Cases described as congenital elephantiasis really belong to a different category, namely, to persistent hereditary trophoedema or Milroy's disease, to which reference will be made in the article on "Tropho-neuroses of Soft Parts," in Vol. VII. They differ from elephantiasis in that the skin rarely shews the characteristic thickening associated with lymphatic obstruction.

The general features of elephantiasis arising in this country so closely resemble those of filarial origin, that the reader should refer to the account given under that heading (Vol. II. Part II. p. 945). It may be added that Mr. Handley has treated elephantiasis successfully by lymphangioplasty.

Chylous Ascites.—Chylous ascites is due to the escape of chyle into the peritoneal cavity, whether from transudation of chyle through distended lacteals or from rupture of the thoracic duct, of the larger lymphatic trunks, of varicose lymph-vessels, or of a lymphangioma. It is a rather uncommon condition, and in the past its existence has been questioned; but it is not so rare as might be gathered from a total of 68 recorded cases, all that Dr. Batty Shaw could collect in 1900.

There is an allied form of ascites in which the fluid contains fat-globules, and thus resembles chylous ascites; but there is no evidence

that it comes from the lymphatic system. This condition, to which further reference will be made, is called *chyliform or fatty ascites* (p. 829), in contradistinction to true chylous ascites. In yet another variety of ascites the naked-eye appearances resemble those in the former groups ; but analysis shews that there is no fat, or only a trace, and that the milkiness is due to some other cause : such cases are described as milky non-fatty ascites (p. 830).

Between these three kinds of ascites a good deal of confusion exists, and cases examined with the naked eye only may belong to any one of them. Since the distinction between chylous and chyliform ascites may be difficult, a mixed class has been described, in which the fluid partakes of the characters of both categories.

True Chylous Ascites.—The *fluid* has a specific gravity, varying from 1007 to 1040, but usually about 1015. It is alkaline in reaction and rarely neutral or amphoteric. The fluid resists putrefactive changes for a long time, and is usually devoid of smell ; though occasionally the odour of foods taken by the mouth may become apparent. It does not clot spontaneously on standing, and thus differs from the urine in chyluria, which owes its coagulating power largely to admixture with blood ; but it separates into layers, the uppermost being creamy from the supernatant fat and readily soluble in ether. The milkiness and opalescence are due to the presence of minutely-divided fat, the emulsion being much finer than in chyliform or fatty ascites wherein distinct globules of oil are seen. The quantity of fat varies in different cases, and under different conditions in the same individual ; if the diet contain much fatty food the amount in the peritoneal effusion will be enhanced : thus Straus, in a patient fed on butter, noticed a considerable increase in the percentage of fat in the chylous ascitic fluid, and was able to recognise in it the fat given by the mouth. On an average fat is present to the extent of 1 per cent, whilst in chyle it is 0·9 per cent.

The percentage of protein is usually about 3 per cent : in Prof. S. Martin's case it was 4·46 ; in Hoppe-Seyler's 3·66 per cent ; in Gillespie's 3·2 per cent ; and in two analyses quoted by Prof. Halliburton 2·9 per cent and 2·1 per cent respectively. If peritonitis be present the increased exudation would lead to a higher percentage. Admixture with peritoneal fluid and lymph from other sources renders the constitution of the fluid in true chylous ascites different from that of chyle. The total solids come to between 4 and 6 per cent.

Sugar, which is generally present in lymph, and consequently in chyle, and in peritoneal fluid, may be, but is not constantly, found in true chylous ascites. It has been thought that its presence, in the absence of glycosuria, would differentiate true chylous from chyliform ascites ; but this criterion can hardly be of much value, for sugar may occur in other peritoneal exudations ; whilst, on analysis of the chylous fluid in the abdomen, in Straus's case of chylous ascites with definite perforations on the lymphatic trunks of the mesentery, no sugar was

found.   Again, in fatty ascites, in which much cell-metabolism has taken place with discharge of the contents of the cells into the peritoneal fluid, copper-reducing substances would probably be readily produced.

Causes of Chylous Ascites.—Obstruction to the flow of lymph in any part of the course of the thoracic duct or right lymphatic duct naturally tends to produce increased pressure in the lymphatic vessels behind the obstruction, and dilatation of them ; this may be followed by the opening up of a collateral circulation, so that the lymph eventually enters the general circulation ; or, on the other hand, if this means of compensation fail, the pressure in the obstructed lymph-channels may lead to leakage by transudation into the peritoneal cavity or tissues around ; or even to a grosser lesion, such as rupture or perforation of the thoracic duct or its tributaries.   The stomata of the thin-walled lymphatic vessels offer a ready means by which free transudation can take place.

In considering the causes of chylous ascites *seriatim*, the various ways in which the thoracic duct may be obstructed in the different parts of its course must be first borne in mind.   These have already been referred to, and need not be rehearsed again (*vide* Obstruction of the Thoracic Duct, p. 824).   When the receptaculum chyli is involved, the thoracic duct above may be quite healthy, and lymph may then pass into it by anastomotic channels, and no chylous ascites be induced.

The thoracic duct and the receptaculum chyli are liable to be pressed upon or directly invaded by malignant growths or other formations in their immediate neighbourhood.   But although chylous, and especially chyliform, ascites is often associated with malignant disease of the peritoneum, the association of new growth invading the thoracic duct and chylous ascites, probable as it might seem at first sight, has been seldom established.   Schramn describes such a case, but in 13 cases of secondary malignant disease of the thoracic duct collected by Nattan-Larrier there was no example of true chylous ascites, and the three cases of this series shewing chyliform ascites were due to malignant disease of the peritoneum.   In three other cases in which the thoracic duct was found to be invaded by malignant disease the concomitant ascites was serous in two (Troisier), and chyliform from malignant disease of the peritoneum in the other (Menetrier and Gauckler).   In filariasis the parent worms probably obstruct the lower part of the thoracic duct, but chylous ascites is much less frequent than the other well-known manifestations of the disease (*vide* Vol. II. Part II. p. 944).

Traumatic rupture of the thoracic duct is a very rare accident ; in Quincke's case, in a man run over by a cart, chylous effusions into the right pleura and peritoneum followed ; the division of the duct by a stab has been recorded in very few cases.

Wilhelm has recorded what may have been a rupture during the paroxysms of whooping-cough in an infant six months old.   Busey refers to a case in which primary rupture of the thoracic duct was attributed

to vomiting, and to three in which muscular effort was the reputed cause. Injury and rupture in the intrathoracic portion of the thoracic duct, by leading to haemorrhage and thrombosis inside the duct, may produce obstruction to the flow of lymph from the abdomen. Rupture of the thoracic duct below the point of obstruction has been more often assumed than demonstrated in cases of chylous ascites. In Cayley's case, in which the obstruction was at the entrance into the jugular vein, the receptaculum chyli was ruptured ; and, in addition to chylous ascites, a large effusion of lymph took place behind the peritoneum. In Sir W. Whitla's well-known case the middle third of the thoracic duct was obstructed by tuberculous infiltration, and the receptaculum chyli had ruptured. In 27 cases of chylous ascites, collected by Busey, rupture was found 11 times ; this includes secondary rupture from obstruction as well as primary rupture from trauma and so forth.

