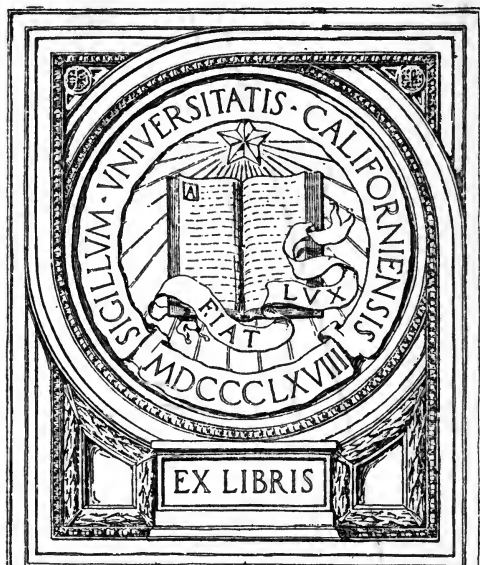


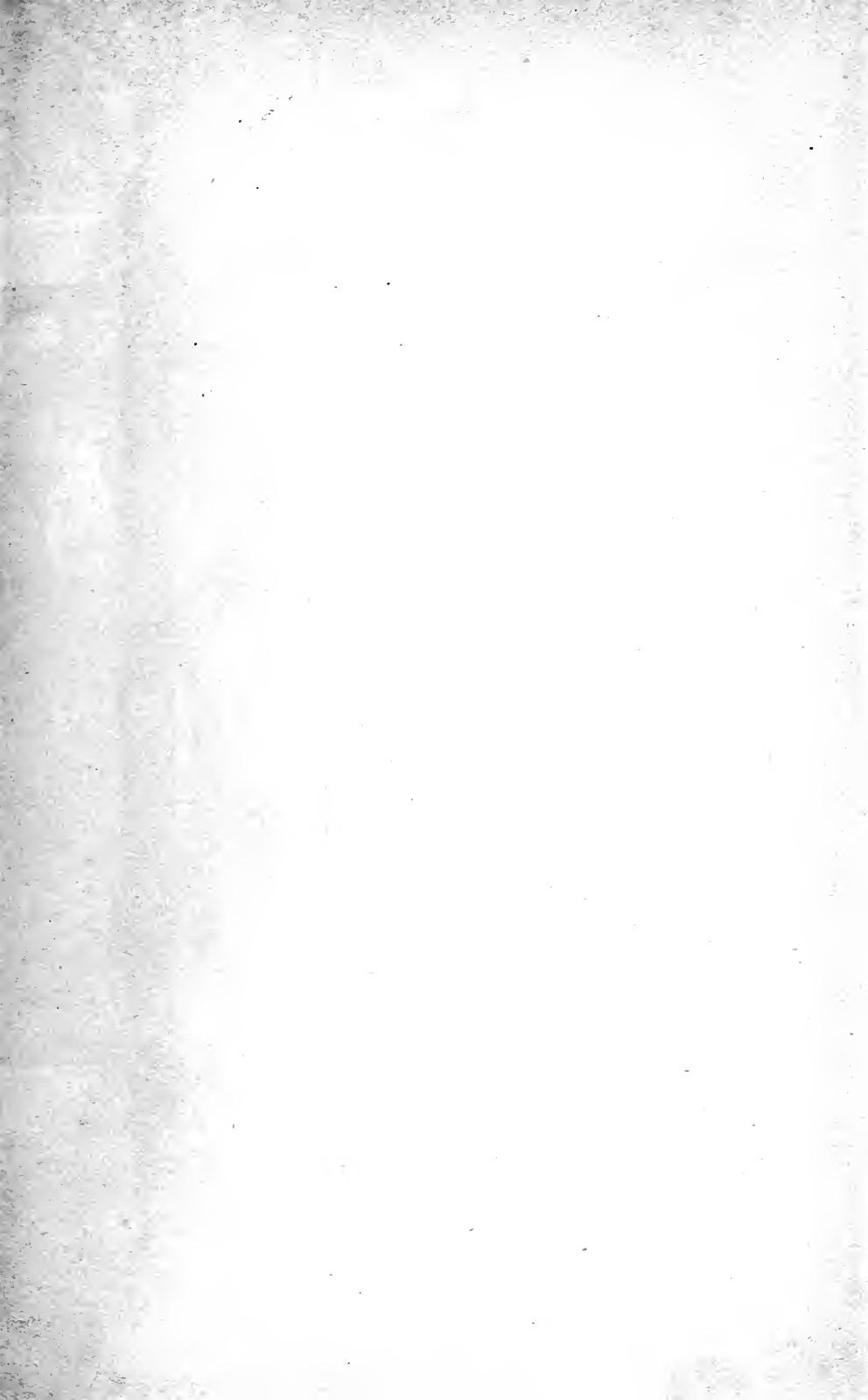


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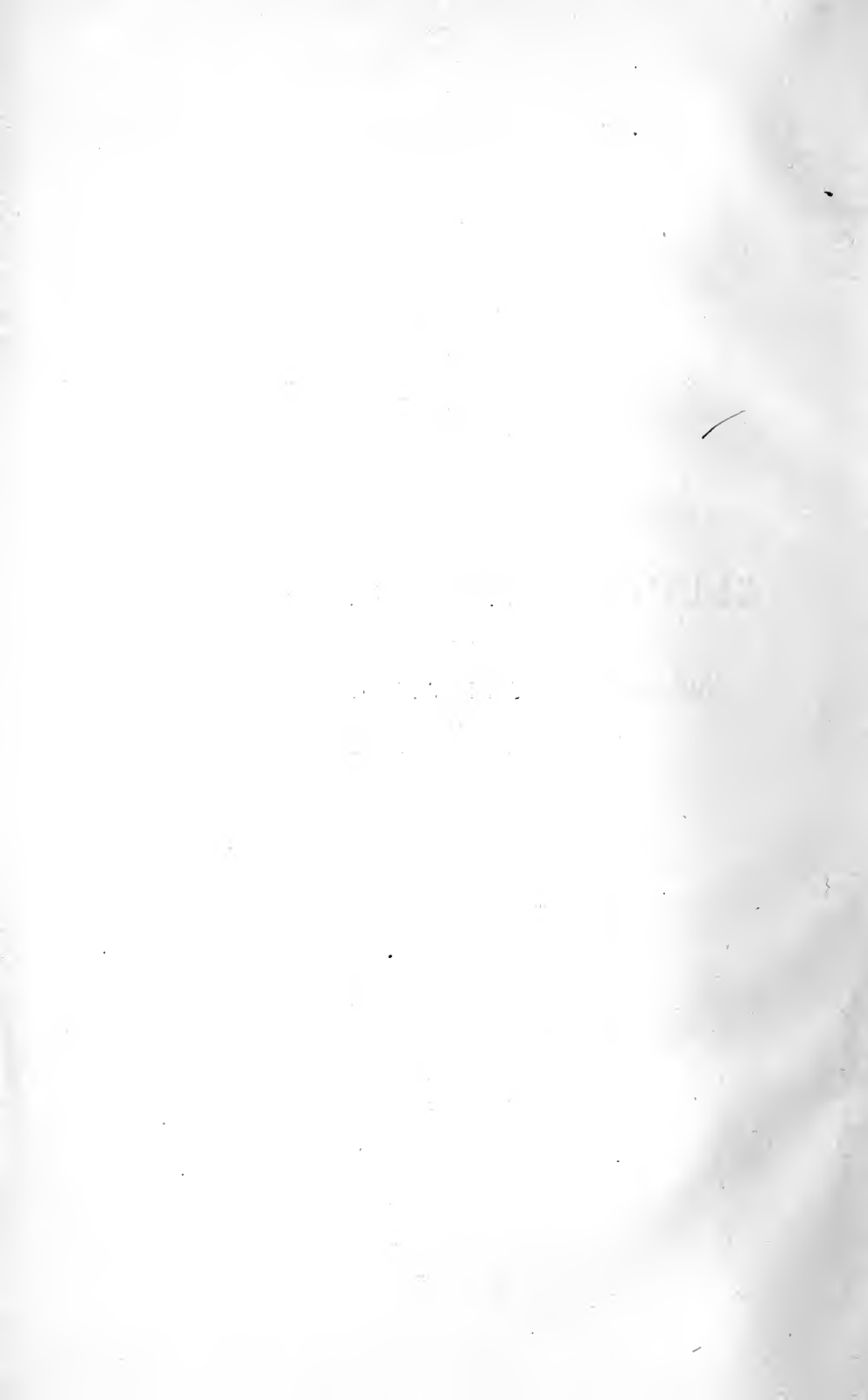


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CLINICAL APPLIED ANATOMY

OR

THE ANATOMY OF MEDICINE AND SURGERY



# CLINICAL APPLIED ANATOMY

OR

THE ANATOMY OF MEDICINE AND SURGERY

BY

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IN SURGERY FOR THE SOCIETY OF APOTHECARIES.

*ILLUSTRATED BY 45 PLATES, OF WHICH 12 ARE COLOURED,  
AND 6 FIGURES IN THE TEXT.*

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## PREFACE.

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IN 1862 there appeared a work which is now classical, "Rest and Pain," by John Hilton. In the preparation of the present volume we have endeavoured to follow, in some degree, the lines suggested by that author, and to indicate the important influence of Anatomy on the incidence and progress of disease, disorder, and injury of the human body.

We are fully conscious that we have but inadequately fulfilled the task we have imposed upon ourselves. There are many omissions; and explanations are not always so complete or convincing as we could desire. We have aimed throughout at keeping the book within reasonable limits, and have avoided the inclusion of highly controversial points. So many excellent treatises on Applied Anatomy have made their appearance of recent years that it might be thought that there was neither room nor necessity for another. The present work, however, differs from these in that it deals with the purely clinical side of the subject, and is written from a practitioner's rather than an anatomist's point of view. Hence all anatomical points which are not essential are omitted. The anatomy of surgical procedures, which is already amply detailed in various manuals of operative surgery, is purposely passed over.

We trust that the book will prove of service to the senior student and to the practitioner, and show that Systematic Anatomy has not yet said its last word in the education of the clinician.

We desire to express our great indebtedness to the various authors who have given us permission to reproduce their diagrams and illustrations. Our requests have in every case been met with the greatest courtesy and consideration. The source of these illustrations is, we believe, acknowledged in each instance. Our thanks are also due to Mr. Lapidge and Mr. C. Douglas for the care they have bestowed upon the preparation of the drawings from which many of the other illustrations have been made.

C. R. B.  
W. McA. E.

LONDON, W.  
*January, 1906.*

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# CLINICAL APPLIED ANATOMY.

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## GENERAL DISEASES AND INJURIES.

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### CHAPTER I.

#### INFLAMMATION.

INFLAMMATION may be aseptic or septic. A very large proportion of inflammatory conditions are septic, that is, due to the invasion of bacteria.

The entrance of bacteria into the tissues may be through the skin or mucous membranes, usually with more or less loss of surface epithelium. When once the blood stream is infected, there is a possibility for remote parts of the body to become inflamed, because the bacteria may be carried to them in the circulation.

Exposed surfaces of skin are liable to injury, and thus an entrance for micro-organisms is obtained. Hence it is that septic inflammation of the head, of the upper part of the neck, and of the hands and fingers is so common.

Inflammation of the cornea or conjunctiva is frequent, and is due to the irritation caused by minute foreign bodies entrapped between the eyelids and the external surface of the eyeball. The rhythmical contraction of the orbicularis palpebrarum is intended to prevent this inflammatory process by the removal of extraneous matter, but it is not always successful.

Other parts which, though usually covered by clothing, are

open to receive blows are apt to become the seat of inflammation, a good example being the shin.

Sometimes the damage done to the tissues by subcutaneous injuries prevents their being able to resist the onslaught of bacteria reaching them in the blood. Hence it is that inflammation, and possibly subsequent suppuration, may occur in the part injured.

Again, portions of skin liable to friction, as, for instance, where the back of the neck is rubbed by the collar, are frequently the site of inflammation, due to bacteria being rubbed from the surface into the hair follicles and glands. Furuncles or boils are thus induced. A "sore heel" from the friction of the boot in walking is another common example of the same kind.

Intermittent pressure, such as happens over the metatarsophalangeal joint of the great toe, will lead first of all to the formation of an adventitious bursa, or bunion, and often subsequently to inflammation of the same.

Parts subject to strains, particularly joints, are peculiarly liable to become inflamed. Of all the joints of the body the knee-joint would seem to be the most susceptible.

The more exposed mucous membranes are those which most frequently suffer from attacks of inflammation. Thus it is that catarrh of the nasal mucous membrane is met with every day, and that tonsillitis is so prevalent. The mucous membrane of the alimentary tract is readily irritated, and consequently inflamed, by material brought to it through ingestion of improper food, and peculiarly so by bacteria.

#### SUPPURATION.

When pus is formed in the tissues it has a tendency to find its way along the lines of least resistance to the skin surface, or into one of the cavities of the body. Therefore it is that the structures within which suppuration occurs have a marked bearing from their anatomical peculiarities and relations upon the actual course taken by the pus in its passage to an exit.



AXILLARY FASCIAE.

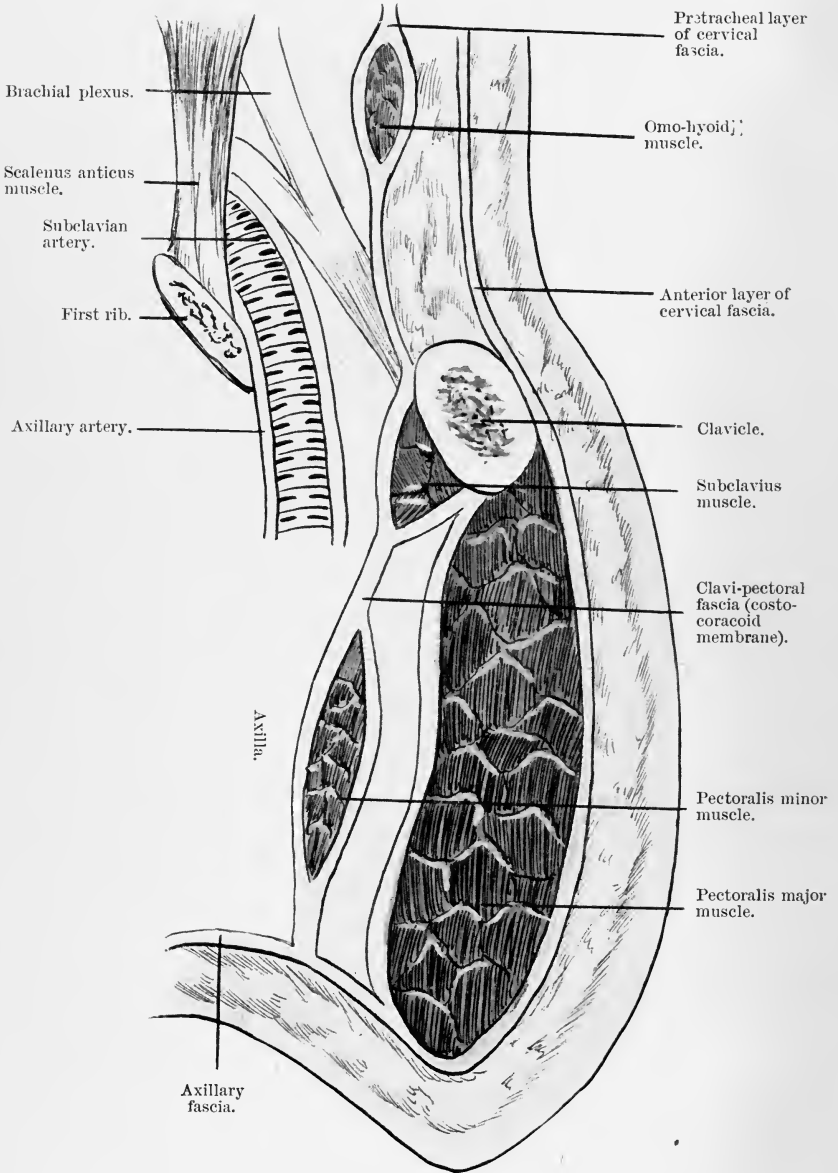


FIG 1.—Diagram to illustrate the arrangement of the cervical fascia in the supra-clavicular region, its connection with the clavi-pectoral fascia, and the connection of the latter with the axillary fascia. (After Edward Taylor.)



Certain abscesses, from their anatomical position, are likely to do grievous harm if allowed to progress unopened by the surgeon. Thus pus in the neighbourhood of joints, which themselves are not inflamed, may track into the articular cavity, and bring about irreparable mischief by setting up suppurative arthritis. Again, a focus of suppuration in the ischio-rectal fossa will most easily find its exit through the bowel wall, causing a troublesome persistent rectal sinus. Pus confined by firm resistant fascia may so press upon important structures as to become a menace to life, as in the case of an abscess beneath the deep fascia of the neck, where it may narrow the trachea and cause urgent dyspnoea. So further, pus within the sheath of a muscle may track widely, and may present at a point very remote from its original starting point, and sinuses extremely difficult to eradicate may be the consequence. Puriform fluid formed deep to the periosteum of a long bone tends rapidly to strip the membrane from the outer surface of the shaft, leading to necrosis.

The track of pus in certain regions is dealt with on other pages, but its particular course in some may be alluded to in this chapter.

**Axillary Abscess.**—Superficial axillary abscesses are due to septic inflammation of the large sebaceous glands in the skin. Most commonly the deep ones are dependent upon suppurative lymphadenitis; sometimes they are due to disease of the ribs or of the shoulder-joint.

When sufficient pus has been allowed to collect in the axillary space, its presence will cause the hollow of the axilla to become obliterated, and the concavity of its floor to be even converted into a convexity. Further, the anterior wall of the space formed by the pectoralis major will bulge. The abscess will increase also in an upward direction behind the clavi-pectoral fascia, sometimes passing deep to the clavicle into the posterior triangle of the neck. The pus will be hemmed in at the back of the space, because of the attachment of the serratus magnus to the vertebral border of the scapula. (See Fig. 1.)

In opening an axillary abscess, the line of the incision is usually made vertically along the inner wall, and half-way between the anterior and the posterior folds of the axilla. In this way the chief blood-vessels will be avoided, namely the axillary, lying along the outer wall, the long thoracic, under cover of the anterior fold, and the subscapular, close to the posterior fold. In addition, this incision will give the best drainage when the patient is sitting up, a position which will be frequently assumed after the evacuation of the pus.

Healing may be delayed unless the arm is kept at rest by the side, and sometimes the cicatricial tissue formed may by its contraction afterwards limit the perfect movements of the scapula over the thoracic wall, and even interfere with those of the shoulder-joint itself.

**Inguinal Abscess, or Bubo.**—Suppurative lymphadenitis is the usual cause of this form of abscess. The lymphatic glands lying above Poupart's ligament are frequently infected in gonorrhœa, and an abscess often has to be dealt with. As the glands lie superficial to the aponeurosis of the external oblique muscle, there is not much difficulty for the pus to find its way to the surface; hence burrowing is not common.

An incision to evacuate the pus may be made either obliquely, parallel with Poupart's ligament, or vertically over the summit of the swelling. Two anatomical objections may be urged against the former: every movement of the thigh will tend to draw the lower edge of the incision away from the upper, and thus retard healing, and there will be a tendency for the inferior part of the wound to form a trough or pocket in which pus may collect. To the vertical incision, especially if made freely, neither of these objections can be brought. It is, however, best to make an oblique incision over the whole length of the swelling, and to dissect out the offending glands and the bulk of the surrounding inflamed tissues by a planned operation under a general anæsthetic.

**Psoas Abscess.**—An abscess within the fascial sheath of the psoas muscle is nearly always the outcome of a tuberculous deposit in the bodies of the lower dorsal or the lumbar vertebræ.

The *psoas magnus* muscle arises from the lower border of the body of the last dorsal and the sides of the bodies of the lumbar vertebræ and the corresponding transverse processes. It is covered with the iliac fascia, the part over the *psoas* being thinner than that over the *iliacus*. Above, this fascia becomes attached to the internal arcuate ligament; on the outer side, to the ventral layer of the lumbar fascia; on the inner side and below, it is attached to the margin of the sacrum and the brim of the true pelvis.

Tuberculous material formed in connection with disease of the lower dorsal vertebræ cannot easily pass forwards, owing to the presence of the anterior common ligament, and it cannot readily pass backwards into the spinal canal, owing to the posterior common ligament; hence it is forced to take a lateral course and gravitate downward in the posterior mediastinum until it reaches the lowest confines of this space. Here it will be directed by the last rib and the twelfth dorsal vertebra behind, and the arcuate ligament in front, into the sheath of the *psoas* muscle, this being the line of least resistance.

Tuberculous material formed in connection with the lumbar vertebræ can pass directly outwards into the substance of the *psoas* along the line of its origin from these bones.

Whichever way the caseous material finds its entrance into the sheath of the *psoas*, once there it will tend to invade the whole length of the muscle, and passing behind *Poupart's* ligament, will enter the thigh. Here it will be at first posterior to the femoral vessels, and subsequently internal to them. The matter as a rule passes to the surface either to the outer side of the femoral vessels immediately below *Poupart's* ligament, or to the inner side of the upper part of the thigh, below and internal to the position in which a femoral hernia is seen.

In some cases fluctuation can be readily obtained between the abdominal and thigh portions of the swelling, though commonly the communication is so small that this definite sign is unobtainable. Moreover, the cavity, in addition to fluid, may contain a considerable proportion of solid contents in the form of caseous masses, which greatly tend against the ready production of fluctuation.

In the treatment of these collections of tuberculous material, it is important to recollect the above anatomical bearings. Effective treatment must be thorough, and to be thorough both the intra-abdominal as well as the crural compartments, if they exist, must be attacked. An incision into the thigh portion from the front is a comparatively easy procedure, due attention being paid to the vessels of the groin. A vertical lumbar incision just external to the outer margin of the erector spinæ is necessary to open that portion of the abscess which occupies the sheath of the psoas. This incision should be the first that is made. After division of the skin a dense aponeurosis is met with, and requires division in the whole length of the wound. The actual sheath of the erector spinæ is best left unopened. The lumbar transverse processes are now felt for round the outer margin of the erector muscle, the quadratus lumborum intervening. This also will require vertical division, care being exercised not to wound the lumbar arteries. As soon as the quadratus is cut through the psoas, which slightly overlaps it, is exposed, and the cavity of its sheath, containing the caseous material, can be readily reached. Stretching across the interior of the abscess cavity there can often be felt the lumbar plexus of nerves, which has been dissected out where it lies in the substance of the muscle. Care must be taken not to perforate extra-peritoneal tissue, and still more so the posterior layer of the parietal peritoneum, which covers the anterior surface of the psoas. The actual seat of the tuberculous disease in the centre of the vertebræ may often be reached and dealt with through this incision.