Malignant disease involving the aortic or mesenteric glands may produce dilatation of the lymphatics. In Straus's case rupture of two lymphatic trunks on the anterior surface of the mesentery gave rise to chylous ascites. But the point of escape of the chyle is, generally speaking, difficult to find ; and in many cases the chylous effusion is presumably a general oozing or transudation. Dilatation of the chyle vessels is by no means always present when chylous ascites and intra-abdominal malignant disease coexist ; but this does not prove that escape of chyle has not occurred, for the obstruction may involve small vessels which have subsequently emptied themselves into the peritoneal cavity, and so are no longer apparent or prominent at the necropsy. On the other hand, the milky effusion in malignant disease of the peritoneum may be chyliform or fatty, rather than due to an escape of real chyle. This point will be discussed later ; but it may be pointed out in passing that it is often very difficult to gather from the records whether the effusion in any individual instance contained small fatty particles (chyle) or larger globules derived from changes in suspended cells—the mere presence of fat being enough to lead to the effusion being called chylous. Malignant disease of the abdomen appears to be the most frequent cause or concomitant of chylous ascites ; thus in 33 cases, collected by Treigny, it was responsible for 10.

Chronic peritonitis, by contracting widely on the smaller lymphatic trunks and producing a change in their walls, might conceivably give rise to transudation of their contents, or even to minute ruptures.

In a few cases chylous ascites has been referred to the backward pressure of heart lesions leading to thrombosis of the jugular vein into which the thoracic duct opens (Ormerod, Sidney Martin) ; to thrombosis of lymph in the thoracic duct from stagnation without venous throm-bosis (Oppolzer) ; or merely to the mechanical effects of lymphatic stagnation (Rokitansky). It has also been described in rare instances in hepatic cirrhosis. In Merklin's case of cirrhosis chylous ascites followed a fall, and thus suggests the possibility of laceration of lymphatic vessels, though this was not made out at the necropsy. In

exceptional instances chylous ascites is due to rupture of a chylous mesenteric cyst.

Fatty or Chyliform Ascites.—Closely resembling chylous ascites in appearance, and in the presence of suspended fat, but differing from it in that (*a*) the fat is present in larger globules or inside degenerating cells, and (*b*) that this fat is not derived from the chyle-vessels or thoracic duct, is the form of milky ascites called by Quincke fatty ascites.

Since fat is the common feature of them both considerable confusion has arisen ; and it is often difficult to be certain from the recorded cases with which variety we are concerned. Some writers have indeed described a mixed group of cases, in which the ascitic fluid possesses the characters of both categories. In 68 cases, collected by Bargebuhr, 48 appeared to be true chylous and 20 fatty ascites ; and Dr. Batty Shaw collected 68 cases of true chylous and 27 of chyliform ascites. They may both arise under the same conditions—that is, in chronic peritonitis and intra-abdominal malignant disease. In spite of the collected cases just quoted I believe that under these conditions the chyliform ascites is the more frequent. As, however, it may be difficult to assign every case to one or other category, and inasmuch as no account of true chylous ascites would be complete without a reference to this closely allied form, a brief description of chyliform ascites is given here, although it is not due to disease of the lymphatic vessels.

Resembling true chylous ascitic fluid to the naked eye, it differs from it microscopically in the size of the oil-globules which are large, and not finely divided as in the emulsion seen in true chylous ascites. The oil-globules may be found enclosed in cells in which they are first formed, and from which they are discharged later, as the result of further degenerative and destructive processes. The opalescence, however, is not always due to suspended fat, for in some instances it persists after all the fat has been removed, and is then due to the same factor that causes the opalescence of the fluid in milky non-fatty ascites.

In cases of multiple intra-abdominal growth the chyliform ascites may be due to fatty degeneration of the constituent cells of the growth, which are freely discharged into the peritoneal cavity, or to degenerative changes in leucocytes. Corselli and Frisco suggest that in malignant disease of the peritoneum toxic bodies are produced by the growth, which induce fatty degeneration in the cells in the ascitic fluid, and so lead to fatty ascites. A similar hypothesis would explain the fatty ascites sometimes associated with tuberculous peritonitis, wherein the leucocytes in the ascitic effusion under the influence of the tuberculous toxin undergo necrosis and caseation.

In cases of chronic peritonitis—and here it may be mentioned that Letulle and others have insisted on the causal relation between chronic peritonitis and chyliform ascites—the fatty material may be derived from degenerative processes occurring either in leucocytes and fibrin, or in proliferated endothelial cells desquamated from the peritoneum. Guéneau de Mussey had previously expressed much the same opinion

when he said that milky pleural effusion is the result of slow modifications occurring in previously formed collections of pus; the leucocytes disappearing and disintegrating into granular and fatty debris.   In oily, fatty, or chyliform effusion there is no manifest obstruction in the lymphatic or chyliferous vessels.   The distinction between the truly chylous and the fatty or chyliform ascites is of little practical importance.   When after several tappings the originally clear fluid becomes milky, the change is due to degenerative changes in the suspended cells; such cases of chyliform ascites may occur in the course of heart disease. It has been suggested that a lipaemic ascites may depend on pancreatic disease (Gaultier).

Some cases of chyliform ascites may be due to a milk diet and permanent lipaemia, such as is present in young animals, and, pathologically, in some cases of diabetics.

Milky Non-Fatty (pseudo-chylous) Ascites.—This form of milky ascites has comparatively recently been distinguished from chylous and chyliform ascites.   The ascitic fluid is milky, and to the naked eye resembles that of the two preceding groups; but neither microscopically nor chemically does it shew any fatty constituents.   It differs from true chylous ascites also in not separating into layers when allowed to stand.   The fluid is alkaline, has no tendency to putrefy, and retains its characters when filtered.   Its opalescence is not due to bacteria, for it may be quite sterile, but to some albuminous body produced by the degenerative changes in cells suspended in an ascitic effusion.   Lion considered this protein body to be allied to casein, and to be one of the glycoproteins.   He refers to six cases of opalescent ascites, from which by analysis Hammarsten obtained muco-albumin.   The milkiness has also been ascribed to lecithin, or some analogous lipoid body, in combination with a protein (pseudo-globulin) (Joachim).   Milky non-fatty ascites may be found in cases of malignant disease of the peritoneum, the milky constituent being derived from the cells of the growth.   But it occurs in the absence of any intra-abdominal neoplasm, and then is possibly due to changes in suspended leucocytes or desquamated endothelial cells of the peritoneum.   It may also occur in association with tubal nephritis (Strauss, Taylor and Fawcett); I have had such cases under my care.

In cases of repeated tapping the later effusions may be milky, although the earlier ones were quite clear.   In such instances the production of the milky ascites resembles that put forward to explain fatty or chyliform ascites, in that it is a degenerative change in cells suspended in the effusion; the difference being that in chyliform ascites fat is produced, whilst in non-fatty milky ascites the product is albuminous.

In what has been said thus far, the production of milky non-fatty ascites has been mainly explained as a local change.   It may, however, be but a manifestation of a lactescent condition of the blood-serum generally. Milky blood-serum may be of two kinds :—(*a*) fatty ; the condition of lipaemia seen in diabetes mellitus, which occasionally occurs in other persons ; this condition, especially when the patient is on a milk diet,

may produce chyliform or fatty ascites : (*b*) non-fatty ; this may be physiological in some persons after a heavy meal ; or it may be pathological, and is then, according to Castaigne, especially associated with acute and subacute epithelial change in the kidney. Turbidity of the liquor sanguinis, due to the presence of some protein body, may account for some cases of milky non-fatty ascites.

The signs, whether of chylous, chyliform, or milky non-fatty ascites, are those of ordinary ascites, from which it cannot be distinguished, though it might be suspected from the presence of some cutaneous lymphangiectasis, until the abdomen is tapped and the milky fluid drawn off. Microscopical and chemical examination will then be required to decide whether it is chylous, fatty, or milky non-fatty ascites.

The onset may be sudden or gradual ; and, after tapping, the fluid, when there is leakage from rupture of the lymphatic trunks, tends to reaccumulate rapidly. In such cases very large amounts may be removed within a few months. When there is transudation of chyle without any breach of continuity of the lymphatic vessels the effusion is less, and it does not reaccumulate in the same way.

Symptoms.—Increasing weakness and debility, from the continued loss of chyle and partial starvation, naturally follow. But well-marked emaciation is prevented, probably in several ways : some absorption of fatty material from the intestine through the portal vein probably occurs normally, and in these circumstances may be increased. The establishment of a collateral circulation, and the possibility that some reabsorption from the peritoneal cavity may take place through the lymphatic vessels which pass up through the anterior mediastinum and open into the thoracic duct above the obstruction, or into the right lymphatic duct, are other factors that must be taken into account. Moreover, there may be signs of chylous effusions into one or both of the pleurae, and oedema of the legs may occur late in the course of the case.

The symptoms of the primary cause responsible for the lymphatic obstruction may, of course, be the most prominent features of the case ; and in chyliform ascites the symptoms may be even less definite, inasmuch as the results of the primary disease are more likely to be in evidence. In chylous ascites the effusion collects rapidly after removal, and always has approximately the same composition.

Diagnosis depends on the removal of the characteristic fluid and its microscopic examination. The existence, in rare instances, of dilated lymphatics or fistulae on the exterior of the body would, of course, suggest that a concomitant ascites is chylous.

An increase in the proportion of fatty materials in the effusion in response to fatty food given by the mouth (Straus's sign) would be corroborative evidence.

The distinction between true chylous, chyliform, and milky non-fatty ascites by means of the constitution of the fluid has been sufficiently discussed already.

Prognosis.—Most, but not all, cases of chylous and chyliform ascites prove fatal. This is not so much because of the character of the effusion, though the impairment of nutrition from interference with the entrance of chyle into the general circulation may be considerable, as from the primary cause, often malignant disease, to which it is due; of 53 cases tabulated by Busey, 33 ended in death. The prognosis would, for this reason, be better in the rare traumatic cases; but rupture of the thoracic duct or receptaculum chyli is formidable on account of the very free escape of chyle.

Treatment chiefly consists in the maintenance of the patient's strength on general principles, and in the mitigation of symptoms as they arise. If dyspnoea or distension occur, tapping should be performed; but this of course increases the drain and loss of chyle, and in true chylous ascites should not be adopted unless necessary. In chyliform and milky non-fatty ascites this objection does not hold.

Chylous and Chyliform Pleural Effusions.—These are comparable to similar conditions in the peritoneal cavity, but are much rarer. Probably for this reason they are usually reported, and thus it results that the recorded cases make up a total which would suggest that chylothorax is only half as frequent as the abdominal condition. Dr. Batty Shaw collected 31 cases of the chylous, and 13 of the chyliform variety, and 10 of milky pleural effusion of doubtful nature; as already mentioned, he collected 68 cases of chylous and 27 of chyliform ascites. But milky pleural effusion is not nearly so common as this.

A chylous pleural effusion may result from obstruction of the thoracic duct in the thoracic part of its course; less often it is due to traumatic rupture, of which Lord has collected 11 examples, to malignant disease of the pleura, and to blocking of the left subclavian vein. It is usually unilateral, but a bilateral chylous pleural effusion may occur. Sometimes, when unilateral, there is a serous effusion on the other side. In Dr. Turney's case the effusion was chylous in the right pleura, and fatty or chyliform in the left. In a majority of the cases chylous ascites was present as well. In a remarkable case in a child aged 9 months paracentesis of the chest was performed 18 times (Jennings and Rich).

Chyliform or fatty pleural effusion is due to much the same causes as chyliform ascites, but especially to chronic pleurisy. The course may be extremely chronic; Nattan-Larrier reports a case which had lasted for more than 6 years. Milky non-fatty pleural effusions of several kinds also occur (*vide also* Vol. V. p. 547).

The existence of chylothorax might be suspected in a case in which chylous ascites is known to exist; but the accurate diagnosis must depend on withdrawal of the fluid and its minute examination.

The signs and symptoms are those of pleurisy with effusion (*vide* Vol. V. p. 538). The prognosis of chylous pleural effusions is usually bad; as just mentioned, chyliform effusion may in exceptional instances persist for years. As to the treatment of true chylous effusions, it is obvious that tapping will favour the loss of chyle from the thoracic duct

or its leaking branches, and is therefore inadvisable, and if it is necessary small amounts only should be withdrawn. Strapping the affected side of the chest to increase the intrapleural tension has been recommended. There is no objection to the free removal of chyliform or fatty pleural effusions.

Chylous and chyliform pericardial effusions are extremely rare. Dr. Batty Shaw's collection contains one example of each of these conditions.

Chylocele or chylous effusion into the tunica vaginalis testis, which occurs in filarial disease, has been met with without any evidence of this affection (Shattock).

Diseases of the other Lymphatic Vessels

Lymphangitis.—By lymphangitis, or angioleucitis, is understood inflammation of the walls of the lymphatic vessels. It is practically always associated with inflammation of the tissues immediately surrounding the vessels — peri-lymphangitis — and with inflammation of the corresponding lymphatic glands.

It is best studied clinically in the superficial forms which attack the skin and subcutaneous tissues ; but it also attacks the deeper structures and the viscera, and there plays a part in the extension of inflammatory and suppurative processes.

Visceral Lymphangitis.—The spread of infection through an organ is necessarily largely by the blood and lymphatic vessels, but the process is not so easily watched in the lymphatics as in the case of the more manifest blood-vessels. It is probable that in some cases of suppurative nephritis, lymphangitis is more important than is generally recognised ; especially when the primary source of infection is at a distance, as in the bladder ; and when there is no manifest continuity of inflammation in the ureter. As Oertel points out, acute lymphangitis and peri-lymphangitis by leading to obliteration of the lymphatics retard resolution in the affected organ, for example the lung or the liver, and may thus lead to fibrosis.