**Retro-pharyngeal Abscess.**—A collection of pus may occur in the loose cellular tissue between the prevertebral layer of deep cervical fascia and the posterior wall of the pharynx.

An *acute* abscess in this region may be induced by punctured wounds of the pharyngeal wall, by septic processes tracking from the surrounding parts, and by acute suppurative lymphadenitis of the retro-pharyngeal gland.

As a rule the suppuration takes place about the level of the mouth, and the swelling caused by the pushing forwards of the

RETRO-PHARYNGEAL ABSCESS.

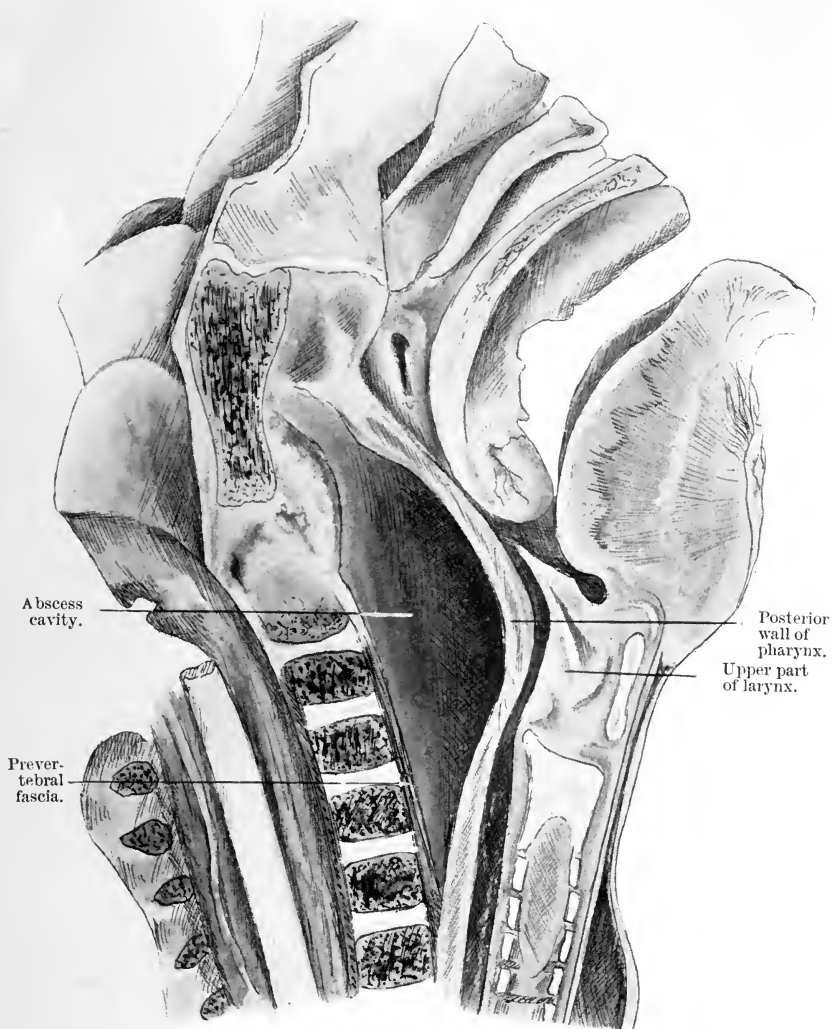


FIG. 2.—A retro-pharyngeal abscess. (St. Bartholomew's Hospital Museum.)

[To face page 8.



posterior wall of the pharynx can be readily seen on oral inspection. The pus here will have a tendency to induce dyspnœa, from its close proximity to the upper opening of the larynx, and also dysphagia, from its obstructing the passage into the lower part of the pharynx. (See Fig. 2.)

The symptoms in these cases are often so urgent as to require prompt surgical intervention in the way of evacuation of the pus, or the performance of tracheotomy, to relieve the dyspnœa.

There is not time for the pus to track any distance before serious symptoms arise; hence its release by an incision made through the mouth is the most speedy and satisfactory. If the incision is made in or near the middle line of the posterior wall of the pharynx, there are no structures of importance that can be exposed to injury. As soon as the pus is liberated the patient should be turned over on to the face, so that the fluid may be less likely to enter the larynx by easily running out of the mouth.

In *chronic* retro-pharyngeal abscess the collection is usually dependent upon tuberculous disease of the cervical vertebræ, or a tuberculous deposit in the retro-pharyngeal lymphatic gland. In these cases the symptoms are, as a rule, much less urgent, and consequently time is given for the tuberculous material to track somewhat extensively. It will thus tend to spread laterally, and may then present in the neck behind or even in front of the sterno-mastoid, or it may pass downwards into the posterior mediastinum. The anterior common ligament of the spine lies in front of the bone abscess, and the latter may bulge on each side of the ligament.

The evacuation of the material may be carried out through the tissues of the neck by an incision made along the posterior border of the sterno-mastoid. The deeper part of the dissection will lie behind the carotid sheath, and must be carried out with the utmost care. By this method of opening these tuberculous collections no chance of contamination through the mouth will occur, and the actual site of disease in the bodies of the cervical vertebræ may even be explored.

**Submandibular Abscess.**—Abscess in the region beneath the mandible is by no means rare, and is generally induced by septic lymphadenitis of the submandibular glands caused by infection from the mouth.

Suppuration may be late, but extensive infiltration and œdema is often early, and may induce dyspncea by involving the loose cellular tissue around the upper opening of the larynx. Incision and free evacuation of the pus is necessary.

### SEPTICÆMIA AND PYÆMIA.

Any focus of micro-organisms is a source from which bacteria or their toxins may pass into the blood or lymph circulation.

When pyogenic cocci enter the blood stream of the systemic circulation it is generally into the capillaries and venous radicles that they pass. If they are not rapidly killed or their toxins neutralised, they are carried to the right side of the heart. Here endocarditis may occasionally occur. The bacteria, however, are usually swept into the lung capillaries, possibly to set up pulmonic and pleural disease. If they traverse these capillaries, which are large and allow ready passage, the left side of the heart is next invaded, and again endocarditis may supervene.

Passing into the arterial system from the left ventricle, the bacteria may travel to any part of the system, and may lodge in any capillaries, but particularly those of the kidneys and the synovial membranes of joints, in the former because the renal organs are excretory, in the latter because there is always some liability to slight degrees of trauma.

If at the seat of infection thrombosis of small venules occurs, there is a probability of infected emboli being detached and carried in the blood to distant parts. These from their nature are more apt to plug capillaries either of the pulmonary or systemic circulation than are bacteria alone.

When pyogenic bacteria enter the radicles of the portal circulation, they are carried direct to the capillaries of the liver, there to excite portal pyæmia or pylephlebitis. If they are not caught



in these capillaries, they pass on through the hepatic veins to the right side of the heart.

When pyogenic bacteria enter the lymph stream, they are carried to the lymph glands, there to be filtered off and killed in a large proportion of cases, with or without evident inflammation of the glands and their surroundings. If the cocci escape the meshes of the filter, they may pass by the efferent vessels to the larger lymphatic channels, and so on into the general venous circulation at the entrance of the thoracic duct into the junction of the left subclavian and left internal jugular veins, or that of the right lymphatic duct into the junction of the right subclavian and the right internal jugular veins.

Although pyæmia may ensue from infection in any region of the body, it is perhaps peculiarly prone to follow infection in certain definite spots. Thus in the head, the middle ear is a common seat of origin. Further the skin of the cheek and the red surface of the lip are dangerous positions should inoculation occur. The facial vein communicates by its radicles with those of the ophthalmic vein, particularly about the inner canthus, and through it with the cavernous sinus, and further the deep facial vein has connections with the pterygoid plexus, which again communicates with the same intra-cranial sinus by small venous channels traversing the foramen ovale and foramen lacerum medius. Moreover, the facial vein tends to remain patent when incised, and has no valves, so that septic material may easily pass from it to the internal jugular.

The uterine veins are a frequent source of septic emboli. So also are the veins of the periosteum of bone, and particularly those bones which are much exposed to injury, such as the tibia.

### WOUND INFECTIONS.

**Cellulitis and Erysipelas.**—These two acute inflammatory conditions are probably both of them dependent upon one and the same specific virus—a streptococcus, the species of which may slightly differ in the two infections, so as to cause some modification of the signs and symptoms.

In cellulitis the infection seems to originate and to spread chiefly in the subcutaneous tissues; in erysipelas the infection occurs and spreads chiefly in the skin. In cellulitis the skin becomes rapidly cut off from its blood supply, so that it has a tendency to slough, while at the same time death of the subcutaneous tissues is the rule; in erysipelas, even if the inflammation is sufficiently severe to induce the formation of bullæ, no loss of tissue occurs except of the most superficial layers of epithelium. In cellulitis the streptococcus travels by means of both lymphatic and venous channels, and red lines of inflamed lymphatic vessels extending to the first chain of glands are not infrequent; in erysipelas the streptococcus wanders in the fine cutaneous lymphatic capillaries, and there is no lymphangitis, although always lymphadenitis of the first chain of glands.

**Tetanus.**—Tetanus is due to the entrance of tetanus bacilli into the tissues and the action of their toxins upon the central nervous system. Owing to the anaërobic nature of the organism, the bacilli are unable to grow upon small and superficial wounds; hence punctured wounds which lodge bacteria deep in the tissues place them in a soil well-fitted for their vital activity. Since garden mould, road refuse, and similar materials form the chief habitat of the bacilli, punctured wounds of the hands and feet from sharp pointed metallic bodies and lacerated wounds of the head, caused chiefly by falls from vehicles, are those which are not infrequently followed by the development of tetanus.

It is perhaps needless to say that wounds in the web between the thumb and the index finger are not more liable to give origin to tetanus than are wounds elsewhere, except from the fact that the hands so often come into contact with the material containing the specific virus. The bacilli remain localised at the point of infection; it is very rare indeed for them to wander into the blood stream and be disseminated by this means, their anaërobic nature being again an obstacle to their migration.

There is little doubt that the *toxin* gains access to the central nervous system by way of the trunks of the peripheral nerves,

probably by means of the protoplasm of the axis cylinders. Experimentally the incubation period, other things being equal, is directly proportional to the length of these nerves.

When large doses of toxin are produced, the poison may be also carried to the central nervous system by the blood stream.

A very characteristic symptom in tetanus is the painful spasmodic contraction of the diaphragm, which gives rise to severe epigastric pain radiating to the back.

The antitoxin of tetanus may be administered by injection into the theca of the spinal cord, by subdural injection, or by injection into the brain itself, these methods affording the most direct route to the central nervous system in which the toxin becomes locked up.

A peculiar form of the disease known as cephalic tetanus occurs when the focus of infection is in the facial nerve area. In this form the facial nerve becomes paralysed, possibly from compression of the swollen trunk by its bony canal.

**Anthrax.**—Anthrax is a specific disease dependent upon inoculation with the bacillus anthracis, usually derived from the bodies or skins of infected animals. It is possible that inhalation of infected dust or ingestion of infected foods may give rise to the disease. Wool-sorters, butchers, farmers, veterinary surgeons, tanners, and pathologists are all liable to have abrasions of the fingers, hands, forearms, or face infected with the characteristic bacillus; hence it comes about that the malignant pustule, the evidence of the site of inoculation, most commonly occurs on these parts. The bacteria are rapidly carried over the whole body by the blood stream.

**Glanders.**—Almost always glanders in the human subject arises by infection from diseased animals, most frequently horses, by the virus being blown from the nostrils of the affected animal into the eyes, nose, or mouth of the horsekeeper, ostler, farrier, veterinary surgeon, or slaughterer. The disease is spread from the primarily infected mucous membrane by the lymphatics, and lymphangitis and lymphadenitis are common occurrences. The vascular system also transmits the virus,

and so secondary foci, resembling in the chronic form of the disease those of syphilis and tubercle, may be found in the internal organs, muscles, and subcutaneous tissues.

**Actinomycosis.**—Actinomycosis may occur in the human subject; it is an infection by the actinomyces, or ray fungus. The fungus most commonly gains entrance through the mucous membrane of the alimentary tract, particularly of the mouth and the appendix vermiformis, from the chewing and ingestion of infected grain.

In the human subject the disease is not infrequently seen in the cheek or subcutaneous tissue of the neck. When the infection occurs lower in the alimentary tract it seldom shows itself until the liver or spleen becomes involved, when these organs frequently become attached to the surrounding tissues by inflammation, and the characteristic actinomycotic pus is discharged externally. The fungus will then have a great tendency to burrow in the superficial fascia and to give rise to a considerable number of tortuous sinuses.

Infection may also occur through the respiratory tract, from which it spreads extensively, not only in the lung tissue, but over the pleura and into the neighbouring bones, and again a large number of sinuses opening in all directions is likely to be seen.

## CHAPTER II.

### TUBERCULOSIS AND SYPHILIS.

**Local Tuberculosis.**—The site of primary invasion in tuberculosis is largely determined by anatomical factors. The uninjured epidermis affords an efficient protection, so when infection through the *skin* does occur it is in parts exposed to free contact and to mechanical injury, such as the margin of the nostril, the lip, and the angle of the eye. Tuberculosis of the skin may follow trivial injuries, such as tattooing, hypodermic injections, or scratches received during post-mortem examinations. Another example of wound infection is the local tuberculosis which sometimes follows ritual circumcision. When tuberculosis attacks the skin it is not always by direct inoculation; the primary focus of infection may be in a mucous membrane close by, as occurs in some cases of facial lupus. The skin may also be infected through the medium of its blood-vessels, but for some reason this is rare, possibly because the comparatively low temperature of the surface does not much favour the growth of the bacillus.

The *digestive tract*, like the skin, is exposed to direct infection, and the parts most likely to suffer are those which are most richly supplied with lymphoid tissue. The tonsils and the lower part of the small intestine are peculiarly vulnerable on this account. The tonsil, with its crypt-like structure, is not only exposed to primary infection, but is often secondarily infected by tuberculous sputum, and the same applies to the small intestine.

The soft palate, which during swallowing and coughing serves to shut off the posterior nares, is liable to primary infection by the passage of tuberculous food and to secondary infection by the forcible impact of phthisical sputum.

The mouth, pharynx, and œsophagus are well protected by

squamous epithelium. Moreover, neither food nor sputum tarry long in these situations. When tuberculosis attacks the tongue the ulcers are usually on the margins, near the tip, positions presumably liable to slight injury.

The stomach and upper part of the small intestine usually escape, the former on account of its motor activity and acid contents and the latter because of the scantiness of its lymphoid tissue and the rapid passage of its contents. The discharge of the bile and pancreatic juice into the second part of the duodenum also conduces to the immunity of the upper part of the small bowel. There are certain positions in which the digestive tract may become locally involved by extensions of the tuberculous process from lymphatic glands. The upper part of the pharynx may be infected from retro-pharyngeal glands which drain the nasal fossæ, and the œsophagus may be invaded by tuberculous glands of the mediastinum.

The *respiratory tract* is likewise exposed to direct infection.

The nose is particularly well guarded against invasion. The vestibule is lined by thick skin as far back as the *limen nasi*, and, moreover, is armed with thick recurved vibrissæ. The Schneiderian membrane is thick, erectile, endowed with very high reflex excitability, and freely provided with a mucoid secretion inimical to bacterial growth. The ciliated epithelium of the respiratory portion of the nose is an additional protection. Notwithstanding all this, tuberculosis does at times invade the nasal cavities. The infection may be introduced on the finger, and slight injury at the same time inflicted; tuberculous sputum may lodge in the nasal cavity as the result of imperfect cough, or invasion may occur by continuity of tissue.

The complicated structure of the larynx, its exposure to cough pressure, and the absence of ciliated epithelium on its cords, render it very liable to infection by tuberculous sputum during cough. By virtue of its position it may also become primarily infected, but this is exceptional.

In pulmonary phthisis it is said that the larynx is often involved on the same side as the diseased lung. The accuracy of

this statement is disputed. The unilateral infection is attributed to a weakening of the corresponding half of the larynx due to pressure on the recurrent laryngeal nerve. The nerve is supposed to be involved by the tuberculous process at the lung apex or pressed upon by tuberculous glands in its neighbourhood. The larynx, thus weakened, cannot properly clear itself. The exposed position of the crico-arytenoid joint often leads to its invasion in laryngeal tuberculosis, with consequent fixation of the vocal cord.

The trachea and main bronchi are protected internally by their smoothness, their ciliated epithelium, and their impermeable basement membranes, but may be invaded from without through the lymphatic glands of the tracheo-bronchial group.

The lungs are the commonest site of tuberculous infection. The virus is probably air-borne. The terminal air-passages—*i.e.*, the terminal bronchioles and the alveoli—are forced to rid themselves of inhaled particles by the slow process of lymphatic drainage, whilst similar particles on the upper parts of the respiratory tract are rapidly removed by the upward current of mucus produced by the ciliated epithelium and fail to pass inwards through the basement membranes. In children the bacilli easily traverse the lung and lodge in the bronchial glands, but in adults, in whom absorption is hindered by the greater firmness of the mucous membrane and possibly also by partial choking of the lymphatics, broncho-pneumonic lesions result, commencing in the terminal bronchioles and extending to the alveoli in connection with them.

The anatomical causes of the vulnerability of the apices are much disputed. Imperfect blood and lymph circulation, deficient expansion, and deficient expiratory power have all been alleged as causal factors.

Once established in the lung, the tuberculous focus can spread by various paths. Infection may be carried from the apex of an upper lobe to the apex of a lower or to the opposite lung by aspiration, the infected sputa being inhaled along the short wide bronchus which leads to the apex of the lower lobe, or across the bifurcation of the trachea to the opposite lung. From all

established foci local extension may occur radially by means of lymphatics and also by direct continuity of tissue. During the progress of a tuberculous lesion an incompletely thrombosed artery may be opened, and localised embolic miliary tuberculosis be set up in the part of the lung to which the invaded vessel is distributed.

A distinctive feature of lung tuberculosis in childhood is the direct infection of lung tissue by adjacent tuberculous lymphatic glands. Since glands can be traced into the lungs as far as the fourth subdivision of the bronchi, they are apt to appear as embedded caseous masses, and may be confused with true pulmonary deposits.

It is also possible for the lungs to be invaded through the blood stream as part of a disseminated miliary tuberculosis, and some authorities believe that phthisis is of embolic origin.

The *serous membranes* are frequently the sites of tuberculosis.

Invasion of the pleura is often secondary to that of the lung and the bronchial glands. The intimate relation of the visceral pleura to the lung, the subpleural course of many efferent pulmonary lymphatics, the termination of these lymphatics in the bronchial glands, and the close relation of the glands themselves to the pleura, all facilitate invasion of the latter, which may either occur by direct continuity or possibly by lymphatic backflow. The pleura may also be invaded by the medium of the blood stream, but its vessels are comparatively unimportant. Yet another path for pleural invasion is from the peritoneum through the lymphatics of the diaphragm. Carious ribs or vertebræ may infect the pleura by contact.

Very similar considerations apply to pericardial infection. This sac is adjacent to the lungs and pleuræ, also to the sternum, ribs, and dorsal vertebræ; the bronchial and other mediastinal glands are in close apposition to it. From any of these sources tuberculosis may spread. It is also open to infection from below through the diaphragm and by the blood stream.

The peritoneum may be invaded by means of the blood stream, but, as is the case with the pleuræ, its vessels are small and



unimportant. It is, however, exposed to infection from the digestive and the urinary tracts and from the genital organs. Caseous mesenteric or retroperitoneal glands may involve the peritoneum by continuity or actually burst into the sac. Tuberculous ulceration of the intestine almost invariably gives rise to local peritoneal tuberculosis, and may originate a more widely spreading infection. Tuberculous infiltration of the Fallopian tubes is very favourably situated for direct extension to the peritoneum through their ostia or coverings. Of the male genital organs the seminal vesicles are in close contact with the lower part of the great sac, and the tunica vaginalis of the testis may communicate with the peritoneal cavity through an unobliterated funicular process. A pleural tuberculosis may extend to the abdominal cavity either by means of the lymphatics of the diaphragm or by actual continuity when a defect exists in the posterior part of the diaphragm. Such a defect is not uncommon between the costal and vertebral attachments of the muscle. Diffusion of tuberculosis in the peritoneum is aided by the constant movements of the abdominal walls and the peristaltic action of the intestines.

The *genital system*, like other systems, may be invaded through the blood stream, and the localisation of tuberculosis in the epididymis is explained on this hypothesis as being due to the sluggish blood and lymph circulation in that part of the gland which lies at the junction of the blood supply of the testicle proper and the blood supply of the vas deferens. However, it is believed by some authorities that genital tuberculosis, although usually secondary to pulmonary phthisis, even in these instances is due to an ascending infection along the urethra. The mucous membrane of the prostatic portion is bored like a sieve, and so affords a ready lodgment to bacilli. Excretion of living tubercle bacilli by the kidney may also infect the parts below. Whether the prostatic deposit of tubercle be brought by the blood stream, or ascend the urethra, or descend the ureter, it lies at the junction of the urinary and genital systems, and may serve as a focus of infection for both,

the process spreading on the one hand to the seminal vesicles and along the vas to the epididymis, and on the other to the urinary bladder.

Of the female genital organs, the Fallopian tube most commonly suffers. As is the case with gonorrhœa, the infection may ascend, but the vagina is well protected by its squamous epithelium, and the cervical canal by its cylindrical epithelial coat and its narrowness. Infection may be carried to the uterus, tubes, or ovaries by the blood stream. Intestinal tuberculosis may directly infect the tubes, ovaries, uterus, or bladder when infected coils of gut are in contact with the viscera mentioned.

Tuberculosis of the *urinary system* may originate in the kidney as a blood-borne infection, the bacilli settling in the vascular tufts or in the tubes during the process of excretion. The infection may ascend from the genital system in the male, for the trigone of the bladder is easily invaded from the prostate, and since the mucous membrane of this region is here most fixed, it is less likely to free itself during the processes of distension and evacuation of the bladder. The kidney may be infected from the bladder by an extension along the ureter, or by way of the lymphatics of that tube, or by regurgitation of urine. The kidney is sometimes invaded in genital tuberculosis without the intervention of bladder tuberculosis; in such cases it is suggested that the infection spreads from the epididymis by way of the lymphatics of the vas deferens, thence to the lymphatics of the ureter, and so to the kidney. The bladder is thus excluded from the track of infection. The fact that the bladder in the female is less likely to become tuberculous than the bladder in the male is accounted for by the absence of prostate, which is a common focus of infection.

The *brain* and its *membranes* are usually infected as part of an acute miliary tuberculosis derived from a caseous focus elsewhere in the body. Direct infection from the ear sometimes occurs, and in some cases an infection from the lymphatics outside the skull appears to be possible. The continuity of the lymphatic sheaths of the cranial nerves with the lymphatics outside the skull supplies

a pathway in the latter form of invasion. The sheath of the optic nerve is connected with the lymphatics of the orbit and eye; the sheath of the auditory nerve is in communication with the perilymph spaces of the internal ear. The olfactory nerve sheaths are intimately connected with the lymphatics of the olfactory mucous membrane.