Lymphangitis may be acute, chronic, or recurrent. Acute lymphangitis is the best-recognised form.

Etiology.—Lymphangitis is not a specific disease, and thus differs from erysipelas, which it resembles in many other ways. Lymphangitis may be due to *Staphylococcus pyogenes aureus* and *albus, Streptococcus pyogenes* and the closely-related *Streptococcus erysipelatis, Pneumococci,* and *Gonococci* ; and filarial, tuberculous, and syphilitic forms of lymphangitis occur. It is moreover an accompaniment of many specific diseases, both acute and chronic. The lymphatic vessels are in such intimate communication with the spaces and interstices of the tissues that any poisons or micro-organisms in these situations readily pass into the lymphatics ; in this way lymphangitis may spread from an abscess or a phlebitis. The

lymphatic vessels may, however, be found full of micro-organisms without their walls being inflamed.

It is hardly necessary to enumerate the various methods by which micro-organisms and their products can gain an entry into the lymphatics. The skin may seem intact, and the orifice by which the infection entered may not be visible. On the other hand, it may start from the abraded surface in skin affections; or from pricks, leech-bites, and so forth. Thus, severe lymphangitis may rapidly follow in a person who has made a necropsy on a septic case, who had either no manifest breach of cutaneous surface or only some slight abrasion near the nails. With the advance of putrefaction of the dead body, the risk and danger of infection from it diminishes. In the present day, when the subjects for dissection are carefully injected and preserved, lymphangitis is rarely set up by dissection wounds.

Besides a low or impaired state of nutrition, previous local injury and disease, there are some general factors which dispose to the incidence of lymphangitis ; such as alcoholism, gout, and chronic renal disease. The frequency of various secondary infections in renal disease is well known, and the occurrence of lymphangitis is another example of this.

The lymphatic system being better developed and apparently more susceptible early in life, lymphangitis will, other things being equal, be more likely to occur then.

Trauma without any breach of surface only gives rise to lymphangitis when the resistance of the affected parts has been reduced by some pre-existing disease. This is the case, as has already been mentioned, in elephantiasis and macroglossia. In this connexion the possibility that micro-organisms have remained latent in the tissues until stirred up to fresh activity must be borne in mind.

From observations on fatal cases of burns, made by Bardeen, it appears that inflammatory and degenerative changes in the lymphatic vessels and glands are due to poisonous bodies reaching them by means of the blood-stream. In other words, infection from within must be reckoned with as a cause of lymphangitis.

Acute Lymphangitis.—Acute superficial lymphangitis may come' on a few hours after infection, and rapidly spread with the production of severe constitutional symptoms ; or, on the other hand, it may have a long period of incubation, and not appear until the wound, through which the infective agent presumably was introduced, has healed.

Morbid Anatomy.—The intima of the vessel becomes swollen, and at the same time the endothelial cells proliferate, undergo degenerative changes, and are thrown off ; and the walls become swollen by infiltration with small cells. By extension peri-lymphangitis is set up, the tissues immediately around being occupied with leucocytes. The contents of the vessel first become turbid, and then coagulation leads to thrombosis. When suppuration occurs the clot in the vessel breaks up, and the other changes are intensified, pus being found both inside and outside the vessel, as well as in its walls.

Signs and Symptoms.—Lymphangitis may attack either the lymphatic capillaries (reticular lymphangitis) or the larger trunks; usually these two conditions are combined.

When the smaller vessels are affected there is redness and swelling, and the condition may resemble erythema. If the inflammation be of sufficient intensity, a certain amount of oedema of the skin may be superadded.

The trunks may be affected without the reticular form of lymphangitis being visible; and may then appear as red streaks meandering from the point of infection towards the nearest lymphatic glands. The red lines may be irregular, and not manifest in the whole course of the lymphatic vessels. The redness is broader than the lymphatic trunk, the extension being due to peri-lymphangitis. If there be much infiltration the lines may be palpable as cords under the skin. The glands soon become painful and swollen from adenitis.

The *symptoms* are—(i.) local:—pain in various degrees is usually present in the affected region; it is increased on movement, and is accompanied by some tenderness on pressure: and (ii.) the general constitutional disturbance accompanying the febrile state—malaise, headache, thirst, loss of appetite, and shivering. The symptoms vary, of course, with the degree of the infective process, and in marked cases may be extremely severe.

Results.—In slight cases resolution rapidly occurs; commonly after a week to ten days the inflammation passes away, the thrombus inside the vessel is absorbed, and the exudation in the immediate neighbourhood is removed. But the glands do not subside so soon, and chronic adenitis may be left behind. Desquamation of the skin may occur, as in erysipelas; but the scales are not so large.

In other cases organisation of the thrombus and of the inflammatory products may occur, and give rise to chronic lymphangitis and sclerosis of the lymphatic trunks. This may lead, further, to thickening of the subcutaneous tissues and a condition of elephantiasis.

In severe cases of lymphangitis suppuration and numerous abscesses may occur, which of course require free incision. There is then danger of septicaemia, or even of pyaemia.

The prognosis depends largely on the form of lymphangitis, on the occurrence of suppuration, and on the state of the health, antecedents, and resistance of the patient.

The diagnosis of lymphangitis is generally easy. In its slighter forms it must be distinguished from erythema. From erysipelas, its course in the line of the lymphatics and the absence of a definite margin differentiate it. From superficial phlebitis the absence of the blocked vein will distinguish it.

Treatment.—The original wound, if there be one, should be carefully disinfected and treated antiseptically. The part should be kept at rest and, if it be a limb, should be raised. An application of equal parts of extract of belladonna and glycerin to the area of lymphangitis usually

gives relief. Lead lotion may be applied to reduce the pain. Hot poppy fomentations may also be tried. Antiseptic lotions, and baths containing carbolic acid or other antiseptics, may be employed; dressings of perchloride of mercury, 1 in 1000, have also been recommended. Salzwedel has obtained extremely good results from the application of dressings containing alcohol (95 per cent) to parts attacked with lymphangitis. When suppuration occurs incisions are of course required as soon as practicable.

For the oedema and thickening left behind, massage and the application of pressure by means of bandages may be employed.

The general health must be sustained by fresh air ; and removal from depressing surroundings is desirable. Good diet and tonics, such as iron and quinine, should form part of the treatment.

Chronic lymphangitis may be the sequel of an acute attack or may be chronic from the onset, as a result of infection with organisms of low virulence ; whilst recurrent lymphangitis, like erysipelas, is prone to arise in parts damaged by a previous attack. Thus lymphangitis readily occurs on slight provocation in areas affected with elephantiasis ; and also tends to supervene where the lymphatic vessels are dilated, as in macroglossia and especially in cystic lymphangioma of the neck.

As a result of chronic inflammation the lymphatics become thickened and obstructed, and a condition of elephantiasis with hypertrophy of the connective tissues rather than true oedema results.

The treatment consists in attempts to remove any chronic infection. Mr. Handley's method of lymphangioplasty promises well for the relief of the chronic oedema and elephantiasis produced by obliterative endolymphangitis. Subcutaneous injection of fibrolysin followed by elevation of the bandaged parts has been recommended (Castellani).

Tuberculous lymphangitis forms an essential part of the spread of local tuberculosis, and can be well studied in the peritoneum in the cases of tuberculous enteritis.

In the skin it is comparatively seldom seen, and, curiously enough, is rare in connexion with tuberculous glands in the neck. It is sometimes seen, however, on the extremities, and usually follows local inoculation ; it has then a special tendency to produce local abscesses in the course of the lymphatics. These have been thought to depend on secondary infection with pyogenetic microbes, and to be favoured by the fact that the lymphatic vessels of the limb, before any glands are reached, are larger than elsewhere on the skin, and the flow through them slower. The glands are liable to be affected, and the infection may become generalised. Various forms of tuberculous lymphangitis have been described by the French school.

In the course of *syphilis* the lymphatic vessels leading from the primary sore to the amygdaloid glands may form hard cords.

The affection of the *lymphatics in glanders* is described in the special article on that disease (Vol. II. Part I. p. 201).

Lymphangiectasis or dilatation of lymphatic vessels merges into simple lymphangioma; since the lymphatic vessels are continuous with the interstices of the tissues, it is obviously more difficult than in the case of blood-vessels to say where the process of dilatation ends and that of tumour-formation begins. Lymphangiectasis is probably always due to some form of obstruction, but this may not be obvious; and in such cases the word lymphangioma is sometimes employed. Some transient inflammation of the part may give rise to peri-lymphangitis and to fibrosis, sufficient to compress the efferent lymphatic vessels, without leaving any more tangible impress of its occurrence. Thus dilatation of the lymphatic vessels of the tongue has been known to follow erysipelas of adjacent parts, just as elephantiasis of the limbs is a sequel of the same inflammatory process. Dilatation of the lymphatics in the skin is soon followed by hyperplasia of the connective tissue, leading to thickening; when the condition is diffuse it is spoken of as elephantiasis or pachydermia, when localised as lymphangioma circumscriptum or lymphatic naevus, which will be dealt with in the article on " Tumours of the Skin " (Vol. IX.). Diffuse lymphangiectasis may occur on the skin of the trunk, or even on the mucous membrane of the alimentary canal, and is a notable factor in macroglossia (*vide* p. 840) and macrocheilia (*vide* p. 841), but it is most commonly seen in the lower extremities, as in elephantiasis due to filarial disease which is described elsewhere (Vol. II. Part II. p. 939). Lymphatic oedema of the upper extremities due to the spread of mammary carcinoma may produce such considerable enlargement and disability that in the past the limbs have been amputated. Lymph-scrotum, elephantiasis of the vulva, and varicose groin-glands may be due to causes other than filarial disease, but in other respects the condition is the same, and need not be separately described. Localised areas of lymphangiectasis specially affect the face and neck, and suggest some slight error of development in connexion with the closure of the fissures present in these areas in fetal life (Adami). These formations may break down and give rise to a discharge of lymph (lymphorrhoea).

Dilatation of the lymphatic vessels of the lung is sometimes due to pressure at the root of the lung; and in such cases the branching and dilated lymphatics are well seen under the visceral pleura. Two remarkable cases of numerous cysts of various sizes, and with clear contents, in the great omentum have been described (Cripps, Berry). In both instances there were a few peritoneal adhesions, and the possibility of their being dilated lymphatics due to obstruction was raised. Small lymphatic varicosities may occur on the peritoneum ; an exaggeration of this condition may give rise to mesenteric cysts (*vide* Vol. III. p. 992). In Dr. R. Crawfurd's case of hepatoptosis in which jaundice was due to torsion of the bile-duct, the intrahepatic lymphatic vessels were much dilated. In a case of diabetic lipaemia I have seen the lymphatic vessels around the coeliac axis and in the portal fissure markedly distended with chyle.

These examples serve to illustrate the occurrence of lymphangiectasis; and it may be noted that simple dilatation of the lymph-channels occurs also in cases of cavernous and cystic lymphangioma.

A few cases of lymphangiectasis of the intestine have been observed, and in some no definite obstruction was forthcoming; in others there has been undoubted obstruction of the thoracic duct with chylous ascites. The symptoms of lymphangiectasis of the intestine, so far as they are recognised, are vomiting and exhausting diarrhoea.

Lymphangioma.—The tumours included in this category are analogous to haemangiomas, but are composed of lymphatic vessels or spaces. According to their structure and formation several varieties are described ; but, as will be seen, this is a convenient rather than a rigid division of these tumours, for two or more of the different forms may coexist in the same growth. The three forms of lymphangioma are : (1) simple, (2) cavernous, and (3) cystic. Prof. Adami, however, regards all these as telangiectatic or spurious lymphangiomas, and refers to a few examples of lymphangioma proper, a growth characterised by notable proliferation of the lymphatic endothelium.

(1) *Simple lymphangioma* is, as has already been shewn, extremely closely related to lymphangiectasis ; the distinction between them is that lymphangiectasis connotes some obvious obstruction, whereas lymphangioma designates the condition when from the absence of any cause it appears to be a neoplasm. The two conditions are so much alike that what has been said about localised lymphangiectasis may be taken to apply to simple lymphangioma.

(2) *Cavernous Lymphangioma.*—This is analogous to cavernous angioma, and is due to the formation of new lymphatic vessels, which in the first instance arise as solid cords formed of endothelial cells proliferating from the inside of lymphatic vessels. These cords unite with others, and then become hollowed out so as to form lymph-channels,—homoplastic formation. It is therefore a further stage of a simple lymphangioma, and may be compared with the transformation of a simple into a cavernous angioma.

The interstitial tissue between the lymph-spaces, as the result of the constant pressure to which it is subjected by the growth, usually undergoes atrophy and tends to disappear to a greater or less extent. It may, however, contain a certain amount of fat in addition to fibrous and areolar tissue, and shew connective-tissue cells, leucocytic infiltration, and blood-vessels. Mr. Stiles regards the presence of smooth muscular fibres in the walls of the lymph-spaces as a means of distinguishing cavernous lymphangioma from lymphangiectasis. The contents of the spaces are clear and resemble lymph ; when they occur in the abdomen or in its immediate neighbourhood they may contain chyle (chylangioma).

Another (heteroplastic) mode of origin of lymphangioma has been described by Wegner, in which the new formation of lymphatic channels takes place independently of pre-existing vessels. Granulation-tissue first develops and then lymphatic spaces are formed inside it. This

mode of origin, though it has been generally accepted, is difficult to prove by histological examination.

Cavernous lymphangioma, or lymphatic naevus, is often associated with lymphangiectasis or with cystic lymphangioma ; these combinations may be seen in macroglossia and congenital serous cysts of the neck.

The distinction between cavernous and cystic lymphangioma is rather one of degree than of kind.