The spinal meninges usually suffer in company with the meninges of the brain, but may be involved alone as the result of the invasion by a tuberculous focus in the body of a vertebra, and in the same way the spinal cord may become locally tuberculous.

Of the *organs of the senses* the ear is open to direct infection along the Eustachian tube or to invasion from the meatus or invasion by means of the blood stream.

The posterior part of the eye usually suffers in a blood-borne tuberculosis, the richly vascular choroid affording a nidus. The affection is then bilateral. Unilateral affection of the anterior parts of the eye points rather to local infection from without. The conjunctiva and lachrymal apparatus may be similarly infected. The preauricular, submandibular, and cervical lymphatic glands are enlarged in these cases of anterior local infection. The vascular iris is liable to suffer in common with the back of the eye in general infection, also may be invaded from the front in local infection. The rarity of local tuberculous infection is due to the efficiency of the lachrymal apparatus and the protection afforded by the squamous epithelium of the conjunctivæ.

Tuberculosis of the *bones and joints* is generally secondary to a focus elsewhere. Infection is conveyed to the bone or joint involved by the lymphatic or vascular systems, usually by the latter. As a rule the primary focus is in the bronchial glands or in the lung. The petrous bone and mastoid may be directly infected by way of the Eustachian tube or the external auditory meatus. The carpal and tarsal joints may, in some instances, be invaded directly from contiguous tendon sheaths. Cancellous bone, from its richer blood supply, is more likely to suffer than bone which is compact; vascular, rapidly growing epiphyses are especially liable to infection, particularly where

their situation exposes them to risk of injury. The numerous articular vessels which supply an epiphysis in active growth render it more vulnerable than the diaphysis with its solitary artery. The vessels which supply an epiphysis also supply the adjacent joint; hence disease of the two is often associated, but the joint may also be invaded by direct extension from the epiphysis. In adults after the period of rapid epiphysial growth has passed the synovial membrane rather than the bone is likely to be the part attacked.

The vascular cancellous tissue of the bodies of the vertebræ, the head of the tibia, the condyles of the femur, the neck of the femur, the lower end of the humerus and of the olecranon, is favourable to a tuberculous deposit. The bones of the tarsus, in which cancellous tissue is abundant, and which are peculiarly exposed to slight strains and injuries during locomotion, are often the site of tuberculosis. Those exposed to the greatest strain are said to be most frequently attacked, the order of liability being os calcis, first metatarsal, astragalus, cuboid, scaphoid, and cuneiform. The proclivity of the inner longitudinal arch is very obvious. The relation of the tarsal bones to the synovial cavities of the foot determines the subsequent direction of the tuberculous process. In this respect disease of the scaphoid bone is serious, since it may extend to both the anterior and posterior segments of the tarsus. When the carpus is attacked the fact that the synovial sac between the bones of the first row and the radius is shut off from the second row of joints is important. The joints of the second row communicate with the carpo-metacarpal joint by a channel between the trapezoid and os magnum.

The cancellous tissue of the flat bones of the skull, of the finger bones, of the acetabulum, and of the glenoid fossa of the scapula affords a good nidus for the tubercle bacillus.

Necrosis is a natural result of inflammation in an unyielding tissue like bone. In very acute tuberculous inflammation of the ends of the long bones a wedge-shaped sequestrum may form, pointing to embolism of an artery as the origin of the process.

The secondary results of bone tuberculosis depend on the anatomical relations of the bone involved. Increased epiphysial growth is sometimes seen; joint tuberculosis is a common sequel. When a diseased bone is in intimate relation with a serous sac the latter may be involved by extension; tuberculous peritonitis, pleurisy, and meningitis at times arise in this way. Disease of the bodies or laminae of the vertebræ may give rise to paraplegia by causing pressure on the spinal cord or by directly invading it. The intimate relation of the psoas muscle to the vertebræ explains the frequency of psoas abscess in tuberculous caries. Destruction of bone gives rise to various deformities of the spine and joints.

*Tendon sheaths* are liable to tuberculous infection, which manifests itself as a chronic teno-synovitis. Primary infection of these structures is probably blood-borne, but the intimate relation of tendon sheaths to certain joints allows direct infection from the joints themselves. Thus the flexor and extensor sheaths may suffer in disease of the carpus, and the sheaths of the peroneal and tibial muscles in disease of the tarsus. Conversely disease of a tendon sheath may infect a joint over which the tendon passes; the tendon sheaths around the wrist are peculiarly liable to invade the wrist-joint in this way.

The *mammary gland* may be invaded by way of the milk ducts, and then the inflammation is primarily parenchymatous or through fissures of the nipple, when interstitial mastitis may be expected. Secondary lymphatic glandular enlargement follows.

The *lymphatic glands* are often the site of localised tuberculosis. The drainage area of the group of glands first involved must be assumed to be the place of primary infection. The glands which most frequently suffer are the tracheo-bronchial group, the cervical chains, and the mesenteric group, corresponding respectively to infections of the lung (or possibly of the trachea and œsophagus), of the nose and throat, and of the small intestine. The primary lesion may be insignificant, and there is good reason for the belief that the bacilli may even penetrate

mucous membranes without producing any lesion at their point of entry.

The bronchial glands are remarkably liable to infection in childhood. In early years the afferent lymphatics are more permeable, and the pulmonary lymphatics in particular have not yet been choked with dust and carbon particles. Moreover, the occurrence of lung inflammations connected with whooping cough, measles, and other infectious diseases to which children are prone, renders the adjacent glands susceptible. By some it is believed that these glands may be directly infected from the trachea or œsophagus.

The invasion of cervical glands is partly explained by the frequency of trivial lesions of the mouth, fauces, and scalp, the presence of enlarged tonsils and carious tooth sockets, and the exposure of these parts to infection by food or air. If a deep gland of the neck is primarily infected, the deeper lymphatics following the great vessels constitute the track of infection; if the primary infection is of a superficial gland, the glands in immediate relation with this are next involved.

Primary involvement of the mesenteric glands naturally suggests food infection.

Tuberculosis of the inguinal glands is rare. When it does occur it is usually the result of tuberculous affection of the external genital organs.

Widely disseminated glandular tuberculosis is sometimes due to infections brought to the glands by the blood stream.

The firm capsules of lymphatic glands for a long time protect the surrounding tissues from invasion; when the capsule is perforated local extension follows.

**Disseminated Miliary Tuberculosis.**—Disseminated tuberculosis results when the blood-vessels or the lymphatic channels are invaded by the bacilli. In the former case the secondary deposits are widespread, in the latter more or less localised. The direct invasion of the large blood-vessels by numerous tubercle bacilli is the usual basis of widespread miliary tuberculosis; for small vessels tend to become thrombosed during invasion, and so

general dissemination may be prevented. An old caseous deposit is generally the starting-point of the blood infection. Tuberculosis of the vessel walls ensues, not only at the site of invasion, but also in other parts of the vascular system by direct settlement of the organisms or by penetration of the vasa vasorum. General dissemination throughout the body soon results. Bacilli which have entered an artery are carried as emboli into the tissues supplied by that vessel only; but bacilli which enter a vein or the thoracic duct may be carried into the general circulation and reach all parts of the body. Unfortunately veins and the thoracic duct are more likely to be perforated than are arteries, since the walls of the latter are thicker. When the walls of the thoracic duct are penetrated the general circulation is soon invaded by way of the great veins into which the contents of the duct are poured. It is noteworthy that the afferent lymphatics of the pleural and peritoneal membranes are not interrupted by protective lymph-glands on their way to the thoracic duct.

There are several localities in the body where veins are especially liable to become infected from tuberculous foci. The pulmonary veins are involved more frequently than any other, the infection spreading from tuberculous glands around the bifurcation of the trachea or from the lung itself. The mesenteric veins are liable to suffer by reason of their intimate relations to the mesenteric glands. The lower part of the vena azygos major is sometimes infected by tuberculous retroperitoneal glands, the jugular veins by cervical glands, and the innominate veins by glands in the superior mediastinum.

Branches of the pulmonary artery may be directly invaded in the lung, and even the main trunks of the aorta and pulmonary artery have been infected from tuberculous mediastinal glands.

When bacilli enter the circulation by way of the systemic veins or thoracic duct, they traverse the right cavities of the heart, and are chiefly deposited in the lungs, the capillaries of which form the first filter they meet; hence the most copious miliary deposit

may be expected in this situation. Others will pass on through the left side of the heart and be generally distributed by the arterial system.

If a pulmonary vein be the place of entrance the infection will sweep on directly to the general arterial system and be distributed to the organs this supplies. Generally the brunt of the infection will be borne by those organs which are most richly vascular, such as the spleen, liver, and kidney. The lung will be invaded along the bronchial arteries, and so to a comparatively slight extent.

A focus of infection in the portal vein area will chiefly infect the liver, and any bacilli which find their way through this barrier will emerge by the hepatic veins and find a resting-place in the lungs. It is well to remember that the liver may be infected through the hepatic artery when bacilli are in the arterial blood stream.

When the endocardium or the aorta are involved the route of infection is by all the systemic arteries, but when a small arterial trunk is invaded, which may occur when tuberculosis is active in such organs as the lungs and kidneys, the micro-organisms will settle as infarcts in the terminal distribution of the vessel involved.

The great serous sacs are but scantily supplied with blood-vessels, so the peritoneum, pleuræ, and pericardium do not show a profuse deposit of tubercles in blood infections. The choroid, although small, is very vascular, and tubercles deposited in it may sometimes be seen with the ophthalmoscope. The actively growing tissues of children, with their free blood supply, afford a good nidus for bacilli when the latter have once entered the blood stream.

In disseminated blood tuberculosis the bacilli are in the vessels, and therefore, despite the copious deposits in the lung mentioned above, they will not be found in the expectoration unless there be an old pulmonary focus, or, as sometimes happens, the disseminated foci are of different ages, and some of the older deposits have gained access to the air-passages. Deposits of different



ages point to successive blood infections. Similar considerations apply to the serous sacs; but few bacilli are found in the effusions they contain when they have been infected by the blood stream. When meningeal symptoms are present, bacilli are sometimes to be found in the arachnoid fluid.

The difficulty in obtaining bacilli from the blood stream in general tuberculosis may be explained by the consideration that the bacilli do not continuously circulate, but are soon entangled and settle down.

Disseminated tuberculosis may also spread by means of lymphatic vessels. The lymphatic stream is sluggish, and the obstructions are numerous, so the miliary tubercles are mostly seen near the original focus and have not the wide distribution which is met with when the blood stream carries the infection. Lymphatic dissemination may be seen when tuberculosis is spreading from foci in the lung. It may also be observed when tuberculosis is invading the lung from a bronchial gland, the spread in this instance being retrogressive. It is possible that the nodular forms of tuberculosis of serous membranes and spleen are the result of lymphatic infection, and that this form of infection may precede and originate a general blood tuberculosis.

## SYPHILIS.

**Syphilitic infection** is favoured by damage to the surface epithelium, for it is probable that the virus of syphilis invades the intra-epithelial lymphatics at the point of inoculation. A surface lesion may be absolutely necessary before the lymphatics of the thick skin covering such parts as the finger or cheek become infected, but in parts where the epithelial covering is more delicate, as, for instance, the conjunctivæ, abrasion may not be a necessary antecedent. Tattooing affords an easy method of inoculation, and the readiness with which a cracked lip or a bite may become infected also bears witness to the vulnerability of the tissues when their epithelial covering has been damaged. During the period of lactation the nipple often

presents small excoriations, and in wet-nurses these may become infected; sometimes both nipples are invaded simultaneously in this manner. A finger chancre is usually found by the side of the nail, a spot very liable to slight laceration. The proclivity to infection of the groove between the prepuce and the corona is probably an example of the same natural tendency.

It appears that the poison is carried from the point of infection by the perivascular lymphatics, and so invades the nearest group of lymphatic glands. These glands undergo a slow and painless enlargement. Indurated lymphatics can sometimes be traced from the chancre towards the glands involved. The glands which first enlarge are those in direct anatomical continuity with the part infected. With a genital chancre the horizontal set of inguinal glands is usually affected first, with a central chancre of the lower lip the supra-hyoid glands, and with a chancre situated laterally the submandibular group. A finger chancre is followed by swelling of the outer group of axillary glands, or, if on one of the inner fingers, the epicondylar gland may first suffer. An eyelid chancre will first infect the preauricular glands. The occurrence of "crossed bubo," where the chancre is on one side of the prepuce, and the bubo in the opposite groin, admits of an anatomical explanation, for although the lymphatics of the skin of the penis and of the prepuce, after following the course of the superficial dorsal vein, pass mainly to the groin of the same side, yet a decussation may occur, and as a rarity one trunk only may be present, so that all the lymphatics converge to the same groin.

The lymphatics of the glans penis run with the deep dorsal vein of the organ within the aponeurosis. Some of them pass into the abdomen through the crural canal, having traversed the row of lymph-glands round the femoral vein, whilst the rest enter by the inguinal canal, and join the same vertical set of lymph-glands at a higher level where it surrounds the external iliac vein.

When syphilis enters on its **secondary stage**, the phenomena become generalised. The symmetrical distribution and wide

extent of the skin lesions point to a blood infection, and the slight general enlargement of the lymphatic glands throughout the body as contrasted with the localised glandular swellings dependent upon the primary inoculation is also to be regarded as an indication of systemic infection.

The occasional manifestations of jaundice, albuminuria, and swelling of the liver and spleen are also evidences of general blood infection, as also are the later affections of the eyes, middle ears, and testicles. In congenital syphilis, large parenchymatous organs like the liver and spleen, which have a free blood and lymph supply, may suffer severely.

The characters of the eruptions in the secondary stage are modified by the position of the lesion. On ordinary skin suppuration rarely occurs. Where two skin surfaces come into contact, and there is warmth, dirt, and moisture, condylomata result; on mucous membranes, which are naturally moist, mucous patches occur. Hence mucous patches may be expected in the mouth or on the fauces, condylomata in the creases below the breasts, in the groins, in the axillæ, between the toes, between the auricle and the mastoid, at the anal margin, rarely at the umbilicus and in other positions.

The tongue, which, from its position and functions, is constantly liable to slight injuries, and is covered by a thick layer of moist epithelium, may become the seat of refractory secondary manifestations. Laryngeal lesions are more protected, and not so liable to ulcerate as are lesions nearer the mouth.

The involvement of epiphyses and joints admits similar anatomical explanations to those advanced in connection with tuberculosis.

It is not surprising that the arterial system, which forms the tract by which general distribution occurs, should suffer severely. The actual evidence of vascular disease is rather late in making its appearance. Endarteritis occurs, and may end in thrombosis, which causes irretrievable damage to important nervous structures. The virus, by affecting the vasa vasorum of the larger vessels, may lay the foundation of aneurysm, or give rise to a

localised ulceration of the interior of the ascending aorta, or cause a widespread arteriosclerosis.

The manifestations of the **tertiary stage** of syphilis differ from those of the secondary stage in their asymmetry. The original symmetrical distribution by the blood stream may be said to have left residual proclivities or possibly infected foci, which in many instances are stirred up to activity by local accidents or peculiarities.

When these local inflammations attack tubular structures the results may be very serious. The posterior surface of the palate may become adherent to the pharyngeal wall at the level of the Eustachian tubes; strictures of the larynx, trachea, bronchi, or œsophagus may be induced; the small intestine, the ileo-cæcal valve, and, more particularly, the rectum, may be similarly affected. Cerebral arteries may be occluded by a like process, and strictures of the common bile duct and of the nasal duct have been met with. The nasal duct, from its proximity to the nasal bones, may become involved as the result of disease of the latter.

The irregular areas of caseation, so characteristic of the gummatous inflammations of the tertiary stage, are the result of vascular obliteration. At the periphery of a gumma, where the blood supply is less interfered with, organised fibrous tissue will be found. A gumma, which by situation is exposed to injury or to the access of septic organisms, is liable to soften and to ulcerate. Gummata in the subcutaneous tissues, in the mouth and in the pharynx may be given as examples of this tendency. In the deeper and more protected viscera such changes are rare. Tertiary visceral syphilis is commonest in the liver and the spleen. The liver suffers more frequently than other organs, possibly because its greater size increases the possibility of infection. It is also liable to slight injury by the overlying ribs. Liver gummata tend to result in depressed cicatrices, and these are often met with on the surface of the organ.

The tendency of subcutaneous gummata to ulcerate has been

mentioned. Gummatous ulcerations are common in parts exposed to injury and pressure, such as the tissues around the lower part of the knee, on the buttocks, and over the sacrum.

With regard to bones, the most vascular and cancellous and those which are exposed to injury from their position are liable to suffer. The proclivity to inflammation of the spongy vascular bones of the nasal cavity, of the palate, of the vertex of the skull, and even of the spinal column, is well known. The tibiæ and the clavicles, which are bones especially exposed to injury, are prone to gummata. It is possible that the attachment of powerful muscles to bone and periosteum may determine the incidence of syphilitic nodes, for example the clavicular origin of the greater pectoral, the attachment of the calf muscles to the lower end of the femur, and the insertion of the masseter into the mandible. The persistency of the bone pains in gummatous periostitis may be explained by the tension of the resisting periosteum, whilst the pains which accompany bone sclerosis are due to increasing pressure on the nerves which lie in the contracting Haversian canals. Syphilitic necrosis is brought about by vascular occlusion, due to pressure and to endarteritis. The nasal deformities of syphilis are explained by the destruction of the supporting bone and cartilage of the septum, and in children also by retarded growth of the inflamed structures.

Bursæ, by virtue of their functions, are exposed to pressure, and those which are most exposed are most likely to show syphilitic manifestations. In persons who kneel much the prepatellar bursæ may become gummatous; in those who sit much the bursæ over the ischial tuberosities may become similarly diseased. Other bursæ likely to suffer are those about the insertions of the muscles on the inner side of the leg below the knee, those over the great trochanter, and the bursæ between the ilio-psoas muscle and the hip-joint.

Syphilis may affect the central nervous system in various ways. Arterial disease, gummatous and sclerosing inflammations of the interstitial tissues, and primary degenerations of

nerve elements all occur. Arterial disease leads to softening of the parts supplied; the arteries most affected are the large trunks at the base of the brain, and it is possible that their relation to the bony base of the skull has some bearing on their liability to suffer. Gummatous inflammation of the meninges will cause various and erratic forms of cranial nerve paralysis.

## CHAPTER III.

### GANGRENE, BURNS AND SCALDS.

**Gangrene** may in general be said to be due to (1) obstruction to veins alone, (2) obstruction to arteries alone, (3) obstruction to both veins and arteries, and (4) obstruction to capillaries.

Gangrene occurs much more commonly in the lower extremity than in the upper, this being due to the fact that the farther removed a part is from the heart, the less *vis a tergo* there is to drive blood to it, and the greater the difficulty of return of blood from it. The least vascular structures, such as fascial and areolar tissue, slough most readily.

*Obstruction to Veins alone* is not often seen in the extremities, but a typical example is the death of a loop of intestine in strangulated hernia. Here the pressure from without, while not sufficient to cut off the entrance of blood through the arteries, is ample to prevent the proper return through the veins: hence the tissue of the bowel becomes more and more congested and deprived of its proper nutrition in consequence of the interference with the circulation through its vessels, and gangrene will inevitably result if the pressure is not removed early enough.

Septic thrombosis of the main venous channel of a limb, as, for instance, of the common femoral or of the subclavian vein, may lead to rapid moist gangrene, but slowly formed aseptic clotting within the vessel may merely tend to passing or perhaps permanent venous congestion.

*Obstruction to Arteries alone* is perhaps most commonly due to emboli which, becoming detached from the heart, block the vessel at certain points. Should the occluded artery be a terminal or end vessel, with no collateral anastomosis, gangrene is almost inevitable. A good example is that of embolic obstruction of

the superior mesenteric artery, as a result of which the blood supply of a portion of the small intestine is suddenly arrested, often leading to symptoms simulating mechanical intestinal obstruction.

Embolism in main arteries with anastomoses most usually occurs at points where the vessels bifurcate: for instance, the common femoral may be blocked at the spot where the deep femoral arises, or the brachial may be occluded where it divides into the radial and ulnar. The embolus may also be arrested by the curvature of an artery opposite the flexure of a joint, as, for instance, in the popliteal trunk behind the knee. The greater the anatomical difficulties in the way of opening up a collateral circulation, the greater is the likelihood of the supervention of gangrene. Main arteries may also be temporarily or permanently obstructed by external pressure, particularly in the case of unreduced fractures and dislocations. This is well seen in the instance of pressure upon the popliteal artery by the lower posteriorly flexed fragment in transverse fracture above the condyles of the femur.

Atheroma of an artery may lead to its obstruction, owing to thrombosis being induced by the roughening of its internal coat. This, however, is unlikely except in the case of the medium sized vessels of the lower limb. Arteritis obliterans may further be the cause of the onset of gangrene in both the lower and the upper extremity.

The line of demarcation in gangrene which has resulted from arterial embolism, or from occluding arterial disease, is determined by the anatomical arrangement of the arterial tree. An embolus, being likely to lodge at a point of bifurcation, will, if the vessel be completely occluded and no collateral circulation be possible, give rise to death of the tissues thus deprived of their blood supply. Therefore the line of demarcation may be expected at the lower limit of the last branches derived from the trunk above the obstruction. For instance, if the popliteal artery be occluded at its point of division, a line of demarcation may form in the upper third of the leg. If an



embolus be small enough to pass into one of the tibial vessels the collateral circulation is usually sufficient to preserve the vitality of the leg. When arterial disease is present the smaller branches are likely to be occluded first, hence in the lower limb gangrene of toes may occur, or the line of demarcation may form just below the ankle joint, or, if the disease be more extensive, both tibial arteries may be occluded and the popliteal still patent owing to its greater calibre. The conditions will then approximate to those present in embolism mentioned above.

Besides the toes and the feet the skin of the middle third of the front of the leg, being exposed to slight injuries, is liable to become gangrenous in old people in whom the circulation is feeble and the arteries are narrowed.

*Obstruction to both Veins and Arteries.*—This is by no means an infrequent cause of death in the periphery. It is perhaps most typically seen in the instance of gangrene following upon ligation of the superficial femoral artery for the cure of a popliteal aneurysm. The aneurysmal sac itself has pressed upon the accompanying vein, and has thus given rise to more or less congestion of the parts below. The blood flow through the main artery is then suddenly arrested, and the *vis a tergo*, which before the ligation of the artery was only barely sufficient to return the blood past the obstruction in the vein, now completely fails to do so. In consequence, the distal portion of the limb becomes greatly congested and nutrition interfered with, and moist gangrene may result. It is possible also for the aneurysmal sac to so press upon the surrounding articular arteries as to interfere very seriously with the anastomotic circulation.