(3) *Cystic lymphangioma* may be met with in various parts of the body. Usually these growths are subcutaneous; but they may be deeply situated, for example, on the peritoneum or among the abdominal viscera.   In the neck, ·where they are frequently found, they are beneath the deep cervical fascia, and may have deep connexions and travel along the inter-muscular processes of cervical fascia.   They may occur on the limbs, giving rise to macromelia ; on the trunk, in the neighbourhood of the sacrum ; on the tongue, and, more rarely, on the face.   Their structure is on the same lines as that of cavernous lymphangioma ; but the spaces are larger and form cysts of varying sizes, which, except in their endothelial lining, no longer resemble lymphatic vessels.

The cysts may be separate, or they may communicate with each other; and, by destruction of the intervening walls, a multilocular cyst may be transformed into a unilocular cyst.   Except for the projection of intra-cystic buds the interior of the cysts is smooth ; though when inflamma-tion has supervened their lining may become granular and rough.

The fluid in the cysts is clear, alkaline in reaction, and contains albumin, and salts, chiefly $NaCl$; but, when the cysts have been in-flamed, it may be mixed with blood in various stages of retrogression, and contain cholesterin or pus.   The contents of the cysts may present widely different characters in parts of the same tumour.

The tissue between the cysts may be of various kinds—fibrous tissue ; fat ; sarcomatous tissue ; blood-vessels, sometimes numerous ; smooth muscular tissue ; elastic fibres ; and nerves.   The presence of blood-vessels, which may project in the form of buds into the cysts, has in the past given rise to the opinion that these congenital serous cysts are derived, by obstructive and other changes, from an ordinary haeman-gioma and not primarily from lymphatic vessels.   It is true that cystic formations containing lymph are sometimes found in the middle of erectile tumours ; but this can be explained by supposing that from the first such tumours are composed both of lymphatics and of blood-vessels. This surmise of an haemangiomatous origin appears less probable than the alternative that these congenital serous cysts are cystic lymphangiomas. Although they cannot be absolutely proved to be lymphatic in origin, the facts (*a*) that they are often associated and sometimes anatomically connected with other congenital defects of the lymphatic system, such as macroglossia and macrocheilia, and (*b*), that they are in communica-tion with the lymphatic trunks, are in favour of the view that these congenital serous cysts are lymphangiomatous.

The number of blood-vessels in a cystic lymphangioma may increase

in number, so that the tumour might eventually be regarded as a combination of haemangioma and of lymphangioma. In other cases the interstitial fat increases in amount, so that the tumour may be said to undergo transformation into a lipoma, and so to be cured. When aspirated, blood is often poured out; and Mr. D'Arcy Power considers that the fibrous growths, sometimes seen after numerous tappings, may be due to changes in connexion with this extravasation.

Like other kinds of lymphangioma, the cystic form is extremely prone to recurrent attacks of inflammation; this fact must be borne in mind in connexion with their operative treatment. Inflammation may follow mere aspiration; and though this may, and in rare cases does, lead to rapid and almost spontaneous cure, it may be the cause of severe and fatal suppuration. Cystic lymphangiomas may remain stationary, and in some instances have been known to undergo spontaneous involution; on the other hand they may grow rapidly. In most cases they are congenital in origin, by some authors they are said always to be so. In many instances, as has been already pointed out, a cystic lymphangioma is combined with the other two forms, simple and cavernous lymphangioma. The more dangerous situations for cystic lymphangioma are in the neck and in the sacro-perineal region.

When superficial, lymphangioma may give rise to lymphorrhagia, a condition which, from its inconvenience, may require surgical interference; but otherwise, unless excessive, it is not of any great importance.

Lymphangioma must be diagnosed from naevus, from fatty tumour, and, when occurring on the extremities, from local giant-growth. When lymphangiomas project through the inguinal rings they may be mistaken for hernia.

The treatment of lymphangioma is entirely surgical; pressure may be applied so as to empty the contents into the adjacent lymphatics and lead to consolidation. Puncture is often employed, and may be frequently repeated; electrolysis, again, may be tried, but under aseptic precautions excision is probably the most successful, and certainly the most trustworthy form of treatment.

Macroglossia.—Although congenital enlargement of the tongue may be due to other factors, it depends in the great majority of the cases on dilatation of the lymphatics and coexisting hyperplasia of the surrounding connective-tissue elements. The condition, it is true, may arise in later life as the result of lymphatic obstruction; thus Robin and Leredde refer to dilatation of the lingual lymphatics following erysipelas; but, generally speaking, it is congenital. It may remain latent or comparatively stationary for a time, and then increase in size, gradually or suddenly. Various forms of lymphangioma are met with, and frequently the simple and cavernous varieties are combined in the same specimen; more rarely the cystic form is present as well.

The tongue in the condition of lymphangiectasis, or simple lymphangioma, is covered with minute cysts extending into the papillae.

In other cases there may be a localised cavernous lymphangioma exactly comparable to an ordinary cavernous haemangioma. Usually, in well-marked examples of macroglossia, there is a combination of lymphangiectasis and the cavernous lymphangioma, the later having supervened on the simpler condition.

Inflammation is readily induced in the tongue affected with macroglossia, and the attacks are apt to recur on very slight provocation ; though the individual attacks are usually of but slight intensity, eventually they lead to very considerable enlargement of the organ. The attacks of inflammation induce small-celled infiltration around the lymphatic spaces, and tend to exaggerate the elephantoid condition of the tongue, which may reach huge dimensions, and even touch the sternum. In well-marked cases the substance of the tongue is widely excavated by lymphatic spaces ; in the slighter cases, previously referred to, the surface of the tongue may be affected almost exclusively.

A very rare condition—macroglossia neurofibromatosa—has been described (Abbott and Shattock) in which the enlargement of the tongue depends on plexiform fibroma of a nerve, and is of course quite distinct from lymphangiomatous macroglossia.

Treatment should first be applied in the form of pressure, either directly to the tongue by an elastic band, or, indirectly, by keeping the mouth closed (except during meals) by a bandage or some other appropriate means, whereby the tongue is compressed against the hard palate. If this fail, or if the tongue be so enlarged already that it projects from the mouth, a wedge-shaped piece of it should be removed.

Macrocheilia is a similar condition to macroglossia, with which it may be associated. It attacks the lips, by preference the upper lip ; and has been known to be unilateral, or to occupy both lips.

Mr. Arbuthnot Lane successfully treated a marked case of macrocheilia affecting both lips by electrolysis, and considers this plan preferable to excision of part of the lip.

Congenital Serous Cysts of the Neck.—Hydrocele of the neck. Cystic hygroma of the neck. Cystic lymphangioma of the neck.

These cysts form a distinct group ; they are congenital in origin, and occur in the seat of election of congenital serous cysts, that is in the neck. As mentioned already, under the heading of cystic lymphangioma, it has been suggested that these congenital serous cysts are derived from haemangiomas by a process of obliteration of their vascular connexions and the establishment of a secondary communication between the blood-vessels thus isolated and the lymphatic system. But the connexion of the congenital serous cysts with the lymphatic system, and their occasional association with other kinds of lymphangioma, such as macroglossia and macrocheilia, make it probable that they are lymphangiomas from the first. In the case of congenital serous cysts in the neck, two other sources of origin—the salivary glands and the intercarotid gland—have been suggested, but on no sufficient basis of fact. For, as a rule, these cysts are quite independent of the salivary glands ;

and, even when invaded, the glands appear otherwise healthy : in addition to this the congenital cysts are lined by endothelium, and not by epithelium, as they should be if derived from the salivary glands.