*Obstruction to Capillaries.*—The cessation of the flow of blood in the capillaries is most commonly brought about as a result of certain anatomical dispositions.

The first of these is the distance of the capillaries from the central pump, the heart. The flow of blood through the minute vessels in a weakened state of the force of the heart's systole may become slower and slower till at last stasis ensues, and if this is prolonged, gangrene sets in. Such a condition is seen in certain

instances of so-called senile gangrene, where, however, arterial disease is usually present, and perhaps more typically in that symmetrical peripheral gangrene known as Raynaud's disease, in both cases the smaller arterioles and capillaries are obstructed by clot following stasis. Senile gangrene begins almost always at the extreme periphery of the lower extremity, namely, in the toes. These are the farthest off of any part from the heart, and a slight traumatism, such as the cutting of a corn or the pressure of an ill-fitting boot, determines a lessening of resistance which, with the poor circulation, induces death of the area.

Raynaud's disease is most frequently seen at the end of the upper extremity in the fingers, parts which, although nearer the heart than the toes, are perhaps more frequently exposed from their uncovered condition to the effects of cold. So also are the tips of the ears and the nose, which may likewise suffer in this disease.

The gangrene induced by frost-bite is due to the cessation of circulation in the terminal vessels, and again is seen almost entirely in the extreme peripheral parts, and particularly those subjected to most exposure—the toes, fingers, ears and nose.

The loss of large quantities of blood may so retard the circulation that gangrene may result in the extremities, and for the same reason, when the carotid artery is tied after severe hæmorrhage hemiplegia from anæmia of brain tissue is to be expected.

The second anatomical consideration is that parts exposed to pressure, especially if that pressure be long continued, are extremely liable to necrosis. Inflammatory exudations under firm fasciæ and under the periosteum of bones may give rise to sufficient pressure to induce gangrenous or necrotic processes. It is from pressure that bedsores occur over bony points in patients who have to remain in one position for some length of time. Pressure sores may also be caused by the application of a splint not sufficiently padded, where it comes in contact with bony points. In association with this may be mentioned the not very infrequent occurrence of a sore at the posterior part of the heel

during the splint treatment of fracture of the bones of the leg. In this instance the thin amount of soft tissue covering the heel is compressed between the os calcis and the splint, and therefore dies. The same process may occur on the back of the forearm or wrist from splint pressure.

The spreading gangrene due to *infectious processes* may be classified with that due to capillary obstruction, although the direct action of toxins on the tissues also plays a part in its production. Such gangrene tends to spread by virtue of its cause, and does not show halting places determined by vascular arrangements such as have already been described when dealing with gangrene due to occlusion of arterial trunks.

In states of obstructed or much enfeebled circulation, microbic invasion of tissues may occur and lead to necrotic or gangrenous processes. These processes are common in strangulated gut, and may also be seen in the mucous membrane of the mouth, and as the result of slight injuries of the integument of the extremities in old people.

## BURNS AND SCALDS.

In burns of the third degree where there is partial destruction of the true skin, the nerve endings are exposed, and consequently great pain is induced.

Burns involving the surface of the trunk are more dangerous than those of the limbs, possibly because of the irritation of nerve terminations which are in close connection with important centres.

Parts which are insensitve from lesions of the sensory nerves, or in a patient still unconscious from the administration of an anæsthetic, are very liable to be burnt from the too close application of hot water bottles, and this is peculiarly prone to happen in the lower extremity.

It is doubtful whether ulceration of the first part of the duodenum is at all a frequent complication of extensive burns, even those of the trunk, when the case is kept free from septic

infection. Absorption of toxic products from septic regions after the destruction of skin by heat, and their excretion with the bile into the duodenum, embolism of an artery of the mucous membrane of the duodenum and subsequent action of the gastric secretion on the bloodless part, and over-activity of Brunner's glands after destruction of many of the sebaceous glands of the skin, have all been put forward as explanations of the origin of the ulcer.

It is important to bear in mind that scalds of the mouth and fauces may be followed by œdema of the aryteno-epiglottidean folds surrounding the superior opening of the larynx, causing severe dyspnoea necessitating tracheotomy.

All burns or scalds which have involved the true skin or deeper structures are apt to be followed by contraction, which in certain positions may lead to very marked deformity and disorganisation of the functions of the part. This is particularly so in the flexure of joints, as for instance when the sleeve of a dress has caught on fire, and the skin of the bend of the elbow is destroyed ; the resulting contraction will then effectually prevent proper extension of the elbow. A burn involving the front of the upper part of the neck may induce such contraction as to draw down the chin and the mandible together with the lower lip towards the sternum, and thus seriously interfere with the closure of the mouth and with mastication.

## CHAPTER IV.

### SPECIFIC FEVERS.

#### TYPHOID FEVER.

THE bacilli of typhoid fever usually gain access to the body by way of the digestive tract, and find their first nidus in the aggregations of lymphoid tissue which are situated in the lower part of the small intestine. Since the ileum occupies the lower and right divisions of the belly cavity, it is in the right iliac fossa that gurgling and tenderness are most apt to occur during the course of typhoid fever. The shape of a typhoid ulcer is determined by the distribution of the lymphoid patch in which it arises; hence, when it is situated, as is commonly the case, in an agminate gland (Peyer's patch) the ulcer is elongated in the direction of the long axis of the gut, and situated at that part of the circumference of the bowel which is most remote from the attachment to it of the mesentery. Ulcers originating in the solitary glands of the bowel may have a rounded outline and occur at any part of the bowel circumference. Ulcers of irregular shape may result from the fusion of smaller ulcers or the partial necrosis of an agminate gland. Although for some reason, possibly the stagnation of intestinal contents above the ileo-cæcal valve, ulcers are usually more or less limited to the lower part of the ileum, yet the lymphoid nodules in other parts of the bowel may become involved. Thus ulcers have rarely been discovered in the duodenum, sometimes in the jejunum, more frequently in the appendix vermiformis and the large bowel, the whole extent of which may be involved in exceptional cases.

The great quantity of lymphoid tissue at the point of entrance of the ileum into the colon accounts for the widespread ulceration

often found at the termination of the small bowel, and a similar explanation holds for the extensive involvement of the cæcum in some cases. Owing to the longitudinal axis of the ulcers, stricture rarely results as a sequel of typhoid ulceration.

The fact that the lymphoid aggregations of the bowel have their bases set in large lymph sinuses accounts for the readiness with which the mesenteric glands and retroperitoneal glands become swollen during the progress of the disease.

The lower part of the ileum is the common site of perforation in typhoid fever. The richness of the lymphoid collections, the free mobility, and the peristalsis necessary to overcome the resistance of the ileo-cæcal valve, sufficiently explain the frequency of perforation here. The large bowel has thicker coats, less lymphoid tissue, and is less likely to be ulcerated; consequently perforation is uncommon. Although the accident has been known to occur in the ascending and in the transverse colon, yet it appears to have been found more frequently in the pelvic segment. This portion of the colon has a full mesentery, is constantly varying in position with alterations in distension of the urinary bladder, and is in close proximity to the sphincters guarding the anus. The conditions therefore are similar to those which obtain at the lower part of the ileum.

Hæmorrhage from the bowel in typhoid fever is usually the result of the erosion of small vessels derived from the terminal branches of the superior mesenteric artery. The terminal arteries of the small bowel encircle the gut, reaching the bowel in the mesentery; at first they run for a short distance immediately beneath the serous coat, then they perforate the muscular layers and run in a deeper plane. It is obvious then that ulceration of Peyer's patches will sooner open an arteriole than would ulceration nearer the mesenteric border of the intestine.

The specific organism readily gains access to the blood stream, and thus not only becomes widely distributed in the body, having been found in the gall bladder, bones and cerebral meninges, but also produces special vascular lesions. Typhoid phlebitis with thrombosis is the most important immediate lesion. Thrombosis

is more common in the lower limbs and in the left femoral vein or its popliteal and saphenous tributaries than elsewhere. This proclivity of the left side is partly explained by the fact that the left common iliac vein, which receives the blood from the left lower extremity, is crossed near its termination by the right common iliac artery, and thus the circulation may be impeded. Clot extending along one common iliac vein may ultimately block the veins of both lower limbs.

When arterial thrombosis occurs it is usually in the femoral or popliteal arteries, both of which may be considered liable to slight injury where they cross the joint flexures. From obstruction in smaller vessels peripheral gangrene may occur. Parts liable from their anatomical situation to suffer in this way are the feet, ears, nose, penis, labia, corneæ, etc.

Clots detached from the veins may be carried through the right heart and produce pulmonary embolism, and the same accident may occur when a thrombus in the right heart is detached by some undue exertion and becomes impacted in the pulmonary artery in a coiled-up form.

Diffuse arteriosclerosis may show itself in later life as the result of the vascular infection which occurs in typhoid fever.

The lenticular rose spots are of vascular origin. They probably depend upon capillary infections and paralysis. The bacilli can be obtained from the spots and the eruption is not seen after death.

Typhoid osteitis and periostitis is said to occur by preference in the regions of the epiphyses in young people in whom these parts have an extraordinarily free blood supply. In adults, the tibiæ, femora, sternum and ribs are most frequently attacked because the tibiæ in particular are exposed to injury, and the sternum and ribs are very vascular. The spine is sometimes invaded.

Laryngeal ulceration in typhoid fever is found in those parts of the larynx which are said to be freely supplied with lymphoid tissue. The ulcers are apt to occur at the base of the epiglottis, on the posterior wall of the larynx, on the false cords, between

the arytenoid cartilages and at the posterior extremities of the true vocal cords. The ulceration at the base of the arytenoid cartilages from its position may invade the perichondrium of the cricoid and lead to the exfoliation of this cartilage.

Peritonitis in typhoid fever is an invasion by contiguity. It usually arises in consequence of the perforation of the bowel, but may possibly arise from bowel without actual perforation in some cases. Infarction and abscess of the spleen, suppuration in mesenteric glands, ulceration and perforation of the gall-bladder, and abscesses in other parts such as the ovary, rectus abdominis, the sheath of which is deficient posteriorly in its lower part, and the urinary bladder may all be the cause of peritoneal infection.

From the relation of the ulcerated bowel to the portal tributaries it is easy to see how portal pyæmia and liver abscesses may arise as complications. The cholecystitis of typhoid fever is probably due to blood infection, but it is conceivable that an ascending infection along the bile duct might occur.

Otitis media as a result of typhoid fever is more common in children than in adults. The reasons for this are given below when dealing with scarlet fever. Extension of infection from the mouth along the parotid ducts gives rise to parotitis. Ulcerations of the palate and pharynx are also secondary infections from the mouth.

Certain muscles are apt to rupture in the course of the fever. Muscular degeneration is the basis for this, but no doubt mechanical factors come into play. The muscles which are most likely to give way are the rectus abdominis, the adductors of the thigh, the psoas or the pectorals.

Bed-sores, being due to continued pressure on debilitated tissues, are likely to occur where bony prominences have a scanty tissue covering; hence they may occur over the sacrum, the trochanters, the points of the heels, front and outer side of the knees, elbows, shoulders, malleoli, scapulæ, and the anterior superior spines of the ilia.

The bacilli may be eliminated in large numbers through the kidney, and thus involve the renal pelves, the ureters, and the



urinary bladder. Urethritis and orchitis, which occasionally occur in typhoid fever, are possibly to be explained as direct secondary infection, *viâ* the urethra, and vas deferens, or as descending typhoid infections from the kidney; but the orchitis is possibly a blood-borne infection.

#### SCARLET FEVER.

Although the virus of scarlet fever may obtain access to the body through wounds, burns, or the raw surfaces left as the result of recent childbirth, yet the common portal of entry is the lymphoid tissue of the faucial and naso-pharyngeal tonsils. When the resulting inflammation is considerable, the swollen pharyngeal tonsil tends to block up the posterior nares, and to obstruct nasal breathing. The secretions escaping by the nose appear in the form of an irritating rhinorrhœa, which excoriates the skin of the upper lip. At the same time the swollen faucial tonsils interfere with deglutition and mouth breathing. Intense inflammation may culminate in widespread local destruction of tissue.

Early enlargement of the lymphatic glands in connection with the fauces and nasopharynx may be expected. This primary adenitis is due to infection, conveyed by lymphatics, and appears in the form of tender submandibular swellings. The glandular infection may be of mixed character, since the buccal, nasal and pharyngeal cavities are liable to become centres of secondary infection by reason of their exposure. The virulence of the infection from the throat may be such that a periadenitis, and even diffuse suppuration in the connective tissue planes of the neck may result. When such extension occurs thin-walled veins may be opened by ulceration, but the arteries, protected by their thickness, usually escape. The œdema of the eyelids, which often accompanies the adenitis, is explained by the fact that their lymphatics traverse glands of the affected groups.

The scarlatinal rash is a manifestation of vascular infection, and is due to distension of small blood-vessels in the true skin. The rash is at first minutely papular and discrete. This punctate arrangement does not appear to be determined by the presence

of hair follicles, but is probably due to some anatomical arrangement of the vessels of the skin. The well-known area of circum-oral pallor, bounded laterally by the naso-labial folds and below by the point of the chin, corresponds to a definite thickening of the skin around the mouth, the transition from the thin skin of the cheeks to the thick skin of the circum-oral zone taking place at the naso-labial lines. A more marked thickening of integument is found in the palms and soles, localities where the rash is also wanting. It seems reasonable to believe that the uniform diffuse character of the eruption on the face, in contrast to its punctate appearance elsewhere, has some relation to the more superficial distribution of the free vascular supply to the "flush-area." The arteries of this area are mainly unnamed branches of the posterior aspect of the facial trunk, and lie more superficial than the named facial branches. The scarlatinal rash is most intense in the regions where flexures of the skin naturally occur, such as the axillæ, elbows, wrists, groins and knees. In these situations small linear hæmorrhages may often be seen.

On the outer aspects of the upper arms, and on the outer sides of the thighs and legs, the skin normally presents coarse papules; these are brought into prominence by scarlatinal injection, and do not fade so readily as the rash elsewhere. A certain amount of exudation accompanies the fully developed rash, and it is this exudation which lays the foundation of the subsequent desquamation. The peeling roughly follows the order of appearance of the rash, but occurs earlier where the cuticle is very thin, especially in those parts which from their situation are subject to friction. The tongue being exposed to friction and moisture begins to peel at its tip and edges within twenty-four hours, bringing its vascular papillæ into greater prominence; friction of the night-dress determines early peeling about the lower part of the neck and adjacent parts of the chest; the lobules of the ears and the cheeks coming into contact with the pillow, early show a fine powdering. The thick skin of the palms and soles, and particularly that of the heels, is the last to be shed, and comes off in larger flakes.

Certain anatomical points are worth noting as bearing on the complications of scarlet fever.

The possibility of the buccal, nasal and pharyngeal cavities acting as foci of secondary infection has already been alluded to.

The secondary adenitis involves that group of glands which is in anatomical connection with the fauces and nasopharynx. Swelling of these glands sometimes immediately precedes the onset of acute nephritis, and there appears to be some connection between the two phenomena.

The otitis media is due, like that which occurs in measles, to extension of infection along the Eustachian tube. The remarkable limitation of this complication to childhood is explained by the shortness of the tubes in early years, by their nearer proximity to the tonsil at this age (they open at the level of the hard palate) and to the greater liability of the nasopharynx to be blocked by adenoid tissue. Under these adverse circumstances the ciliæ of the tubes fail to protect them. The parotid lymph glands, which receive lymphatics from the tympanum, may become swollen as the result of otitis media.

In childhood the air spaces of the mastoid are very superficial, and may actually communicate with the surface, so pus may make its way outwards below the periosteum. Pus superficial to the periosteum may be derived from a suppurating mastoid lymph gland.

The broncho-pneumonia of severe scarlet fever is explained by the nearness of the glottic aperture to the faucial inflammation, and the consequent aspiration of infective discharges from the nasopharynx.

The distribution of scarlatinal inflammation in the kidneys is mainly influenced by vascular arrangements. The typical nephritis is chiefly, but not exclusively, glomerular.

The localisation of what is known as the "septic rash" appearing in scarlet fever is largely determined by pressure; it is found on the extensor aspects of the elbows and knees, but is also seen on the cheeks and buttocks.

Irritating discharges from the nares, angles of the mouth and

eyes, and the external auditory meatus, account for the distribution of the eczematous areas found in these localities. The difficulty in cleansing the recesses of the ear, nose, fauces and accessory air sinuses explains the long-continued infectivity of discharges from these places.

#### DIPHTHERIA.

This disease is one which attacks mucous surfaces, and, being due to infection from without, it infects the nose, naso-pharynx, fauces, pharynx, or larynx primarily. The subsequent extension of its membranous manifestations is usually determined by the continuity of parts. Thus, in bad cases membrane may spread through the larynx, along the trachea, bronchi, and bronchioles; the ear may be invaded through the medium of the Eustachian tube, and the conjunctiva by way of the nasal duct. In exceptional cases false membrane may spread down the œsophagus and even be present on the mucous coat of the stomach. The lachrymal secretion and protective muscular mechanisms of the eyelids no doubt largely account for the general immunity of the conjunctiva, whilst the mechanism of deglutition and motor activity of the stomach may be held responsible for the common escape of the œsophageal and gastric mucous membranes.

The mucous surfaces of the vulva, vagina and anus are sometimes, though rarely, the site of primary invasion. The same parts may become involved during the progress of faucial diphtheria, probably by infection conveyed by the fingers. In addition to spread by continuity, the tissues of the body are invaded by the toxin of diphtheria by the medium of the blood-vessels and lymphatics. The characteristic bacilli may be conveyed by lymphatics to the glands, which drain the area primarily involved, or gaining access to the blood-vessels, may become deposited in such viscera as the spleen, liver and kidneys. As a general rule, however, the bacilli remain localised at the point of infection and parts contiguous to this, and it is the toxin alone which circulates. The bacilli which are occasionally found in the lung alveoli have passed along the respiratory passages.

Skin is an effectual barrier to diphtheritic infection, but wounds or raw surfaces may be attacked.

When diphtheria attacks tissues which are naturally lax, considerable œdema may accompany the formation of false membrane. (Edematous swelling may therefore occur in the fauces, soft palate, uvula, and superior opening of larynx. Occasionally destructive ulceration of the same parts is met with.

Diphtheria may prove dangerous either from the mechanical suffocative effects, which depend upon the localisation of the membranous exudation, or from toxæmia, which is dependent upon absorption.

Suffocative symptoms are produced when the larynx, trachea, bronchi or bronchioles are involved. Of these the larynx is the first affected by virtue of its position, and its involvement produces urgent symptoms on account of the ease with which the upper laryngeal aperture is occluded by membrane and œdema. Even after tracheotomy it is often possible to observe the deadly effect of occlusion of the lower air passages.

Toxic symptoms in diphtheria are determined by two factors: first, the extent of the membranous exudation, and, secondly, the facility with which absorption can take place. The comparative harmlessness of membrane limited to the nasal cavities may possibly be due to lack of facilities for absorption. There are abundant indications that free absorption takes place in the regions of the nasopharynx and fauces.

The arrangement of the lymphatics of the air tubes renders it probable that but little lymphatic absorption takes place in the trachea and bronchi, where the basement membrane completely cuts off the deep lymphatics from the surface lining of the tubes.

The local lymphatic glandular swelling in diphtheria has already been alluded to; the inflammation when it spreads outside the glandular capsules may give rise to a brawny infiltration of the connective tissues of the neck. It is stated that the parotid and submandibular salivary glands may become inflamed in diphtheria; when this really occurs it is presumably due to an extension of infection from the mouth along the ducts of the

glands in question. It is well to bear in mind that lymphatic glands are so intimately related to the parotid and submandibular salivary glands that they may almost be said to be embedded in them, and clinically it may be very difficult to distinguish lymphatic glandular from salivary glandular swellings.

The albuminuria and neuritis of diphtheria are probably both the results of toxins, carried by the blood stream.

#### MEASLES.

This highly infectious disease chiefly manifests itself in the respiratory tract and the skin. The appearance of Koplik's spots on the buccal mucous membrane affords the earliest evidence of the infection. The distribution of these spots is in some way influenced by position, for they usually occur on that part of the buccal mucous membrane which is in contact with the teeth, and so are best seen by drawing the cheek away from the molar teeth and by everting the lips.

Catarrh of the mucous membrane of the nose inducing sneezing, and of the eyes causing photophobia, appears quite early. This catarrhal condition rapidly extends locally; by invasion of the frontal sinuses it causes frontal headache, by spreading to the fauces and mouth it gives rise to a patchy redness, which is followed by congestion and swelling of the tonsils, soft palate, uvula and pharynx. Further extension involves the larynx, trachea, bronchi, bronchioles and even the alveoli of the lung. In some cases the brunt of the catarrhal affection falls upon the larynx in the early stages, and coryza is insignificant. Laryngeal diphtheria is then closely simulated.

The Eustachian tube being in direct continuity with the nasopharynx provides a path for the extension of the catarrh to the tympanum. Deafness, earache and suppuration may result, and the foundation thus laid for more serious complications such as mastoid disease, sinus thrombosis, pyæmia, meningitis or intracranial abscess.

The primary involvement of the respiratory mucous membrane explains the great infectivity of measles in its early stages.

The occurrence of diarrhœa, and sometimes of an ileo-colitis with passage of blood and mucus, points to an affection of the mucous membrane of the digestive tract.

It appears probable that the virus is disseminated in the body by means of the blood stream. This conjecture is strengthened by the fact that the children of mothers suffering from measles are sometimes born with the rash fully out. Moreover, the eruption is itself produced by vascular congestion and small cell infiltration. The congestion and infiltration are best marked in the neighbourhood of the vascular papillæ of the skin, and the resulting infiltration of the subcutaneous connective tissue is a very characteristic phenomenon of the disease. Subcutaneous hæmorrhages are not of very serious import in measles. There is evidence that the supporting vascular stroma of the lung is affected in a manner similar to the skin, giving rise to a special form of interstitial pneumonic infiltration.

Further evidence of the circulatory spread of the disease is occasionally forthcoming in the form of various nervous affections. Convulsions, delirium, coma, hemiplegia, myelitis, lateral column symptoms, peripheral neuritis and transitory insanity have all been known to occur. The myelitis has been found definitely associated with vascular lesions.

The lymphatic glands undergo a moderate enlargement in measles, the submandibular, occipital, tracheo-bronchial, and sometimes the axillary and inguinal groups being involved. The glandular enlargement occurs early, and is usually slight and transitory, but the bronchial glands in particular appear to be rendered prone to tuberculous infection as a result. Often, slight splenic enlargement accompanies the enlargement of the glands. The enlargement of spleen and glands is probably the consequence of blood infection.

Those parts of the body which are liable to bacterial infection from without are prone to suffer severely from secondary infections following measles. Thus the gangrenous process known as noma occurs as a sequel, both in the cheek and on the vulva; secondary laryngeal and pulmonary affections constitute the

great danger of the disease, and the production of otitis media has already been alluded to.

#### GERMAN MEASLES.

Most of what has been already stated about measles also applies to German measles, but it must be remembered that the latter is almost always a much milder and usually uncomplicated disease. Two points of difference stand out: Koplik's spots do not occur in German measles, and the lymphatic glandular swelling is usually more marked. The glands chiefly involved are the occipital, the mastoid and the concatenate groups, but the submandibular, the axillary and the inguinal glands may also be enlarged. In exceptional cases the glandular enlargement may precede the general symptoms for several days.

#### MUMPS.

It is highly probable that the infection of mumps gains access to the salivary glands by way of their ducts, the parotid glands being invaded along Stenson's ducts, and the submandibular along Wharton's ducts. The fact that swelling of the second parotid gland often occurs a few days after that of the first is in favour of ascending duct infection.

The only anatomical connection of the salivary glands with the pancreas, testicles, ovaries and breasts is that effected by the blood stream, so it is assumed that it is by this channel that inflammation of these organs arises as a complication.

The parotid swelling in parotitis corresponds exactly to the position of the gland, and so may be distinguished from inflammatory swelling of the small lymphatic glands which lie close to or are actually imbedded in the parotid. The inflammatory swelling obliterates the sulcus between the mandible and the sternomastoid muscle; extends forwards on the masseter towards the face corresponding to the anterior part of the gland and its socia, and pushes the lower part of the auricle outwards from the head so that the lower part of the ear lies over the central part of the



swelling. The raising of the auricle from the side of the head is very characteristic of parotid swellings. The denseness of the facial envelope which the gland receives from the deep cervical fascia accounts for the great pain and for the absence of inflammatory redness from the skin over the surface. The close proximity of the gland and of its glenoid lobe to the posterior aspect of the mandible, the wedging of the swollen pterygoid lobe between the pterygoid muscles, and the continuity of the parotid fascia with that over the masseter, account for the pain which accompanies all masticatory movements. The relation of the gland to the under wall of the auditory meatus, and the frequent presence of defects in this bony lamina, explain perhaps the occasional extension of inflammation to the tympanum, but the Eustachian tube always offers a direct route for infection of the ear from the mouth. It is said that the so-called "carotid lobe" of the gland, which is prolonged inwards towards the great vessels, may give rise to pressure on the jugular vein, and so produce circulatory disturbance within the skull. The intimate relation of the swollen gland to the sterno-mastoid muscle causes stiffness of the neck, that part being rigidly fixed to avoid pain. The inflammation occasionally extends to the facial nerve which is embedded in the gland, and produces some degree of facial paralysis.

#### ACUTE RHEUMATISM.

Inflammation of the fibrous structures of the body is very characteristic of rheumatism, and, although in adults the capsules of the joints chiefly suffer, rheumatic manifestations are common in the tendons of muscles, in fasciæ and other structures consisting chiefly of connective tissue, such as the pericardium and pleura.

Articular rheumatism chiefly attacks the larger joints, the wrists, elbows, ankles, and knees being particularly liable to suffer, but no joint is immune. The resulting swelling and the accompanying erythema of the skin are most apparent in those joints which are superficial and thinly covered. Consequently

joint swelling and surface redness are best seen in rheumatism of the hands and feet, of the ankles and wrists, and of the elbows and knees. By examining superficial joints it may be obvious that the swelling is not strictly limited to the joint cavity, but also involves the periarticular structures, and sometimes the surrounding tendon sheaths. In joints, such as the hip and shoulder, which are deeply seated and surrounded by muscles, the objective signs of rheumatism may be limited to muscular rigidity.

The attitude assumed by a patient suffering from articular rheumatism is determined in great degree by the joint effusion and consequent joint tension. The position of greatest joint capacity and least joint tension is assumed. Hence the knees and elbows are flexed when inflamed, whilst the ankles and wrists are extended, and there is some tendency for the hand to be deviated towards the ulnar side.

Rheumatism may influence the gait quite apart from articular affection. In children a peculiar form of progression on tip-toe with flexed knees may sometimes be met with, and is attributed to rheumatic inflammation of the tendons of the hamstrings and their surrounding structures. A transitory limp, at first closely resembling that of tuberculous hip disease, may be seen when the fibrous structures around the hip-joint are involved. Wry-neck is the expression of the inflammation of the fibrous structures of the cervical region, and occasionally retraction of the head results from the same cause. The broad lumbar fascia, which gives origin to the erector spinæ, the gluteus maximus, the latissimus dorsi, and other muscles, when inflamed by rheumatism may be the seat of agonising pain readily provoked by movements of the trunk, thigh, or arm.

The subcutaneous nodules of rheumatism have a great tendency to form over thinly covered bony points, and so may at times be found in the following situations: about the olecranon process, the margins of the patellæ, and the malleoli of the ankle; along the vertebral spines, the clavicles, the iliac crests, the temporal ridges, and the superior curved line of the occipital bone.

Forms of interstitial neuritis and of interstitial myositis are also described in connection with rheumatism. Inflammation of the sclerotic coat of the eye and of the iris may occur.

Not only are the fibrous structures of the heart, such as the valves and pericardium, liable to rheumatic inflammation, but the myocardium may also be affected, and here it is said the inflammation is not necessarily of interstitial or fibrous origin.

### Gout.

Clinically the likeness of acute polyarticular gout to acute rheumatism is striking, and the lesions of chronic gout are distributed in the body in a manner similar to those of the more chronic forms of rheumatism.

Gout not only manifests itself as an arthritis, but also invades the cartilages and fibrous tissues of other parts of the body. Such structures as ligaments, tendons, bursæ, and the fibro-cartilaginous helix of the ear, frequently suffer. More rarely the sclerotic coat of the eye, the fasciæ of the palms and soles, the fibrous basis of the aortic and the mitral valves, the pericardium, the laryngeal cartilages and the vocal cords, the tarsal cartilages of the eyelids, the cartilages of the nose, the fibrous sheaths of nerves, and even the cerebral and spinal membranes are known to become the seats of uratic deposits.

The gouty deposit in a joint first occurs a little beneath the free surface of the cartilage and at a point near the centre; it may be inferred that in this position both the blood and lymph streams are sluggish. The presence of synovia and of the fluid of tendon sheaths, both of which contain an excess of sodium salts, is believed by some to be an important factor in determining the local deposit of urate of soda.

As a rule gout first attacks the metatarso-phalangeal joint of the great toe. This joint is subjected to considerable strain in walking, entering as it does into the formation of the inner part of the longitudinal arch of the foot; in addition, it is near the periphery of the circulation, and the arteries in gouty subjects

are frequently diseased. It may be that all these factors conspire in determining the localisation of gout in the joint in question. The first manifestation of gout may, however, occur in some other joint than that of the great toe; the knee, the ankle, and the metacarpo-phalangeal joint of the thumb are sometimes first attacked. These joints are exposed to slight injuries which may appear to determine the incidence of the inflammation.

After the great toe joints, the articulations most commonly affected are the joints of the instep, the ankles, the knees, the joints of the fingers, and the wrists. Less commonly the elbows, shoulders and hips may suffer. When the joints are superficial, and thinly covered, as for instance is the case with the great toe joints, the ankles, wrists, and joints of the thumb and fingers, the accompanying infiltration and dusky erythema may simulate an ordinary cellulitis.

The tendency of fasciæ, ligaments, and tendons to become the seats of uratic deposit may be explained by the poor blood supply afforded to these structures. The tophi are very likely to ulcerate through the skin if they are exposed to slight injury or constant irritation. This tendency may be noticed in connection with gouty deposits in the olecranon bursa, whilst tophi around the great toe joint may, by ulceration, actually lay open the joint cavity. Uratic deposit in important bursæ and in the tendons and ligaments of a joint may cripple a limb as effectually as actual articular gout would.

The deformities produced in the hands by affection of the finger joints are characteristically irregular, the fingers appearing knotted and bent like the gnarled roots of an old tree. The great toe joint becomes deformed and often ankylosed; the knee and elbow may become fixed in a flexed position and some degree of rigid talipes may be also induced.

#### RICKETS.

Although bone changes are not the sole lesions in rickets, they form the most striking feature of the disease, and it is with

them that applied anatomy has mostly to deal. The disease chiefly manifests itself in the epiphyses. Here the normal process of intra-cartilaginous bone formation becomes perverted; the layer of proliferating cartilage becomes increased in breadth and thickness, and the progress of ossification in it is imperfect and irregular. The characteristic thickening of the bones is due to the presence of the imperfectly consolidated cartilage, and so is found near, but not absolutely at the ends of the bones. In the long bones the medullary canal also becomes enlarged, the hard bone is absorbed, and soft spongy bone lying under thickened periosteum takes its place. In estimating the evidence of rickets it is well to bear in mind that the ends of the ribs, where they join their cartilages, are normally slightly enlarged.

The deformities of rickets are produced in various ways. Many are the results of attitude and pressure, some are due to muscular action, and others are caused by atmospheric pressure acting in conjunction with impeded inspiration.

Deformities due to attitude may often be seen in the lower ends of the tibiæ and in the chest. They may also occur at the lower end of the femur. Children who have not learned to walk often lie or sit with the lower ends of the tibiæ crossing each other; the ends of these bones then become bent by mutual pressure, and the curve of one bone fits accurately the curve in the other. The tendency of young children with rickets to lie with the forearms flexed over the front of the chest accounts for the presence of a longitudinal sulcus at the side of the thorax into which the upper arm fits, and which at its lower end is often prolonged obliquely forwards and upwards, corresponding to the position of the forearm. It is believed that a bending inwards of the lower end of the femur produced by sitting or lying with the knees crossed is the basis of some cases of knock-knee, whilst in other cases the lower end of the femur may become so twisted on the shaft that the knee points outwards.

In children who have learned to walk, an exaggeration of the normal forward bend of the tibia in its lower parts may be produced; the weak point of the bone is at the junction of the

middle and lower thirds of its shaft, and it is here that the tension of the calf muscles and the weight of the body conspire to produce the maximum deformity. Similarly the natural forward and outward curve of the femur may become exaggerated and softening of the upper part of the shaft, near the trochanters, or of the neck may cause the latter to sink under the weight of the body, and produce the condition known as *coxa vara*.

The mechanism of production of knock knee is disputed, and possibly not always the same. It may, as already mentioned, be caused by faulty posture which allows the end of the femur to bend inwards, or it may be due to laxity of the internal lateral ligament of the knee joint, which allows the bone to become separated on the inner side. The deformity is perpetuated by the unrestrained growth of the adjacent inner parts of the femur or tibia or of both. This growth is usually supposed to occur at the epiphysis, but some believe that the diaphysis is at fault. As a result, instead of the weight of the body being transmitted through the knee joint and shaft of the tibia to the centre of the ankle, it falls outside the knee joint, aggravating the deformity and also inducing flat foot.

The pelvis, when deformed by rickets, usually undergoes antero-posterior compression; the weight of the trunk transmitted from above causes projection of the sacral promontory and flattening of the cavity. A reniform outline is thus produced. Sometimes, however, especially if the rickety softening occur after the acquirement of walking, a trefoil pelvis similar to that of osteomalachia results. This is due to the additional pressure caused by the indriving of the heads of the femora against the acetabula.

The epiphyses of the upper limb are enlarged first because the child crawls before learning to walk. The bones of the forearms may become bent with convexity outwards if the child is in the habit of supporting the weight of the body with the hands, or of crawling on hands and knees. The humerus also becomes curved outwards, the curve corresponding to the point of attachment of the deltoid muscle.

Atmospheric pressure, acting during inspiration, may drive in the softened ribs in the neighbourhood of the costal cartilages causing a lateral sulcus with curved projection of the sternum and pigeon breast. The action of the diaphragm on the lower six costal cartilages, to which it is attached, is believed to produce constriction of the lower thoracic zone, and at the same time the costal margin is everted by the mass of the enlarged liver, and also by the enlarged spleen.

An accumulation of imperfectly ossified spongy membrane bone at the centres of ossification of the parietal and frontal bones causes an exaggeration of the frontal and parietal eminences. In the parietal bones, and the vertical plate of the occipital bone, a process of atrophy known as cranio-tabes may occur, so that the skull in these regions becomes thin and flexible or is even perforated. The weight of the head, resting on the pillow, may produce flattening and deformity of the bone thus softened. The process of dentition is delayed by rickets, the proper development of the teeth being interfered with. In addition certain deformities of the jaws are induced. The mandible is stunted in its growth, and the alveolar margins of the jaws may become inverted by pressure of the masseters and muscles which surround the oral aperture. In infants the gentle continuous curve of the spine becomes exaggerated, so that it is much more convex backwards than is normal. This is the result of the weight of the head and the relaxation of the supporting muscles and ligaments. The curve usually rights itself when the child is supported by the arms, but occasionally there appears to be some rigidity in the lumbar region. Later on lateral curvature may also show itself.

Greenstick fractures are common in the long bones and especially in the clavicles. The softened bones yield readily to slight violence, but the pulpy periosteum retains the ends in apposition.

## CHAPTER V.

### INTERNAL PARASITES.

PARASITES of the **flake family**, as a rule, enter the body by way of the alimentary tract, and pass thence to the liver by means of the bile duct. They may produce symptoms of biliary obstruction with jaundice and sometimes liver abscess. Occasionally ascites and hæmatemesis also occur, indicating portal obstruction. One member of the family, *Distoma Ringeri*, is parasitic in the lungs of man, being found in small cavities which communicate with the bronchi; it is the cause of endemic hæmoptysis in Japan. Unlike the common flukes, it may be more widely disseminated in the body of its host, and has been found in the brain, the connective tissues of the orbit and the peritoneum. It probably finds access to the pulmonary veins, and so is carried to the right heart, and ultimately into the arterial stream. It is obvious that the liver flukes are not thus favourably situated for dissemination.

The parasite *Bilharzia hæmatobia* also belongs to the fluke family, but differs from others in the fact that the sexes are separate. Its mode of entry into the body is not, as yet, accurately determined; the mouth, urethra, anus, and skin have all been suggested as portals. Sexually mature worms are usually found in the submucous tissues of the bladder and rectum, and in the large venous sinuses of the latter. In the female they may occur in the walls of the vagina. Young worms may be discovered in the portal system and sometimes in the general venous system, as might be expected from the location of the adult worms at the point of union of the two. The spiked ova may be found infiltrating the tissues of the urethra, bladder, ureters, rectum, and even the liver. Urethral fistulæ, vesical calculi and growths, cystitis, dilated ureters, secondary renal inflammations, and sometimes a



characteristic form of cirrhosis of the liver result. Hæmaturia may be present, chiefly at the end of micturition, owing to pressure exerted by the contracting bladder.

**Worms.**—The common *round worms* are practically confined to the intestinal tract, being inhabitants of the small gut; the bowel is sometimes obstructed by tangled masses of these worms. The parasites have a tendency to wander, and are especially prone to insinuate themselves into small openings and canals. They have been found impacted in the biliary and pancreatic ducts, have wandered through holes in the bases of ulcers of the bowel, and escaped also through a perforated vermiform appendix. They have crawled into the œsophagus, or been ejected from the stomach and so made their way into the larynx, trachea and bronchi, into the accessory sinuses of the nose, and even into the Eustachian tube and middle ear, escaping through a ruptured tympanic membrane. By passing through adherent perforated bowel they may reach cavities with which the latter has no normal communication, such as the urinary bladder. The worms are sometimes found in abdominal abscesses, having reached them from the bowel, or possibly, in those which point at the umbilicus, along a patent Meckel's diverticulum. Wandering from the anus they have been known to find their way into the neighbouring vagina.

*Thread worms* are said mostly to congregate in the cæcum and ascending colon; they have been found in numbers in the vermiform appendix. The females, migrating towards the anus to deposit their eggs, set up intolerable itching, and by wandering to the vulva irritate that part. They may even enter the bladder by the urethra. As the result of the irritation and consequent scratching, auto-infection occurs, the ova being unconsciously conveyed to the mouth by the fingers and swallowed.

*Whip worms*, like thread worms, favour the cæcum and bowel in its immediate neighbourhood.

The parasites of *ankylostomiasis* gain access to the body by the mouth and possibly through the hair follicles of the skin in those

parts of the body exposed to friction and contamination with damp, infected soil.

Infection with *Trichina spiralis* occurs as the result of eating measily pork. The investing capsule having been dissolved by the gastric juice, the worms mature and breed in the upper part of the small intestine. It is said the adult female may partially penetrate the intestinal wall, and actually deposit her ova in the lymphatic sinuses of the bowel. Whether the lancet-shaped embryos thus reach the spaces or penetrate the bowel from its interior by their own endeavours is immaterial, they become widely disseminated. Two views are held as to the anatomical route by which this dissemination occurs. One idea is that the embryos, entering the lymph spaces, are carried along the thoracic duct to the veins, traverse the heart and lungs, and are ultimately carried to the muscles by the arterial blood stream; the other view is that, after penetrating the wall of the bowel, they gain access to the root of the mesentery, or cross the peritoneal cavity, and having thus reached the subperitoneal connective tissue, migrate along its planes to the interstitial tissue of the muscles, and in smaller numbers to the subcutaneous tissues of the body. The muscles most affected are, in order, the diaphragm, especially its crura; the intercostals; the cervical and laryngeal muscles; the muscles of the eye; the biceps and triceps. In the larger muscles the parasites are most numerous near the tendinous ends, possibly the lateral pressure during contraction is less at these points. The parasites lie between the individual muscle fibres, and are not actually within the sarcolemma as was formerly supposed.

The muscles infected become swollen, tender and hard. Implication of the diaphragm and intercostals interferes with respiratory movements, whilst infection of the masticatory muscles, the tongue, pharynx and larynx causes the actions of chewing, talking and swallowing to be excessively painful. The patient is quite crippled by the inflammation of the muscles of the limbs and trunk, and assumes a semiflexed attitude to relieve muscular tension as far as possible. The œdema which

accompanies the disease may possibly be due to lymphatic obstruction or inflammation.

**Tape Worms** are confined to the intestine, the head being adherent to the mucous membrane. The spaces beneath the valvulæ conniventes afford a particularly secure and protected point of attachment.

The cysticercus stage of *Tænia solium* occasionally occurs in man, the eggs having been swallowed or possibly having even reached the stomach by regurgitation when the intestine is inhabited by the mature worm. The hexacanth embryos penetrate the blood-vessels and are carried to the brain, heart, eye, and other parts. The rarity with which they are found in the liver throws doubt upon the carriage by the portal vein. When found free in the chambers of the eye they have probably escaped from the vessels of the choroid or iris. The presence of the vesicles in the eye, where they can be seen with the ophthalmoscope, and in the skin whence they can be excised for examination, is of assistance in diagnosis. Pressure largely influences the size and form of the vesicles. In the cerebral ventricles where they are free to grow they attain a larger size than elsewhere, attaining the diameter of nearly an inch in some cases. In the subarachnoid space they may assume a flattened dendritic form.

When the ova of *Tænia echinococcus* are ingested by man the six-hooked embryos are set free in the stomach by digestion of their chitinous envelopes, and work their way through the mucous membrane of the stomach into the blood-vessels of its walls. The embryos having been found in the portal vein, it is reasonable to infer that their distribution is mainly effected by this vessel. This would account for the great frequency of hydatid cysts in the liver, and also account for the fact that, next to the liver, the lungs are the commonest site of the disease, for if the embryos succeed in traversing the liver, the normal course of the circulation would carry them through the hepatic veins to the right side of the heart, and thence to the pulmonary capillaries. The parasites which have succeeded in traversing both the hepatic and the pulmonary filters are carried on by the

blood stream and may settle in connective tissue elsewhere. Possibly embryos may also reach the liver directly by penetrating that part which is moulded on the anterior wall of the stomach, and may also directly infect the peritoneum by entering it after passing through the stomach walls. The peritoneum may be infected by the direct rupture into it of a cyst of the liver or other peritoneal-covered viscus. There is no evidence that the parasites travel by way of the lymphatic trunks, but it is suspected that they may travel along the connective tissue planes in the body; subserous areolar tissue is a favourite nidus.

Hydatid cysts may rupture into various passages and cavities in the body. If these passages or cavities are in free communication with the external air or the digestive tract, as occurs when the cysts rupture into the bronchi or bowel, septic organisms may gain access to the cyst and suppuration ensue.

The severity of the symptoms induced by the growth of hydatid cysts is influenced to a considerable degree by their situation, which determines the amount of expansion possible. Hydatids of the liver and spleen may attain a great size without producing urgent symptoms; in the thorax the unyielding walls and retractile lungs cause dyspnoea to be a comparatively early sign; cysts impacted in the pelvis cause marked symptoms of compression of bowel and bladder; cysts in the cranial cavity and spinal canal soon produce pressure symptoms, which however, are sometimes modified by perforation of the bony coverings.

In the lung the smaller bronchi may freely communicate with the space which is filled by the cyst, and when the latter ruptures, or is torn by the insertion of an exploring needle, the lung may be deluged by the contents of the cyst, with a fatal result.

In the heart, cysts favour the connective tissue of the auriculo-ventricular septum. By rupture into the cavities of the heart pulmonary or systemic embolisms may occur.

Cysts of the liver may grow from its upper surface in the form of dome-shaped projections which encroach on the lung base; such cysts usually project into the right half of the chest, since this is in more immediate relation with the liver. When

examining for such projections the anatomical limits of the lower edge of the lung must be accurately known. The pleural sac intervenes, and irritation of this may induce an effusion which obliterates the domed outline of dulness projecting from the liver.

The relations of the liver determine to some extent in what directions hydatids of this organ may rupture. Cysts near the upper surface may open into the pleural sac or the base of the adherent lung; bile-stained cysts may then be expectorated. The pericardial sac has also been invaded. In some cases the liver cyst presents through the lateral thoracic wall or in the epigastrium. Cysts which project from the under surface of the liver are liable to rupture into the greater sac of the peritoneum or more rarely into the lesser sac. Some such cysts open directly into the duodenum or into the stomach, where that is closely applied to the under surface of the liver. Rupture into the pelvis of the right kidney may also take place.

Splenic and renal cysts are uncommon. The former may bear similar relations to the left lung and pleural sac that liver cysts hold to the right; or they may encroach on the abdominal cavity. Renal cysts may open into either the large or small intestines, or the peritoneal sac or even the lung, but most commonly tend to discharge into the ureters, inducing renal colic with the passage of cyst fragments, scolices or hooklets.

The omenta, the mesenteries, the subperitoneal tissue of the abdomen and the pelvic connective tissue, including that which forms the basis of the broad ligaments, and that which lies between the bladder and the rectum form favourite sites for hydatids. Cysts may also be found in the connective tissue of the ischio-rectal fossæ, of the neck, trunk, and even of the limbs.