Cystic lymphangioma of the neck must be distinguished from the cysts which arise from the branchial clefts, and which are lined by epithelium derived from the fore-gut. These cysts are lined by columnar or by squamous epithelium, and have usually been spoken of as mucoid or dermoid cysts ; but, as Mr. Shattock has shewn, these terms are not very suitable, and in this instance might with advantage be replaced by the name mucosal cysts. Carcinoma may develop in the remains of the branchial clefts—branchiogenous carcinoma—and a cystic tumour may result. This rare and acquired condition must also be distinguished from the congenital cysts of the neck now under consideration.

The cystic lymphangiomas usually occupy the anterior or lateral surface of the neck, and are rarely seen on the back. They may be unilateral or occur on both sides ; when in the median line they tend to extend into both anterior triangles. They vary much in size ; the unilateral are smaller, but the median may extend from the jaw to the sternum, and then resemble the appearances seen in diffuse lipoma.

They have been divided into multilocular, and simple or unilocular cysts ; but there is no fundamental difference between them, for the former by destruction of the septa of its constituent cysts may tend to become unilocular, and processes or diverticula may pass off from a unilocular cyst. Differences, however, exist from the point of view of their clinical aspects and treatment. The unilocular cysts, according to Lannelongue and Achard, are found almost exclusively anteriorly, and on the left side of the neck ; but the compound or multilocular cysts have no such limitations.

These cysts tend to burrow and extend under the cervical fascia between the muscles of the neck, to which they become adherent, and often travel down along the sheath of the subclavian vessels into the axilla ; they have been known to travel into the mediastinum. This tendency to burrow deeply and to become adherent to muscles, vessels, and nerves is important ; for an attempt at removal may reveal a far wider extension of the cyst than was at first apparent.

The cystic and cavernous forms of lymphangioma are often found united in the cysts of the neck.

The other features of congenital cysts of the neck have been referred to under the previous heading of cystic lymphangioma.

Malignant Growths in Lymphatic Vessels.—In the various forms of carcinoma, except rodent ulcer, early and often extensive infiltration of the lymphatic vessels occurs. The carcinomatous infection travels along the lymphatic vessels in the direction of the stream ; but, as Mr. Handley has shewn, the spread of carcinomatous cells takes place almost as readily against the current as with it. He has shewn that dissemination of carcinoma to parts so distant from the primary growth that embolism through the blood-current has been assumed, is really due to extension

along the lymphatics. In sarcoma, on the other hand, lymphatic infection is the exception ; though it is the rule in primary growths of the testes and of lymphatic structures : in the latter, indeed, the growth may be said to be already inside the lymphatic system ; as a good example of this case sarcoma of the tonsil may be cited.

In exceptional cases sarcoma in other parts of the body may lead to lymphatic infection. Malignant endotheliomas, for example those of the testis, also spread largely by the lymphatics.

Primary malignant endothelioma may arise from the lining membrane of lymphatic vessels—lymphangio-endothelioma, and also from the perivascular sheath of blood-vessels—perithelioma.

<div align="right">H. D. ROLLESTON.</div>

REFERENCES

Diseases of the Thoracic Duct—*Inflammation* : 1. COMEY and M'KIBBEN. *Boston Med. and Surg. Journ.*, 1903, cxlviii. 409.—2. DE FOREST. *New York State Journ. Med.*, 1907, vii. 349.—3. NOCKHER. *De Morbis Ductus Thoracici*, 1831, Bonnae.— 4. WARTHIN. "Diseases of the Lymphatic Vessels," in Osler and M'Crae's *System of Medicine*, 1908, iv. 573—*Tuberculosis:* 5. BENDA. *Lubarsch u. Ostertags Ergebnisse*, 1898, v.—6. LONGCOPE. *Bull. Ayer Clin. Lab.*, Pennsylv. Hosp. Phila., 1906, June, 1.—7. WHITLA. *Brit. Med. Journ.*, 1885, i. 1089.—8. WHIPPLE. *Johns Hopkins Hosp. Bull.*, Balt., 1906, xvii. 270, and 1907, xviii. 382.—*Malignant Disease* : 9. NATTAN-LARRIER. *Clinique médicale de l'Hôtel-Dieu*, Paris, 1906, vi. 131.—10. STEVENS, W. M. "Dissemination of Intra - Abdominal Malignant Disease," *Brit. Med. Journ.*, 1907, i. 306.—11. TROISIER. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1898, 3 s., xv. 455.— *Obstruction* : 12. BERNSTEIN and PRICE. *Brit. Med. Journ.*, 1907, i. 617.—13. MARTIN, S. *Trans. Path. Soc.*, London, 1891, xlii. 93. — 14. NATTAN-LARRIER. *Clinique médicale de l'Hôtel-Dieu*, Paris, 1906, vi. 131.—15. ORMEROD. *Trans. Path. Soc.*, London, 1868, xix. 199.—16. WILKS. *Pathological Anatomy*, London, 1889, 189. —**Chylous, Chyliform, and Milky (non-fatty) Ascites** : 17. BARGEBUHR. *Deutsch. Arch. f. klin. Med.*, 1893, li. 161.—18. BUSEY. *Amer. Journ. Med. Sc.*, Phila., 1889, xcviii. 575.—19. CASTAIGNE. *Arch. gén. de méd.*, Paris, 1897, clxxix. 666.— 20. CAYLEY. *Trans. Path. Soc.*, London, 1866, xvii. 163.—21. CORSELLI e FRISCO. *Pisani*, Palermo, 1897, xviii. 79. — 22. GILLESPIE. *Edin. Med. Journ.*, 1897, n.s. ii. 553.—23. GAULTIER. *Compt. rend. Soc. biol.*, Paris, 1906, lxi. 429.—24. HALLIBURTON. *Textbook of Chemical Physiology and Pathology*, London, 1891.—25. HUTCHISON. *Trans. Path. Soc.*, London, 1902, liii. 274.—26. JOACHIM. *München. med. Wchnschr.*, 1903, l. 1915.—27. LETULLE. *Rev. de méd.*, Paris, 1884, iv. 722. —28. LION. *Arch. de méd. expér. et d'anat. path.*, Paris, 1893, v. 826.—29. MARTIN, S. *Trans. Path. Soc.*, London, 1891, xlii. 93. — 30. MENETRIER et GAUCKLER. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1902, xix. 897. — 31. MERKLIN. *Med. Week*, 1897, 253.—32. OPPOLZER. *Allg. Wien. Med. Ztg.*, 1861, 149. — 33. QUINCKE. *Deutsch. Arch. f. klin. Med.*, Leipzig, 1875, xvi. 121.—34. SHAW, H. BATTY. *Journ. Path. and Bacteriol.*, Edin. and London, 1900, vi. 339. —35. STRAUS. *Arch. de physiol. et de path.*, Paris, 1886, vii. 367.—36. TAYLOR and FAWCETT. *Trans. Clin. Soc.*, London, 1905, xxxviii. 169. — **Chylous and Chyliform Pleural Effusions** : 37. JENNINGS and RICH. *Arch. Pediat.*, 1908, xxv. 195.—38. LORD. *System of Medicine* (Osler and M'Crae), 1908, iii. 854.—39. SHAW, H. B. *Journ. Path. and Bacteriol.*, Edin. and London, 1900, vi. 339.—40. TURNEY. *Trans. Path., Soc.*, London, 1893, xliv. i.—**Lymphangitis** : 41. BARDEEN. *Journ. Exper. Med.*, New York, 1897, ii. 501.—41a. CASTELLANI. *Riv. crit. di clin. med.*, Firenze, 1908, ix. 321. —42. HANDLEY. *Proc. Roy. Soc. Med.*, London (clin. section), 1909, ii. — 43. OERTEL. "Lymphangitis and Peri-lymphangitis of the Liver," *Arch. int. Med.*, Chicago, 1908, i. 385.—**Lymphangiectasis** : 44. ADAMI. *The Principles of Pathology*, Oxford, 1909, i. 754.—45. ALLCHIN and HEBB. *Trans. Path. Soc.*, London, 1895, xlvi. 221.—46. BERRY. *Ibid.*, 1897, xlviii. 105.—47. CRIPPS. *Ibid.*, 1897, xlviii. 85.—48. CRAWFURD. *Lancet*, London, 1897, ii. 1182.—**Lymphangioma** : 49. ABBOTT and SHATTOCK. *Trans. Path. Soc.*, London, 1903, liv. 231.—50. LANE. *Trans. Clin.*