An interesting fact is noticeable in connection with hydatids of the brain. The cysts are mostly in the cerebrum, and are twice as common in the right hemisphere as in the left; they are rare in the cerebellum. This distribution points to an embolic origin, and conforms to the ordinary distribution of cerebral embolisms. Hydatid cysts of the brain give rise to little or no surrounding inflammation, and are said never to suppurate

spontaneously ; both these facts are explained by the protection from external infection afforded by their position. The cysts often communicate with the lateral ventricles, and serious loss of cerebrospinal fluid may follow operations for their removal.

Hydatid cysts in the spinal canal may be extradural or intradural. The former may have extended from the vertebral bodies or be primary, and in such cases should be very favourably situated for operative interference.

Hydatids of bones favour the cancellous tissue and medullary cavities. In bone, possibly as the result of pressure, the cysts usually, although not invariably, assume the exogenous form.

As would be expected, by virtue of the blood-borne infection, hydatid cysts are occasionally met with in situations other than those described above. The orbit, pancreas, and the vermiform appendix may be mentioned in this connection.

**Filiaræ.**—Embryo filarial parasites are to be found in the circulating blood, but the mature parasites of this group, so far as they infect man, are found in the subcutaneous, interstitial, subperitoneal or mesenteric connective tissue planes, with slight variations according to species ; they also occur in the lymphatics. Indications of the presence of the mature worm are forthcoming when it is located in the subcutaneous or subconjunctival connective tissue, or happens to block the thoracic duct or some of the larger lymphatic trunks. The female of one species, the guinea-worm, actually perforates the integument to discharge her ova ; the perforation occurring at a spot in the foot or leg likely to come into contact with water, since the young parasites need an intermediate aquatic host. The embryos of other species of filariæ, circulating as they do in the blood stream, are able to be extracted from it by certain blood-sucking insects.

The *Filaria Bancrofti*, which is the parent form of the well-known *Filaria nocturna*, particularly favours the lymphatics of the trunk and extremities, and the complications induced by the presence of this worm can be explained on anatomical grounds. The occlusion of lymphatics, which is caused by the presence of the parasite or the inflammation it has induced, is followed by

lymphatic distension below the obstruction. When the thoracic duct itself is the site of this obstruction its tributaries become varicose, and a collateral lymph circulation is attempted; the chyle stream becomes actually reversed in the lower part of the duct and passes by way of the pelvic lymphatic trunks to the femoral and inguinal regions and scrotum, and thence upwards in the abdominal walls to reach the lymphatic system of the upper part of the body. The varicose lymphatics of the collateral circulation tend to give way and discharge the chyle they convey under these altered circumstances. They may weep on the surface of the skin, fill up the tunica vaginalis testis with chylous fluid or rupture on the surface of some part of the urinary mucous membrane and give rise to chyluria. The inguinal and femoral groups of lymphatic glands which are interposed in the course of the reflux stream become swollen from varicosity of their lymph spaces, and the lymphatic tissues of the scrotum are swollen by a similar mechanism. The enlarged lymphatics are in situations exposed to slight injury, and may become acutely inflamed.

In some cases the lymphatics are thought to be blocked in another way, *i.e.*, by the discharge of immature ova from the *filaria nocturna*, practically an abortion, for the worm is normally viviparous. The ova, on account of their breadth, are not capable of traversing the lymphatic trunks, and consequently become impacted in the efferent lymphatics of the part of the body in which the worm is ensconced. Elephantiasis results in the part thus deprived of free lymphatic drainage. Under such circumstances it is easy to see that filarial embryos may be absent from the general circulation and yet found in the lymph of the diseased member. The presence of filariæ in other parts of the body will probably explain those instances where the embryos are found in both situations.

## CHAPTER VI.

### TUMOURS.

THE genesis, incidence, growth, and spread of tumours is much influenced by anatomical considerations.

Tumours may be classified into four groups: Tumours having their origin in connective tissue, tumours having their origin in epithelium, tumours designated dermoids, and tumours termed cysts.

#### CONNECTIVE-TISSUE TUMOURS.

The fact that every tissue of which an organ is composed can give rise to a tumour implies that a knowledge of the anatomy of the organ, and therefore of its histology, is an excellent guide to an enumeration of the primary tumours which may arise in it.

A growing bone in a young subject affords a typical example. Composing it will be found bone, both cancellous and compact, cartilage, both articular and epiphyseal, periosteum, red medullary marrow and fat. Thus an osteoma, chondroma, sarcoma, myeloma, fibroma, or lipoma may be found in connection with a growing bone.

#### **Osteomata**, or bony tumours.

There are two varieties of osteomata, the cancellous and the compact. Speaking generally, developmental and anatomical considerations account in great measure for the special position and growth of the two forms. *Cancellous osteomata* are nearly always associated with epiphyseal lines, and particularly with that epiphysis from which the bone chiefly increases in length. Thus the lower end of the femur, the upper end of the tibia, the upper end of the humerus, and the lower end of the radius are frequent sites of such bony growths. In their early stages they



ENCHONDROMA OF FINGER.

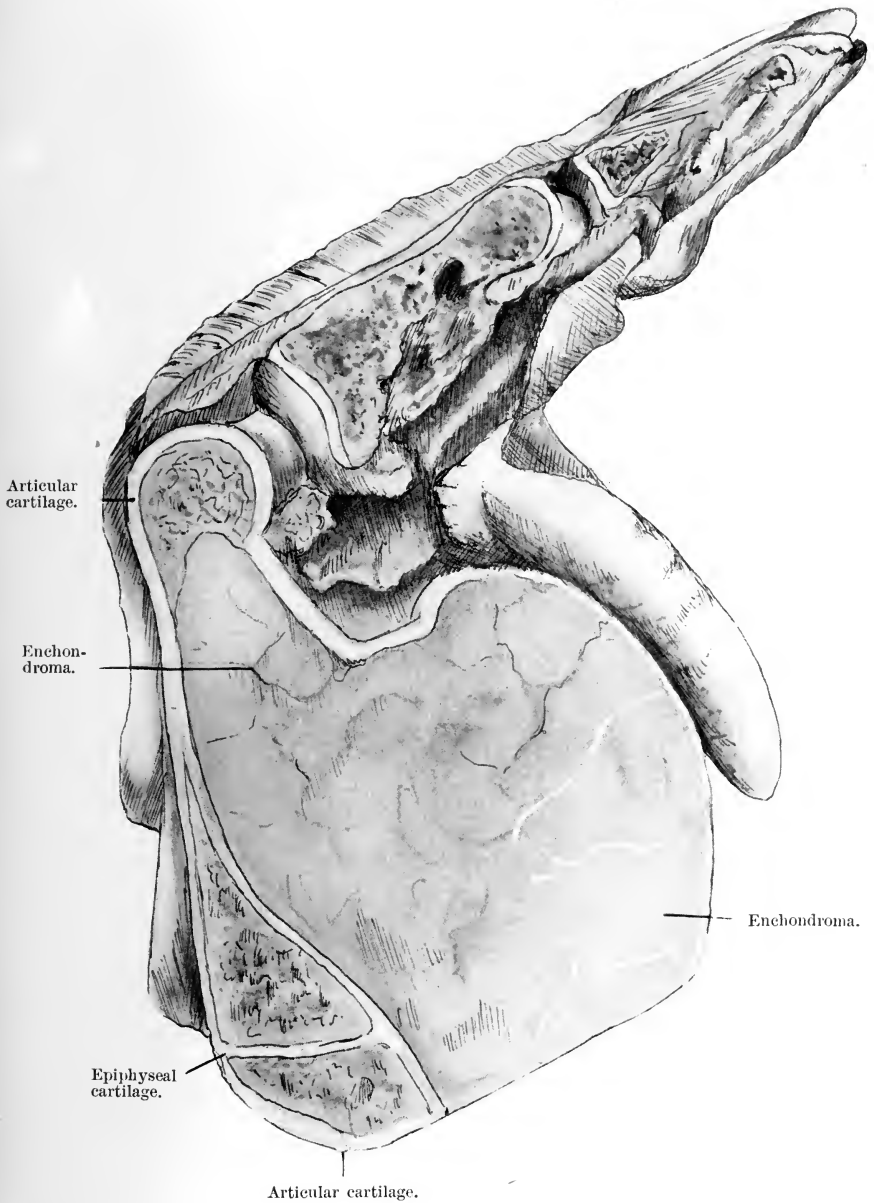


FIG. 3.—Section of the three phalanges of a finger affected with an enchondroma, showing position of varieties of cartilage.

[To face page 66.



are capped by cartilage, and continue to increase in size so long as there is cartilage left to ossify. They are not uncommonly multiple. They are painless as a rule, but if they increase in size to such an extent as to lie close under the skin of a part subjected to pressure, an over-lying bursa may form, and inflammation and pain result. This may be typically present over an osteoma at the lower end of the tibia, projecting so as to cause friction of the skin against the boot. Again, local or referred pain may be in evidence when an osteoma presses upon a nerve. Rarely actual paralysis may result from such pressure as when an osteoma of the lower end of the femur implicates one or other of the popliteal nerves.

*Compact* or *ivory osteomata* are nearly always found upon bones which have been developed in membrane. Hence they are generally associated with the flat bones of the skull, and with the mandible. They occur at times in the osseous wall of the external auditory meatus, and from their position may lead to annoying deafness by filling up the canal. The air spaces of the frontal bone form another site for their incidence, as also does the angle of the mandible.

*Exostoses*, or bony out-growths, occur chiefly at the attachments of certain muscles, as, for instance, the lowest part of the insertion of the adductor magnus, causing an elongated adductor tubercle. Also they are met with frequently on the terminal or unguis phalanx of the great toe, probably induced by the intermittent pressure to which this part is so subject.

**Chondromata**, or cartilaginous tumours.

Tumours composed of cartilage generally arise from the cartilage in connection with growing bones, but they are also formed in organs which at one period of development contained cartilage.

Associated with bones they will naturally be seen as out-growths from epiphyseal cartilages, or more rarely as endosteal tumours derived from islets of diaphyseal cartilage left behind unossified. In the latter case they are usually found expanding metacarpals or phalanges, and are in most instances multiple.

Associated with such organs as the parotid and submandibular glands, they most probably originate in remnants of developmental cartilage. In the case of the parotid, it is possible that remains of the cartilage of the first branchial arch may account for the cartilaginous portion of the mixed tumours. When cartilaginous elements occur in the testis or ovary they can only be explained on the ground that they belong to teratomata.

**Lipomata**, or fatty tumours.

Wherever adipose tissue is found in the body, there lipomata may develop. There are, however, certain positions where they are more commonly seen than elsewhere, and generally there is an anatomical explanation of their existence in these areas.

Subcutaneous lipomata are very frequent. The subcutaneous tissue contains much fat in well-nourished individuals, and in particular regions, often where there is a likelihood of pressure, as for instance about the shoulders and the buttocks, encapsuled lipomata are prone to develop. Diffuse lipomata, on the other hand, affect their own characteristic situations, such as the neck, the axillæ and the groins.

Extra-peritoneal lipomata arise from any part of the fatty tissue lying in contact with the peritoneum. Appendices epiploicæ are natural sub-serous lipomata. Sessile extra-peritoneal fatty tumours may grow to a large size, and may excite injurious pressure on surrounding organs.

Not infrequently small lipomata grow in the extra-peritoneal tissue beneath and protrude through adventitious openings in the linea alba, or through the deep inguinal or the femoral rings, and after a while, by dragging upon the peritoneum, cause a finger-like process of that membrane to be formed. Lipomata of the spermatic cord may be explained by the cord drawing down with it some of the extra-peritoneal adipose tissue. This is further shown by the fact that occasionally such a lipoma contains cells which are closely allied to adrenal elements, derived from the lumbar region in which the testis was originally developed.

The fatty tissue found external to synovial membranes, particularly that of the knee, outside the spinal dura mater, in the

submucosa, and in connection with periosteum, gives rise to sub-synovial, extradural, sub-mucous, and periosteal lipomata.

Lastly intermuscular planes, especially of the abdominal wall, often give lodging to considerable quantities of fat which may form intermuscular lipomata. Occasionally also lipomata are seen within the sheaths of the biceps, deltoid, rectus abdominis and other muscles, constituting intramuscular lipomata.

**Fibromata**, or fibrous tumours.

Fibromata may grow from connective tissue anywhere, but they are peculiarly liable to occur in connection with periosteum, and particularly that periosteum which is associated with mucous membrane, as for instance the muco-periosteum covering the gums, from which an epulis, innocent in character, is derived. Frequently these small tumours are attached to the membrane lining the tooth socket, and are therefore in a way pedunculated.

Again, the fibrous tissue of the connective tissue sheath of a nerve is a frequent starting-place of fibromata, the swelling then being called a false neuroma. On the termination of some of the sensory nerves small fibrous growths occur, extremely painful to the touch and frequently spoken of as painful subcutaneous tubercles. It is somewhat questionable whether these two varieties of swellings should be classified under fibromata or neuromata.

Most of the polypi growing from mucous membranes are fibromata which have undergone a certain amount of myxomatous degeneration; hence nasal polypi and the firmer naso-pharyngeal outgrowths are primarily fibromata.

In some instances pedunculated fibromata appear attached to the skin, particularly about the scrotum and labium. Although in some cases such tumours are thought to have their origin in connection with nerve terminations, the amount of fibrous tissue in the actual growth rather leads to their being classified under fibromata than under neuromata. When very numerous the nodules constitute a disease spoken of as neuro-fibromatosis.

**Angiomata**, or blood tumours.

Angiomata are tumours composed of an abnormal formation of blood vessels. As a rule they are spoken of as *nævi*.

One form of the subcutaneous capillary *nævus*—the “port-wine stain”—is most often found upon the face, but it may be seen upon the trunk or even a limb. Its frequency upon the face and its actual distribution in particular regions of the cheek may be dependent upon nerve distribution, the influence of the nerve during development possibly having some obscure bearing upon the excessive formation of capillaries.

The other, circumscribed, form is very common, and may be found in almost any part of the skin, but it is peculiarly liable to be seen on the face or head, on the trunk, and on the external genital organs.

The more deeply placed *nævi*—the cavernous angiomata—which are often in structure extremely like the vascular portions of the corpus spongiosum, may be found in the same regions as the capillary form, or beneath the deep fascia, in muscles, or even in the viscera, as for instance in the liver.

The last form of angioma, frequently termed plexiform, is rare, and almost entirely confined to the scalp and upper limb, and consists for the most part of vessels which should rank as arteries.

**Lymphangiomata**, or lymphatic tumours.

A lymphangioma bears the same relation to lymphatic vessels as an angioma bears to blood vessels.

A lymphatic *nævus* is generally seen in the condition known as macroglossia, in which there is congenital enlargement of the tongue. Occasionally it may occur in the skin, and particularly in regions where lymphatic glands are collected.

Another form of lymphangioma occurs as the so-called “hydrocele of the neck,” which is in reality a lymphatic cyst. Cysts may also arise in the axillæ and the adjacent part of the thoracic walls. They are usually present at birth, and bear some resemblance to the large subcutaneous lymph spaces of frogs.

**Myomata**, or muscle tumours.

Myomata are generally described as being tumours composed of unstriated muscle fibre, and may be associated with any of the organs in which involuntary muscle may be present. They are peculiarly liable to occur in connection with the uterus, and may in these situations be mixed with a considerable quantity of fibromatous tissue. In connection with the alimentary tract myomata have been most frequently seen in the œsophagus, the stomach and the duodenum—in other words, in the proximal portions of the tract.

**Myelomata**, or red marrow tumours.

These tumours are composed of tissue very similar to the red marrow of growing bone, and were formerly spoken of as myeloid sarcomata. They are nearly always found at the cancellous ends of long bones, and most frequently at that end which is the epiphysis from which the bone chiefly grows in length, as for instance the lower end of the femur, the upper end of the tibia, and the lower end of the radius. Another common place for a myeloma to originate is in the cancellous tissue of the mandible, the growth expanding the bone so as to produce the characteristic thinning of its compact outer layer.

**Sarcomata**.—This is the only truly malignant connective-tissue tumour. It may occur in any mesoblastic tissue, and therefore in almost every organ of the body. While this is true, the growths are found much more commonly in certain regions than in others; periosteum, fascia and subcutaneous tissue are peculiarly liable to be the seat of round- or spindle-celled sarcomata.

Again, all composite glands, such as the kidney, testis, parotid, etc., are prone to the incidence of sarcomata.

The pigmented form of the neoplasm—melano-sarcoma—arises in the skin, in the uveal tract, and sometimes in mucous membranes.

The spread of sarcomata is governed to a great extent by anatomical considerations. All tumours of this nature are extremely vascular, and the blood vessels connected with them are ever prone to carry the elements of the growth to distant

parts; hence, organs far removed from the primary seat of disease rapidly become infected with secondary deposits. While, however, it is true that sarcomata are chiefly disseminated by the blood stream, it is also true that a considerable number are also spread by the lymphatic system, for sarcomata of periosteum, bone, tonsil, testis, ovary, parotid and skin, as well as all melanomata, frequently give rise to secondary deposits in lymphatic glands.

### EPITHELIAL TUMOURS.

Epithelium occurs on the surface of the body, covering the skin and the appendages thereto, and lining mucous membranes, or, deeply, as a lining to the secreting portion and ducts of glands.

#### TUMOURS ARISING FROM SURFACE EPITHELIUM.

**Papillomata**, or warts.—These, the common, innocent epithelial tumours—occur on the skin, but particularly on those parts which are liable to be irritated or infected; therefore they are common about the hands, the glans penis, prepuce, vulva, and surrounding skin. The crops of warts so frequent on the fingers are due to the exposed position of these organs, and therefore their constant liability to be irritated and infected.

There are two surfaces covered by stratified epithelium other than the skin which may give origin to warts. The first is the dorsum of the tongue, on which papillomatous tumours are by no means infrequently seen, and are again as a rule the outcome of irritation and infection. The second is the true vocal cord, where again papillomata, sometimes multiple, are frequent. Their occasional disappearance after tracheotomy in children may be in part due to the rest given to the larynx.

**Epitheliomata**.—An epithelioma is a squamous-celled carcinoma, and therefore may arise on any surface covered by a stratified epithelium. It is undoubtedly true that epithelioma



occurs where there is long-continued irritation, consequently epithelioma is most often seen on those spots where irritation (and it must be remembered possibly also infection) is most likely to occur. Hence any exposed portions of skin, such as the hands and lips, are frequently the sites of epitheliomatous deposits.

Other common sites are those in which two skin surfaces tend to come in contact, as for instance about the anus and vulva where there is great liability to irritation, to small wounds and possibly to infection. Also, where the skin is thrown into rugæ there is again the liability for squamous-celled carcinoma to make its appearance, as on the scrotum, forming the so-called "chimney-sweep's cancer." It is often said that the line of junction of skin and mucous membrane—that is, where transition from one kind of epithelium to another takes place—is a region in which epithelioma commonly originates. While this is undoubtedly true, there is no clear evidence that it is due to the change from one form of epithelium to another, but it is of everyday observance that these same spots, if on the surface, are those commonly the site of slight injuries.

Again, as in connection with warts, so the epithelium covering the tongue, and also that forming the superficial layer of the true vocal cord, may give rise to squamous-celled carcinoma. Hence the connection between the simple papillomata and epitheliomata is self-evident: both originate from epithelium, and both are found frequently where irritation, and particularly prolonged irritation, is common, and slight injuries such as a crack or a fissure often occur.

Epitheliomata, like other malignant growths, spread in one or more of three ways. First, by direct extension into the surrounding tissues, which extension is termed infiltration. It is interesting to note however that this infiltration by no means occurs only in the line of least resistance, and so differs from the extension of innocent tumours, which merely push the parts aside, or of abscesses finding their way to the surface. Secondly, epitheliomata spread to distant parts by means of the lymphatic

system. In most cases the nearest chain of lymphatic glands rapidly becomes the site of secondary deposits, but it is comparatively rare for the afferent lymphatic vessels and the tissues around them to become early involved. It is possible, however, that when the nearest lymphatic glands have been removed by operation or have become greatly implicated by the new growth, the afferent lymphatic vessels have great difficulty in conveying the lymph stream, with the result that the lymph as it were is dammed back and any virus it may contain affects the walls of the vessels and may escape into the surrounding tissues. It is conceivable that so-called *cancer en cuirasse* is thus occasioned. The third way of dissemination, which is decidedly rare in epithelioma, is by the blood stream.

#### TUMOURS ARISING FROM THE EPITHELIUM OF THE SECRETING PORTION OR DUCTS OF GLANDS.

**Adenomata.**—The secreting epithelium of glands occasionally gives rise to a tumour constructed upon the type of the gland but without the function of producing its secretion. Such a tumour is called an adenoma, and is innocent in nature. While all glands are theoretically capable of producing adenomata, certain organs are the most common sites of the new growth. Thus the mammary gland, sebaceous glands, the prostate, the parotid, the thyroid and the ovary are not so very infrequently the birthplaces of adenomata.

**Carcinomata.**—The epithelium lining the acini of glands is an extremely frequent starting-place of carcinoma; hence occurs the increasingly common origin of carcinoma in the female mamma, and in the glands of the cervical canal of the uterus. Other favourite situations, common to both sexes, are the glands at the pyloric orifice of the stomach, and those in the rectum and other parts of the large intestine.

Such carcinomata rapidly infiltrate the surrounding tissues, usually extending beyond the gland in which they originate, and later involve the skin or mucous membrane and so induce

ulceration and fungation. Further, they disseminate rapidly to the lymphatic glands near by, by the lymph stream, and to distant organs by the vascular system.

#### DERMOIDS AND TERATOMATA.

In the vast majority of dermoids, skin or dermal tissue forms the basis of the tumour. They may be classified in four varieties:—sequestration dermoids, tubulo-dermoids, ovarian or testicular dermoids, and dermoid patches. A knowledge of developmental anatomy explains many of the points observable concerning dermoids.

**Sequestration Dermoids.**—If the surface epithelium becomes included in the deeper tissues a dermoid is likely to be formed. Hence it will be readily seen that such dermoids occur where there is the greatest chance for portions of epithelium to become sequestered. The middle line of the body is therefore a common situation, seeing that there is here a junction of the two halves of the fœtus. In the process of fusion, small portions of epithelium may be cut off by mesoblastic tissue from the surface and thus become buried, and therefore middle line sequestration dermoids are easily explained. On the dorsal aspect they are most often seen in the region of the sacrum and coccyx, where also dimples and pits may be observed and may be considered as abortive attempts to sequestrate surface epithelium. Dermoid cysts in this region which have suppurated and discharged explain at least one form of persistent sacral sinus. In the median line on the ventral aspect dermoids are more common. Occasionally they are found within the thorax, epithelium having been caught by the closing sternum, and thus deeply buried. In addition, those facial and branchial fissures which are found in the face and neck frequently and easily become the sites of inclusion of epiblast. The commonest situation for a facial dermoid is just above the outer canthus of the eye, that is at the external extremity of the orbito-nasal fissure. Sometimes this cyst may have a deep process passing through the bone and into the cranium, hence the

danger of removing them unless there is absolute asepsis. The other fissures of the face much more rarely have true dermoids associated with them.

The branchial clefts in the neck may sometimes develop dermoids lined wholly with skin, but more frequently the cysts formed in connection with them are of the nature of tubulo-dermoids, sometimes called "hydroceles" of the neck.

Dermoids may be occasionally seen about the pinna of the ear, and occur there owing to sequestration of epithelium during the fusion of the primitive auricular tubercles.

Dermoids in connection with the scalp and dura mater may be explained by the original continuity of these two layers, which later become separated by the formation of membrane bone between them.

Implantation cysts, which bear a resemblance to sequestration dermoids, are found in various parts of the body, and are due to portions of epithelium being driven by violence into the deeper tissues, or left behind accidentally during the progress of an operation. Hence such cysts are usually seen about the palm of the hand and the fingers, because these parts are so exposed to cuts and pricks.

**Tubulo-dermoids.**—These arise in congenital tubes, which as a rule become obliterated before birth. There are chiefly three which may be the origin of tubulo-dermoids, the thyroglossal duct or tract, the post-oral fissures, and the post-anal gut.

From the first may arise tubulo-dermoids in the middle line of the base of the tongue, in front of the thyro-hyoid membrane, and in the middle line of the neck below this and down to the isthmus of the thyroid body. It is important to recollect that the cyst may pass up behind the hyoid bone into the substance of the tongue, and to rid the patient of the trouble necessitates complete extirpation of the dilated duct, and not a mere opening of the fluid swelling, which is so commonly mistaken for an abscess.

Similar cysts have been described with pedicles passing between

the mandible and the hyoid bone and between the cricoid and thyroid cartilages.

The post-oral fissures sometimes develop tubulo-dermoids, which will appear in the anterior triangle of the neck, and are placed beneath the deep fascia.

The post-anal gut gives rise to two forms of tubulo-dermoids. One, which is ordinarily termed a congenital sacro-coccygeal tumour, projects from between the rectum and the coccyx, and presents characters of both alimentary and nervous tissue. The other lies between the rectum and the hollow of the sacrum, and may sometimes find its way through the rectal wall, and, ulcerating, discharge its contents through the anus.

**Ovarian and Testicular Dermoids.**—It is particularly interesting that there may be associated with the ovary and with the testis a species of tumour which has all the characteristics of a dermoid. In connection with the ovary they probably arise from the oöphoron, or that portion of the organ which develops the follicles, and are probably due to cells which were originally connected with epiblast. They are therefore essentially the resultant of a true embryonic intrusion into the ovary, and may stand in close relation to teratomata or parasitic foetuses.

Dermoid tumours of the testis must not be confounded with scrotal dermoids, that is, those sequestration dermoids which are to be found in the middle line of the scrotum—in the raphé—the evidence of the original fusion of its two halves. There appear to be true dermoids of the testis proper, developed in probably the same manner as dermoids of the ovary, namely from a misplaced portion of epiblastic embryonal tissue.

**Dermoid Patches**, or hairy moles.—These are essentially moles, or rather hairy moles. It is questionable whether all moles should be classified as dermoid patches, but those which occur in the middle line and in the lines of congenital fissures, as for instance in the line of the fissure between the maxillary process and the mandibular arch, are certainly of such a character. Hairy moles are also seen on the limbs and on the conjunctiva.

## CYSTS.

Cysts may be conveniently classified under three headings, firstly, cysts due to distension of previously existing cavities ; secondly, cysts of new formation ; and thirdly, cysts of congenital origin.

**Retention Cysts** occur wherever there is a secreting organ which possesses a duct, the lumen of which becomes occluded, while at the same time secretion continues. Possibly the most frequent form of this variety is seen in the common sebaceous cyst of the skin, or the mucocele of the mucous membrane.

**Distension Cysts** are those in which a potential cavity, lined with a secreting membrane but with no exit therefrom, becomes distended with its own secretion. A typical example of this is distension of the tunica vaginalis testis with serous fluid, forming a vaginal hydrocele.

**Cysts of New Formation** are those originating in connection with a new growth or a parasite, an excellent example of the latter being a hydatid cyst.

**Cysts of Congenital Origin** are very numerous, and for the most part depend upon the dilatation of developmental tubes which should cease to exist or remain merely as remnants. Congenital neural cysts are due to arrest of the fusion of the edges of the neural groove. Spina bifida usually occurs in the lumbar region where the neural plates are the last to close. The plates are also late in closing at the anterior end of the groove, and a meningocele or encephalocele may form at this site and project at the root of the nose or into the nasal cavity. A cerebral meningocele may also project between the two cartilaginous portions of the supra-occipital bone.

# SPECIAL DISEASES AND INJURIES.



## CHAPTER VII.

### FRACTURES.

#### GENERAL ANATOMICAL CONDITIONS.

A **fracture** of a bone implies in addition some injury to soft parts because of the close relationship of muscles through their attachment to the osseous tissue, of the proximity of nerves and vessels running in bony grooves and canals or through foramina, and of the subjacent position of viscera. It is when severe injury of soft parts is induced that a complicated fracture is in evidence.

Bones enter into the formation of joints, therefore a fracture occurring near or through the articular end of a bone may involve a joint; or a dislocation may be associated with the fracture, constituting a further form of complicated fracture.

The attachment of muscles to bones causes in most cases the deformity which so often follows upon a fracture, and the precise degree or variety of deformity depends upon the position of the fracture and the relative attachment of muscles to the fragments. The laceration of small blood vessels and the consequent extravasation of blood produces in part the swelling which so quickly follows a fracture.

The abnormal mobility present after a fracture is the result of the solution of the continuity of the bone, and the direction of the movement is to a great extent dependent upon the anatomical relations of the bone.

Pain is experienced at the seat of fracture owing to nerve endings being torn or bruised; pain may also be referred in consequence of the irritation of nerve trunks implicated opposite the fracture.

A fracture which does not in any way communicate with the outer air is said to be closed, or "simple." A fracture which does communicate with the outer air, it matters not how, is said to be open, or "compound." It is well to remember in this connection that the track along which air and therefore septic micro-organisms may reach the fractured ends of a bone may be a very devious one. This is particularly so in bullet injuries, where the wound of entrance may be very remote from the site of fracture.

Emphysema, or air in the tissues, may follow the fracture of certain bones because of their anatomical position. Most commonly it is found after fracture of the ribs because of a co-existent wound of the underlying lung. Fracture of the nose leads frequently to laceration of the nasal mucous membrane, and thus to escape of air into the frontal tissues, as also will a fracture involving the frontal sinus, or the antrum of Highmore. Occasionally fractures of the hyoid are associated with escape of air derived from the larynx, and fractures of the pelvis with gas derived from the rectum.

#### SPECIAL FRACTURES.

**Fractures of the Nasal Bones.**—The nasal bones forming the roof of the nose are usually prominent and thus considerably exposed to injury. The bones are to a certain extent protected by the overhanging forehead, and supported by the septum and nasal processes of the maxillæ. Fracture is therefore most commonly through the lower third where they are least supported. A fracture caused by direct violence is usually accompanied by laceration of the mucous membrane of the nasal fossa, even if the skin is entirely untornd. Epistaxis follows; frequently the nose is blown to clear out the blood, and air may be thus forced



through the rent in the lining membrane into the tissues at the root of the nose, and up on to the forehead.

The grave lesion of an open fracture of the ethmoidal portion of the base of the skull may occasionally be produced by the violence which breaks the nose, and therefore prognosis in this common accident must not always be too sanguine, since septic meningitis may ensue.

**Fractures of the Maxilla (Upper Jaw).**—The maxilla is quite superficial, and contains a large air cavity, the antrum, the walls of which, at least anteriorly, are of no great thickness. In spite of these anatomical facts, however, fractures of this bone are not common. But violence, such as a kick from a horse, however, may break it in spite of the protection afforded by the prominent malar bone, the nose, and the symphysis menti. If the fracture involves the antrum, air may escape into the tissues of the cheek, giving rise to a characteristic crackling sensation on palpation. If the alveolar border be fractured, teeth are apt to be displaced, and the mucous membrane of the gum torn, the fracture being then an open (compound) one. The infra-orbital nerve running in its groove and canal is liable to primary or secondary involvement.

The floor of the orbit being formed chiefly by the upper surface of the maxilla, it follows that a fracture of the bone including this portion may cause extravasation of blood into the tissues of the orbit, which, if sufficient in amount, may lead to proptosis.

The lachrymal (or nasal) duct is also liable to laceration or pressure in consequence of a fracture of the maxilla, passing as it does through the inner part of the bone on its way to open into the inferior meatus of the nose.

**Fractures of the Mandible (Lower Jaw).**—The mandible is the only bone of the face which is moveable. The chin, and each angle, form projections which are liable to be met by blows, and yet a fracture at any one of these spots is not at all common, probably owing to the greater thickness of the bone at these points. The weakest part of the bone is in the body, rather nearer the symphysis than the angle, where the incisive fossa,

the canine socket, and the mental foramen all tend to lessen the solidity of the jaw.

All the fractures of the body of the mandible involve the mucous membrane of the gum, and are therefore open (compound) fractures. Hence hæmorrhage into the mouth is a usual, if not a universal, sign. There is generally some displacement in these fractures, but the amount depends very greatly upon the force applied, whether direct or indirect. As a result the regular curved line of the teeth, or their horizontal level, may be altered, a tooth or teeth may be missing, and one may even have dropped between the fragments. In the most typical displacement, the larger or anterior of the fragments where the fracture is on one side of the symphysis, is drawn backwards and downwards by the depressor muscles, the digastric, mylo-hyoid, genio-hyoid and genio-hyo-glossus, and the posterior or smaller portion pulled upwards and outwards by the elevator muscles, the masseter, internal pterygoid, and temporal. If there is a fracture on both sides of the symphysis, there will be a tendency for the median fragment to be drawn backwards and downwards by the depressor muscles, and as a consequence the tongue may fall back, covering the upper opening of the larynx, and inducing dyspnœa or even suffocation.

The inferior dental nerve runs in a canal in the body of the mandible, but is not as a rule torn when the bone is fractured, owing to the fact that displacement is not great. If it is lacerated, the lower lip and the anterior teeth may be anæsthetic. Later, if the nerve becomes secondarily involved in callus, its functions may be interfered with, or there may be much neuralgic pain over its distribution. Fracture of the ramus is rare owing to the protection afforded by the masseter and internal pterygoid muscles. Further, if fracture is present, these muscles, acting practically as splints on either side, prevent much, if any, displacement of the fragments. The condyle may be broken by indirect violence, such as a fall upon the chin. The coronoid process is well protected by the zygoma, and is rarely broken without this arch being fractured at the same time.

## FRACTURES OF THE UPPER EXTREMITY.

**Fractures of the Clavicle.**—The collar bone is one of the commonest bones to be broken. There should, therefore, be certain anatomical reasons for its liability to fracture.

The bone is set between the sternum and the scapula. It thus has to bear the brunt of all falls upon the shoulder, and to a great extent those upon the outstretched hand. Thus fracture by indirect violence is easily produced. The bone is long and slender, and has a thick layer of brittle compact tissue, derived from ossification in membrane, enclosing cancellous tissue, without a medullary cavity. Therefore it readily gives way under direct violence. Lastly it is ossified at quite an early intra-uterine age, and is consequently less capable of withstanding injury in infant life than are some other bones which are still partly composed of cartilage. Fractures of the clavicle in young life are often incomplete, "greenstick," owing to the thick periosteal covering. When the clavicle is broken by a fall on the extended and abducted arm or upon the greater tuberosity of the humerus, force is applied to it through the glenoid fossa of the scapula and the coraco-clavicular ligament, and causes a species of torsion of the clavicle at the junction of the outer with the middle third. It is at this point, therefore, that an oblique fracture usually occurs.

Immediately there is a triple displacement of the outer fragment. The weight of the arm, aided perhaps by the traction of the pectoralis minor, the lower fibres of the pectoralis major, and the latissimus dorsi, induces a downward displacement. Moreover, the trapezius is as it were thrown out of gear, and can no longer support the weight of the upper limb. Inward displacement is caused by the pull of the pectorals, the trapezius, the levator anguli scapulæ, the rhomboids and the latissimus dorsi. In addition, the outer fragment is rotated so that its inner end looks backwards, the point of the shoulder turning forwards. This change of position is brought about chiefly by the action of the pectorals.

The inner fragment is but slightly if at all altered in its position. This is due to the antagonistic action of the clavicular

fibres of the sterno-mastoid above, and the clavicular fibres of the pectoralis major below, in conjunction with the costo-clavicular ligament. Owing, however, to the downward displacement of the outer fragment the skin tends to become stretched over the outer end of the inner fragment, which on this account appears as if it were tilted upwards.

Thus it comes about that the two fragments override one another, the inner surmounting the outer, and a shortening of the long axis of the bone is occasioned.

In the recumbent posture, the vertical weight of the arm is removed, and thus the downward displacement lost or greatly minimised. In this position the weight of the limb tends to draw the shoulder back, and so to reduce the amount of the inward displacement and of the rotation of the shoulder forwards.

It is rare for the important structures which lie posterior to the clavicle to be involved in fracture by indirect violence. The subclavian vein, the subclavian artery, the brachial plexus of nerves, the suprascapular vessels, and the dome of the pleura, usually escape injury. In some cases of fracture by direct violence, one or both fragments may be driven against them. It is further rare for a fracture of the clavicle to be an open one. This is owing to the laxity of the tissues superficial to the bones which allow them to slip aside in direct violence, and to stretch over the bony projection in fractures due to indirect violence.

**Fractures of the Scapula.**—The only fairly common fracture of the scapula is the breaking off of the acromion process, frequently really a separation of the acromial epiphysis which does not coalesce with the rest of the bone until about the age of twenty-two. This fracture is usually the result of direct violence, such as pitching upon the shoulder, or running the shoulder against some unyielding structure. Much displacement is, therefore, uncommon, and the more so because of the thick fibrous covering of this part of the bone. The deltoid may drag the detached fragment somewhat downwards.

The coracoid process may be fractured by direct violence, or by

muscular action. It will then be detached from its base, but its displacement is slight, owing to the hold upon it exerted by the coraco-acromial and coraco-clavicular ligaments, which are seldom torn.

The rarity of fracture of the body of the scapula is due to its free mobility, and its close investiture by the supraspinatus, infraspinatus, and subscapularis muscles. Separation of the fragments is but small, and crepitus may be difficult to elicit, because of the position of the same muscles.

In a fracture through the neck of the scapula, which is the constricted portion internal to the glenoid fossa, the coracoid process will be attached to the outer, smaller fragment. Hence the fragment will be drawn downwards by the pectoralis minor, the biceps and coraco-brachialis, and if the coraco-clavicular and coraco-acromial ligaments are torn, there will be a distinct lengthening of the arm.

**Fractures of the Humerus.**—*Fracture of the surgical neck.*—The surgical neck of the humerus is the one which is most commonly the seat of fracture, partly on account of its being a weak part of the bone, but chiefly because in certain positions of the upper extremity it is subjected to a very considerable strain, as, for instance, when the arm is abducted. The bone breaks as a rule between the base of the tuberosities and the upper part of the insertions of the latissimus dorsi and the teres major muscles. When the lesion has occurred, there is generally a very characteristic displacement of the fragments occasioned by the action of muscles. The upper fragment is abducted by the supraspinatus and externally rotated by the infraspinatus and the teres minor. The lower fragment has its upper end drawn upwards by all the muscles running in a vertical direction, namely the biceps, the deltoid, the coraco-brachialis, and the triceps. It is also adducted by those which have a more or less horizontal direction, namely, the pectoralis major, the latissimus dorsi, and the teres major. In addition, there may be some forward displacement by the action of the great pectoral muscle. If the two fragments remain in apposition as sometimes happens, it is probable that the long

tendon of the biceps acts as a strap, and, together with the long head of the triceps, prevents the other muscles from causing any alteration in the relationship of the two fractured ends. Separation of the upper epiphysis of the humerus, which is an accident which may occur in persons under the age of eighteen, entails a very similar displacement to that mentioned above. The epiphyseal cartilage in general remains attached to the epiphysis rather than to the diaphysis. It is in these two lesions that the circumflex nerve may be involved.

*Fracture of the shaft.*—This commonly occurs about the middle of its length, and may therefore be just above or just below the insertions of the deltoid and the coraco-brachialis. If above these insertions the displacement will be that the upper fragment is drawn inwards by the muscles passing from the trunk to the arm, namely the pectoralis major, and the latissimus dorsi, while the lower fragment will be drawn upwards by the deltoid, the biceps, the coraco-brachialis, and the triceps, and slightly outwards by the deltoid. If, however, the line of cleavage is below the insertions of these two muscles, the upper fragment will now be abducted by the deltoid, and the lower fragment is drawn upwards and inwards, to a slight extent by the biceps and the triceps, still further tending to push the lower end of the upper fragment outwards. The intervention of the muscular fibres, which surround the bone in the region of the fracture, between the fragments may effectually prevent union. In some instances these fibres are actually driven into the medullary cavity of the bone. Two other explanations of non-union from an anatomical point of view are also given, one that the nutrient artery which enters the bone near the seat of the fracture is itself liable at the same time to be torn across, and the other that there is apt to be movement at the site of the fragments, when the elbow joint is fixed by the splint adjusted for treatment.

The musculo-spiral nerve runs a course from within outwards behind the shaft of the bone, lying in a musculo-osseous canal close against the bone itself. Hence it follows that there is a great

liability for it to be either injured at the time of the fracture by the broken ends of the bone, or to become involved in the callus which quickly forms around the fragments. The first lesion produces what is called primary paralysis, and the second produces what is termed secondary paralysis, and is the more common of the two. The effect of this paralysis is to produce wrist-drop, and it is therefore readily recognisable.

The musculo-spiral nerve is best exposed behind the middle of the shaft of the humerus by an incision which is made posteriorly exactly in the middle in all directions, that is, half way between the apex of the shoulder, and the tip of the elbow, and half way between the outer and the inner sides of the arm. If the incision be deepened through the substance of the triceps the nerve is exposed without difficulty. It is important to remember, when the nerve has been freed by operation from the callus, that a layer of muscular tissue derived from the adjacent triceps should be inserted between the nerve and the bed of callus from which it has been lifted, otherwise the nerve may be again caught. When implicated in fractures of the shaft of the humerus below its middle, probably the best incision to expose the nerve is one placed obliquely across the centre of a line drawn from the insertion of the deltoid to the outer condyle of the humerus, when the nerve will be found lying between the brachio-radialis (supinator longus) and the brachialis anticus. If the incision is made too internal, the external cutaneous nerve may be exposed and mistaken for the musculo-spiral.

It is interesting to note that the humerus is more frequently broken by muscular (indirect) violence than perhaps any other bone, with the exception of the patella. This is commonly the outcome of a great effort in throwing, and the shaft snaps just below the deltoid insertion. The contraction of the muscle suddenly arresting the movement of the bone at this spot, the impetus already gained carries the lower end of the bone and the attached forearm forwards, and under the strain induced, the humerus gives way.

*Fracture of the lower end.*—Several fractures may occur in

this region. Of these the two commonest are fracture of the internal epicondyle, and a transverse fracture just above the level of the olecranon fossa.

That part of the inner condyle which is outside the capsule of the joint is styled the epicondyle (or sometimes the epitrochlea). From its prominence it is greatly exposed to injury and is frequently detached by violence. When separated, the flexor muscles arising from it tend to drag it downwards and somewhat forwards. The close proximity of the ulnar nerve to the posterior aspect of the process renders it liable to injury or to be involved in callus.

The transverse fracture is generally the outcome of indirect violence and is only transverse so far as its lateral direction is concerned, for it is oblique from behind downwards and forwards when its antero-posterior axis is examined. Thus the displacement of the fragments is easily explained. The lower end of the upper fragment comes forward and apparently downwards and will even injure the brachial vessels and the median nerve lying in front of it. In some cases it may actually perforate the skin above the crease of the elbow. The lower fragment, together with the bones of the forearm, is carried backwards and upwards by the action of the triceps aided by the pull of the biceps and brachialis anticus. Thus the arm, measured from the acromion process to the external condyle, will be shortened.

**Fractures of the Ulna alone.**—There is only one common form of fracture of the ulna alone, and that is a fracture of the olecranon process, which may be broken either by direct or indirect violence. It is a fairly common lesion owing to the exposed position of the process and the strength of the muscles which act upon it.

By direct violence the fracture may be either near the tip, or some little distance lower down where there is a slight narrowing, at the spot where the bony projection joins the shaft.

In indirect violence—that is, by the action of the triceps—as a rule only the tip of the process, or the olecranon epiphysis is dragged off.



A fractured olecranon always involves the elbow joint. Immediately after the fracture the triceps, acting upon the upper fragment, draws it away from the lower, the extent of separation depending first upon the activity of the muscles, and secondly upon the amount of tearing of the tendino-aponeurotic fibres investing the process laterally. The fragments may be further separated by the dipping in of a fringe-like apron of the stretched and lacerated surrounding aponeurosis. The result of this separation is that in most cases where wiring is not undertaken union occurs only by fibrous tissue.

Fractures by direct violence are often associated with but little separation, and some of these may possibly unite by bony tissue. In the majority of cases of fracture of the olecranon process with marked separation, exposure and wiring of the fragments is indicated.

**Fractures of the Radius alone.**—This fracture is most commonly seen close above the distal extremity of the radius in the form of the well-known Colles's fracture. It is usually produced by indirect violence, as by a fall upon the outstretched palm of the hand, by which there is induced extreme dorsi-flexion of the wrist. This will bring about a nearly transverse fracture of the radius within three-quarters of an inch of its lower articular surface. Immediately upon the break of the bone, the weight of the body continuing to act forces the lower fragment towards the radial side of the forearm. It is further, for the same reason, carried upwards and backwards, and also rotated so that the lower articular surface, instead of looking directly downwards, now looks downwards and somewhat backwards. Thus it happens that the tip of the styloid process of the radius, which normally is at a lower level than that of the ulna, now comes to lie at the same level or even higher. Not infrequently the triangular fibro-cartilage drags off the ulnar styloid process. When impaction occurs, as it frequently does, the posterior portion of the upper fragment is impacted into the anterior portion of the lower fragment.

The unsatisfactory results following upon Colles's fracture are

due to several anatomical causes: first, because the deformity will not have been corrected if impaction has not been reduced; secondly, because if the fragments have been unimpacted muscular action tends to reproduce the deformity unless the fracture is very carefully set; and, thirdly, because there is a possibility of the extensor tendons becoming involved in the callus. Most cases, however, even those in which the deformity has not been entirely removed, obtain free movement if massage and manipulation are assiduously employed. It is well, generally speaking, that passive movements of the wrist and the more distal joints should be carried out during the whole process of treatment, so as to make the possibility of stiffness remote.