Soc., London, 1893, xxvi. 223.—51. LANNELONGUE et ACHARD. *Traité des kystes congénitaux*, Paris, 1896.—52. POWER. *Brit. Med. Journ.*, 1897, ii. 1633.—53. ROBIN et LEREDDE. *Arch. de méd. expér. et d'anat. path.*, Paris, 1896, viii. 459.—54. SHATTOCK. *Trans. Path. Soc.*, London, 1897, xlviii. 254.—55. STILES. *Edin. Hosp. Rep.*, 1893, i. 520.—56. WEGNER. *Arch. f. klin. Chir.*, 1876, xx. 641.—**Malignant Disease of Lymphatic Vessels** : 57. HANDLEY, W. S. *Lancet*, London, 1905, i. 1048.

H. D. R.

INDEX

END OF VOL. VI

PRESS NOTICES OF
THE SECOND EDITION OF
A SYSTEM OF MEDICINE

VOLUME I

LANCET.—" Considerable alterations have been made in the arrangement of the subjects dealt with in this volume, several new articles have been added, others have been transferred to later volumes, and yet others have been modified or extended, with the result that it is larger than its predecessor by over two hundred pages. . . . As a whole, the work is one of which the profession of medicine in this country may well be proud, and one which, like its predecessor, will be of great value to the investigator and the practitioner who desires to keep himself informed of the advances in medical knowledge and to have the current state of that knowledge conveniently summarised."

NATURE.—"Altogether this volume commands admiration, and if its high standard be maintained, as it doubtless will be, in the succeeding volumes, this *System of Medicine* will form a lasting monument of the high place which British medicine holds at the present time."

DUBLIN JOURNAL OF MEDICAL SCIENCE.—" We have genuine pleasure in congratulating the joint-editors on the first-fruits of their labours. The excellence of this first volume augurs well for the success of the new issue of *A System of Medicine by Many Writers.*"

VOLUME II.—PART I

BRITISH MEDICAL JOURNAL.—" True to their promise, Professor Allbutt and Dr. Rolleston have issued the second volume of the new edition of the *System of Medicine* within the current year. So large, however, is the amount of matter to be dealt with that the substantial book before us represents the first part only of Vol. II. Within its 1087 pages are contained the completion of the section on Infections and Intoxications. The list of contributors will be found, as heretofore, to contain the names of recognised experts in the several subjects comprised under this heading. Some of the articles have only been revised and brought up to date by their authors, others have been entirely rewritten, while a certain number are quite new. The same thoroughness of treatment characterises them all. . . . The new instalment of the *System* will thus be seen to be as full and complete a work of reference as the most expert writers and the most discriminating of editors can make it. . . . A work which presents a trustworthy compendium of all that is really known upon the subjects with which it deals."

SCOTTISH MEDICAL AND SURGICAL JOURNAL.—" Great care seems to have been taken by all the writers in the volume to provide the reader with the very latest available information. It is impossible in the short limits of this review to attempt to do justice to the great value of Dr. Allbutt's *System*. Every article which it contains is worthy of a prolonged consideration. We can only conclude by wishing the second edition the same success which has been so signally attained by the first, and by suggesting that even those who possess the first edition will do well to secure the second, which, as we have endeavoured to shew, is to all intents and purposes a new book."

PRESS NOTICES—*continued.*

VOLUME II.—PART II

BRITISH MEDICAL JOURNAL.—"The list of contributors, thirty-three in number, contains the names of so many eminent authorities that the different articles should represent the standard of our knowledge of them at the present day. . . . We should have liked to mention some of the articles throughout the volume in detail, but space will not permit; it is sufficient to say that the hope of the editors that the volume will serve as a complete work on tropical medicine has been realised; it should, in fact, become the standard of the subject in the English language, both as a book of reference and for study."

PRACTITIONER.—"By all those interested in tropical diseases, it will be welcomed as a most valuable book of reference and study."

EDINBURGH MEDICAL JOURNAL.—"As the subjects are treated in the exhaustive manner characteristic of this system of medicine, with the matter thoroughly up to date, it will be eagerly welcomed by all interested in these branches of medicine. . . . The editors are to be congratulated on the production of an excellent work, which, containing in a compact form such a mass of information with copious references to the literature on the various subjects, should prove invaluable not only to students of tropical medicine, but to practitioners at home and abroad."

VOLUME III

LANCET.—"The present volume well maintains the credit gained by those which have previously appeared, and there is every indication that the new *System* will retain the place secured by the former edition as the most popular standard medical text-book in this country."

BRITISH MEDICAL JOURNAL.—"The volume in every way maintains the high standard which has characterised this publication throughout."

MEDICAL PRESS AND CIRCULAR.—"The third volume more than maintains the reputation of its predecessors."

VOLUME IV.—PART I

LANCET.—"We once more congratulate the editors on the success of their labours. The present volume is a valuable addition to the *System of Medicine*. It contains much new matter, and will serve as a most excellent work of reference."

BRITISH MEDICAL JOURNAL.—"Limits of space prevent our mentioning all the new articles, but we would refer our readers to this volume with great satisfaction as being a perfectly adequate presentment of all the subjects with which it deals."

MACMILLAN AND CO., Ltd., LONDON.