Separation of the lower radial epiphysis, occurring before the age of seventeen, may be considered as equivalent to a Colles's fracture in young persons.

**Fractures of both Bones of the Forearm.**—This is most commonly caused by direct violence, and the line of fracture will be transverse and at the same level in both the bones. As a rule it occurs about the middle of the forearm, or a little below this point. When the fracture is situated distal to the insertion of the pronator radii teres, the upper fragment of the radius tends to be drawn into the position which is midway between pronation and supination by the action of the muscle, and the upper end of the lower fragment of the radius is adducted towards the ulna by the pronator quadratus. Owing to this adduction there is always a risk of cross union taking place, and the movements of supination and pronation of the hand lost.

In fractures of the bone above the pronator radii teres, the upper fragment is supinated by the biceps and supinator brevis, and for this reason it is necessary to set the fracture with the elbow flexed and the hand in the position of full supination, which position is more likely to bring the fragments of the radius together into line. On the other hand, in a fracture below the insertion of the pronator radii teres, a position of the hand midway between pronation and supination is more advantageous. The position secures accurate apposition, seeing that there is

moderate pronation of the upper fragment, and at the same time it is one which gives the greatest comfort to the sufferer.

**Fractures of the Metacarpals.**—The metacarpal bones are often broken, either by direct violence, or by indirect force applied at the knuckles. When not impacted, there is a tendency for the two fragments to assume an angular deformity, probably owing to the action of the interossei and the flexors, as well as by the direction of the violence and the weight of the finger.

#### FRACTURES OF THE PELVIS.

The bones constituting the **pelvis** have to be looked upon, from a point of view of fracture, both as a whole and individually. As a whole, the sacrum and the two ossa innominata form a complete ring, in which fracture may occur from indirect violence, the circle giving way at its weakest parts. Thus, when a cart-wheel passes over the pelvis of a person lying in the dorsal position, the bony ring is subjected to pressure from before backwards, and consequently the symphysis will tend to approximate to the sacrum. Hence the weak parts of the bones, namely, the rami of the ischium and of the os pubis, on one or both sides, give way. Then, if the compression continues, the iliac bones will be pressed away from the sacrum, but owing to the strength of the sacro-iliac ligaments, either the ala of the sacrum or that portion of the iliac bone immediately external to the articulation is torn away. This, it will be seen, is an evidence of the elasticity of ligaments in contrast with the brittleness of bone.

On the other hand, should the pressure be upon the pelvis laterally, with the patient lying upon his side, there will be again a tendency for the anterior segment of the ring to give way, and for the two halves of the pelvis to approximate in front, with severe dragging upon the posterior sacro-iliac ligaments, frequently inducing fracture of the lateral mass of the sacrum.

Another fracture of the hip bone by indirect violence is seen when the acetabulum is broken by the head of the femur being forced against it from falls upon the great trochanter, the violence being transmitted through the neck of the thigh-bone.

Direct violence will occasion fracture of the prominent parts of the pelvic bones, the crest of the ilium and the coccyx being the most frequently damaged. Falls, kicks, and blows may all react upon these exposed parts. In the case of the crest of the ilium not much displacement will occur, owing to the surrounding muscles holding the fragments together. When the coccyx, however, is broken, it is apt to be considerably displaced from the lower end of the sacrum.

The pelvic bones enclose important viscera, and when a fracture is present there is a great liability for some of these organs to be injured, either by the fragments themselves or by the force which induces the fracture. The urethra passing beneath the pubic arch is most frequently damaged. Extra-peritoneal portions of the bladder are also exposed to injury, and likewise the termination of the rectum. In some cases urine may be extravasated through the wound of the urinary passage, or gas may pass into the tissues through the laceration of the rectal wall.

The nerves and vessels passing through the greater and lesser sacro-sciatic foramina, because of their close proximity to the line of fracture in the ilium or sacrum, may also be injured, with resulting paralysis or extravasation of blood.

#### FRACTURES OF THE LOWER EXTREMITY.

**Fractures of the Femur.**—*Fractures about the neck of the femur* are much influenced by anatomical conditions. They consist of two main varieties, the intra-capsular and the extra-capsular, dependent upon the method of their production.

The first of these, the intra-capsular, is as a rule due to indirect violence. The neck of the femur is the upper part of a long lever, is usually placed at an angle of about one hundred and twenty-five degrees with the shaft in the adult, and it is questionable whether this obliquity diminishes to any extent towards old age unless there is actual disease of the part. Any sudden force, even if slight, applied to the lower end of the lever,—that is, to the foot—such, for instance, as catching the toes in the edge of a carpet, produces

an unexpected strain upon the bone forming the neck, the muscles not contracting sufficiently quickly to relieve the osseous tissue of the brunt of the violence. The neck, therefore, breaks well within the capsule of the joint.

The amount of separation in these cases is but little, and must necessarily be so on account of the anatomy of the part. In the first place, the amount of violence which has occasioned the fracture may be so slight that the cervical reflection of the capsular ligament remains wholly or partially untornd. This reflection is seen in the form of fairly strong bands passing up from the lower attachment of the capsule along the neck to the margin of the articular surface of the head. Secondly, even if these retinacula are torn through, the amount of separation will still be limited by the untornd true capsule itself, and its strengthening bands. Thus it comes about that the shortening in an intra-capsular fracture of the neck by slight indirect violence hardly, if ever, amounts to more than one inch in an adult.

Often although the violence has been so slight, the constitutional disturbance is great, owing to the injury being generally in an aged patient, and attention may not, therefore, be directed specifically to the seat of fracture. Bruising about the hip is but seldom seen, all the blood effused being within the untornd capsule of the joint. The eversion of the foot, however, should draw attention to the possibility of fracture. This eversion is brought about chiefly by the natural weight of the limb, which is greater externally than internally, and partly by the action of the irritated external rotators.

This fracture seldom, if ever, unites by bony tissue, and this probably from three reasons: first, there is great difficulty in obtaining and maintaining approximation, should the fragments have become separated, owing to the inability to manipulate the upper one; secondly, owing to the limited blood supply of the acetabular fragment, which receives only a small vessel from the obturator along the ligamentum teres; thirdly, owing to the osteoporosis, that is, the thinning of the bony tissue and the fatty degeneration of the medullary substance, found in persons of

advanced years in whom the fracture usually occurs. Occasionally, where the retinacula are untorn, they maintain the fragments in close approximation, and may also be a source of vascular supply to the proximal fragment, and thus bony union may occur.

The second or extra-capsular variety of fracture of the neck of the femur is as a rule due to direct violence, usually of a severe character. In this form the fracture is partly within but chiefly without the capsule of the joint, the line of the fracture being posteriorly outside although close to the intertrochanteric crest and therefore external to the capsular fibres, whilst in front it runs internal to the upper portion of the spiral line to which the capsule is attached.

The fracture is most commonly induced by a fall from a height upon the great trochanter, and immediately upon the solution of continuity the proximal fragment is driven into the distal and becomes impacted, sometimes splitting the great trochanter.

The shortening in these cases is much greater than in the non-impacted intra-capsular fractures, and may amount to as much as three inches in the adult. It is occasioned by the displacement caused by the actual violence that induced the injury. The alteration in the position of the lower fragment will bring the great trochanter above its normal level. This relaxes that portion of the fascia lata which lies between the iliac crest and the trochanter, so that there is here a certain amount of loss of resistance on the injured side.

Much bruising about the hip-bone may be present, owing to the fall rupturing blood vessels and giving rise to extravasation.

The position of the foot, which was one of marked eversion in the case of intra-capsular fractures, is in the impacted extra-capsular variety in the position in which it was when impaction took place, and is most commonly somewhat everted.

The union of this variety of fracture is always by bony tissue owing to the impaction, which occasions the interlocking of the fragments and maintains the vascular supply.

*Fractures of the shaft.*—When fracture of the shaft occurs

immediately below the lesser trochanter, a characteristic displacement of the proximal fragment is induced by the action of the ilio-psoas. The fragment is flexed, so that its lower end tends to project forwards, sometimes against the vessels, and in thin persons it may almost appear as if it intended to perforate the integument. Associated with the flexion will be found some amount of external rotation and abduction, caused by the pull of the obdurator muscles and the quadratus femoris. The distal fragment at the same time will be drawn somewhat upwards, backwards and towards the middle line, chiefly by the action of the hamstrings aided by the quadriceps and the adductors, thus inducing an angular deformity in two directions, that is, with the apex of the angle both outwards and forwards.

It is obvious from this description of the deformity that there will be considerable difficulty in getting the two fragments into a proper line of apposition, unless the knee is raised by flexion, and traction is made upon the distal fragment to overcome its upward displacement.

*Fractures of the middle of the shaft* by indirect violence as a rule are oblique, and the displacement may be considerable. Again the muscles passing from the pelvis to the femur, such as the ilio-psoas, pectineus, adductor brevis and adductor longus, tend to flex the upper fragment and externally rotate it, but in contradistinction to the abduction of the proximal fragment as seen in the previous fracture, there is a tendency in fractures of the middle of the shaft for the lower end of the proximal fragment to be drawn somewhat inwards, that is, adducted. The lower fragment is drawn upwards, backwards, and inwards, chiefly by the hamstrings and the great adductor, and its upper end may tend to push outwards the lower end of the upper fragment, thus counteracting the tendency to adduction just mentioned. In addition to the upward, backward and inward displacement, the lower fragment is markedly rotated outwards by the weight of the limb. There may also be, from powerful action of the adductor magnus, some adduction of the lower fragment and the leg, with the result that an angular deformity

with the apex outwards may occur at the seat of the fracture. One of the nutrient vessels of the femur may be torn, and the amount of extravasation of blood is often very great.

In the treatment of such fractures the shortening may be overcome by the application of an extension apparatus, which, whilst acting by a distinct drag upon the lower fragment in a distal direction, will also tire out the muscles and so bring about their relaxation. The outward rotation is easy to correct, but it is difficult to prevent its recurrence owing to the weight of the limb.

Transverse fracture of the middle of the shaft of the femur is not uncommon in children, particularly in those who are the subjects of rickets, but in these fractures the displacement as a rule is but slight.

*Fractures close above the condyles* are not infrequently caused by falls upon the front of the lower end of the thigh, in which case an oblique fracture occurs, running from behind downwards and forwards. The lower end of the upper fragment in such a case, being sharp and at the same time forced forwards and downwards, may penetrate the lower end of the quadriceps and skin rendering the fracture an open one. The upper end of the lower fragment at the same time may be somewhat flexed by the gastrocnemius and plantaris and carried upwards by the hamstrings, so as possibly to press upon or even seriously damage the popliteal vessels. The same effect upon the vessels may occur in cases of simple transverse fracture above the condyles; hence it is always wise to feel for the pulse in the dorsalis pedis and posterior tibial near the ankle.

The flexion of the distal fragment is difficult to overcome, so it is usual to put the limb up on a double inclined plane or an anterior angular wire splint, and thus to obtain flexion of the proximal fragment and bring it into line with the distal. The objection to this method of treatment is chiefly to be found in the fact that whilst pressure upon the vessels by the lower fragment is relieved, the angle of the double plane may itself cause pressure on them from behind and at a slightly lower level.

In separation of the lower epiphysis of the femur the line of



fracture is as a rule immediately distal to the attachment of the heads of the gastrocnemius. The consequence of this is that the lower end of the upper fragment may be drawn backwards and downwards, and press upon the vessels, whilst the epiphysis itself may be dislocated forwards.

**Fractures of the Patella.**—The patella is covered in front by the skin, superficial and deep fasciæ, and a tendinous expansion from the quadriceps. Posteriorly it is coated with articular cartilage save at its tip, where is the attachment of the ligamentum patellæ. Hence it follows that all fractures of the patella which traverse the whole thickness of the bone, except its lowest part, necessarily lacerate the aponeurosis anteriorly and implicate the joint behind.

The position of the patella, placed as it is between the insertion of the quadriceps above and the patellar ligament below, renders it liable to a very great longitudinal strain particularly in such efforts as to save oneself falling after missing a step in ascending the stairs. Hence this bone is more frequently broken by that form of indirect violence known as muscular than any other in the body. Its exposed position on the summit of the knee also renders it liable to direct violence, which may also lead to its fracture.

In the lesion by muscular action it is somewhat doubtful whether the transverse fracture which occurs is due to leverage, the patella resting by its posterior surface on the trochlear surface of the femur as on a pivot. If the movement of the knee in flexion and extension is accurately observed, it will be found that at no time is any part of the patella really unsupported. It glides upon, but always remains co-apted to, the articular surface of the condyles of the femur. Hence it follows that the common transverse fracture is the outcome of an almost pure longitudinal strain, which in the great majority of cases breaks the bone, but may tear the quadriceps tendon at or near its insertion, may lacerate the ligamentum patellæ, or may even drag off the tubercle of the tibia. In most instances where bone, tendon or ligament has to bear a strain, the breaking force is least readily borne by the bone,

which, being brittle rather than extensile, snaps. This peculiarity is seen not only in the case of the patella and the olecranon, but also in Pott's fracture.

In the transverse fracture, the upper fragment as a rule is larger than the lower. Frequently the line of laceration of the tendinous expansion over the front of the bone does not correspond to the line of fracture in the bone itself, but is at a different level. Further, this aponeurosis is stretched before it is torn, so that its lacerated edge may be very ragged, and the ribbon-like portions may easily find their way between the fragments of bone, and possibly bring about a want of bony union.

A transverse fracture may also occur by direct violence, but stellate or comminuted, and vertical fractures are more commonly the outcome of this form of violence.

The amount of separation in a transverse fracture of the patella varies greatly, and is dependent upon several factors. The first of these is the action of the quadriceps muscle, which by its insertion into the upper fragment tends to draw this away from the lower; but the distance that it can be dragged upwards is regulated by the amount of tearing of the lateral aponeurosis associated with the insertions of the vasti. Secondly, two other minor factors come into play, firstly whether or not the joint is passively or actively flexed after the injury has occurred, and secondly the amount of effusion of blood and synovial fluid which occurs into the joint.

Hence it is obvious that it will not be politic to attempt to bring the fragments into apposition until the almost spasmodic contraction of the quadriceps has passed off and some of the effusion into the joint has been absorbed. Thus it is incorrect to endeavour by mechanical, or operative means, to approximate the fragments until three or four days at least have elapsed since the lesion occurred. There are two other reasons for delay in the operative treatment of fractured patella, namely, to allow time for the torn vessels to become closed, and to give ample opportunity for obtaining asepsis of the operation area.

**Fractures of the Bones of the Leg.**—Fractures of the bones of

the leg occur very frequently. Both bones may be broken by direct violence, when the fracture will occur in each at the same level. The fracture is then usually transverse, and the amount of displacement but little.

*Fracture of the tibia alone* by indirect violence is rare on account of the fact that the fibula is a slender light bone, and if the weight of the body is thrown upon it, after a fracture of the tibia, it also snaps, breaking as a rule at its weakest point, in its upper fourth.

On the other hand *fracture of the fibula alone* is extremely common, and generally due to direct violence, the fracture taking place at the spot to which the violence is applied. The fibula in its natural condition, when pressed by the fingers of one hand applied above and the fingers of the other hand applied below, bows towards the tibia, a definite spring being experienced. In order to obtain this practical demonstration of the continuity of the fibula, it is necessary to remember that the fingers must rest definitely upon the bone, and that the fibula is placed in a position which is distinctly posterior to that which it is sometimes thought to occupy. When the bone is broken the spring is lost. There is little if any displacement of the fragments, owing to the unbroken tibia acting as a splint. There may, however, be an extensive extravasation of blood, due to laceration of the peroneal artery or one of its branches, which lie in close apposition to the bone.

That part of the external malleolus which lies below and posterior to the facet for the astragalus is weakened by the presence of the groove which lodges the middle and posterior bands of the external lateral ligament of the ankle. Consequently this part of the fibula may be detached by direct violence or sudden twists of the foot which drag upon the ligamentous fasciculi.

*Fracture of the tibia and fibula with outward partial dislocation of the ankle (Pott's fracture).*—In order to understand the production of this lesion, which is in reality a fracture-dislocation, it should be remembered that it is nearly always due to a strong

abduction of the foot by slipping off from a higher on to a lower level, and thus on to the inner side of the sole of the foot.

This movement throws strain upon the internal lateral ligament of the ankle joint, and through it upon the internal malleolus to which the apex of the deltoid ligament is attached. As a rule, as has already been seen in the case of the patella, the bone snaps before the ligament tears, and thus the internal malleolus is dragged off. Immediately, the weight of the body is thrown through the astragalus against the external malleolus. The fibula, however, is held to the tibia by the strong inferior tibio-fibular (interosseous) ligament, and therefore a strain is thrown upon the lower part of the fibular shaft close above the ligament. It is here that the fibula then breaks, and the upper end of the lower fragment is tilted towards the tibia, simultaneously with a marked deflection of the foot outwards, and the foot is also usually carried somewhat backwards and the heel slightly raised by the action of the calf muscles.

Most commonly a dimple in the skin may be seen in the early stages opposite the site of the fibular fracture, and a marked stretching of the skin over the fractured internal malleolus, at which latter spot the skin is likely to give way, and render the fracture-dislocation an open one.

In reducing the fracture, difficulty may be met with, firstly in overcoming the outward displacement and eversion, probably owing to interlocking of the fibular fragments preventing the restoration of the astragalus to within the malleolar arch; secondly, in correcting the backward and upward displacement, owing to the contraction of the muscles inserted in the tendo Achillis. This pull may to a great extent be overcome by flexing the leg upon the thigh and the thigh upon the abdomen, or altogether by division of the tendon, or by placing the patient under the influence of an anæsthetic.

*Dupuytren's fracture* is present when, in addition to the conditions found in Pott's fracture, the portion of the tibia to which the lower tibio-fibular ligament is attached is dragged off, or



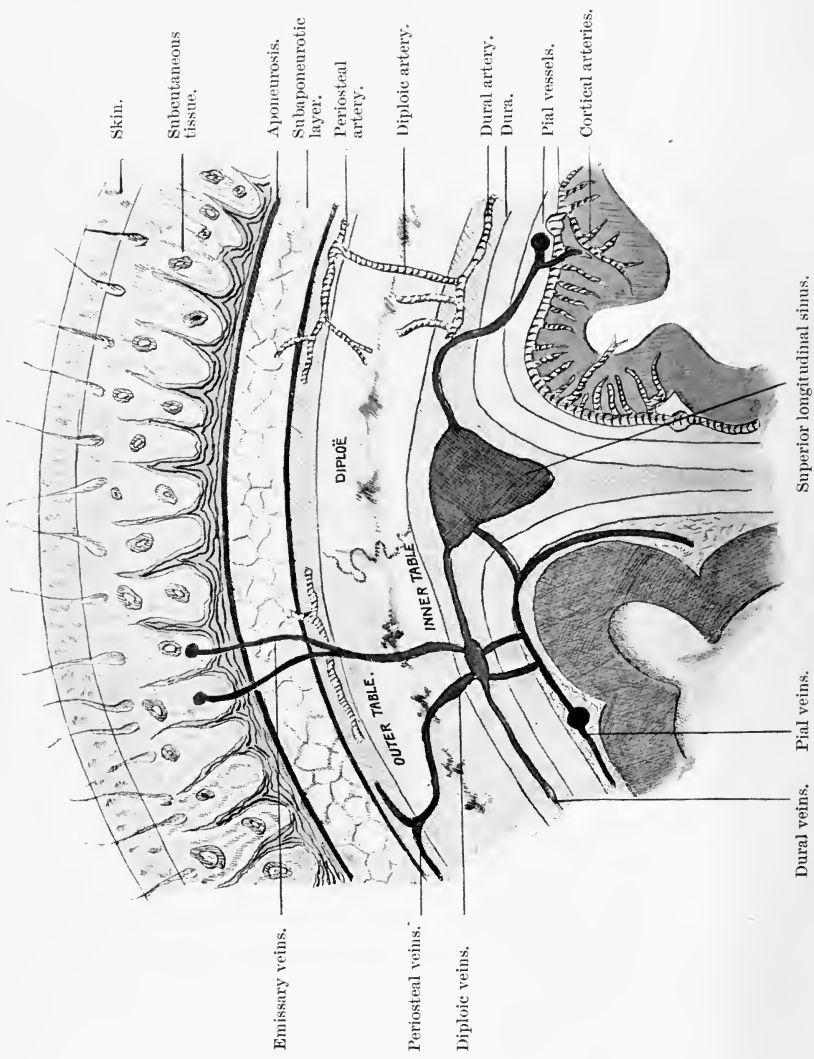


FIG. 4.—Coronal section of scalp, skull, and cortex of brain with its membranes, to illustrate the transmission of infection from the scalp to the interior of the skull. (After Eisendrath, modified from Herman.)

this ligament itself is lacerated, the portion of the tibia remaining intact.

Outward dislocation in these cases is apt to be much aggravated, and the foot is considerably drawn upwards on the outer side of the leg.

**Fractures of the Os Calcis.**—In a vertical fracture of the os calcis near its posterior extremity, the hinder fragment is pulled upwards by the action of the muscles attached by the tendo Achillis.

#### FRACTURES OF THE SKULL.

These consist of (1) Fractures of the cranium. (2) Fractures of the bones of the face.

Injuries about the scalp, with which open fractures of the vault of the skull are practically always associated, are of considerable importance, and it may be as well to allude to them at this point.

**Wounds of the Scalp.**—There are several anatomical facts concerning wounds of the scalp which are of the greatest importance. (See Fig. 4.)

The tissues forming the scalp are the skin, thickly beset with hairs, the subcutaneous tissue, the aponeurosis of the occipitofrontalis muscle, the underlying loose areolar tissue and the pericranium.

Any wound which does not involve the aponeurosis is not serious, except on account of hæmorrhage and the liability of superficial sepsis. The arteries lie in the dense subcutaneous tissue, and when divided this tends to prevent them from contracting and retracting. Hence the severe bleeding which is likely to result, and the difficulty of controlling it either by forcipressure or by ligature. The underlying bone, however, will enable pressure to be applied by the dressing and bandage, and this will usually be sufficient to control the hæmorrhage.

When a scalp wound involves the aponeurosis, blood, or inflammatory fluids such as pus, are liable to spread over the vault of the skull, being limited only by the attachments of

the aponeurosis, which are as follows :—the supra-orbital ridges in front, the zygomatic arches laterally, and the superior curved lines behind.

The arteries found in the scalp are, from the mid-line in front to the mid-line behind, the frontal, the supra-orbital, the anterior and posterior branches of the superficial temporal, the posterior auricular and the occipital. They all run in a direction which is more or less upward, and they freely anastomose with one another. Incisions to free a collection of pus beneath the aponeurosis will have to be planned so as to be vertical, or nearly so, and thus to lie between the scalp arteries.

In an extensive scalp wound a flap may be raised owing to the great looseness of the tissue beneath the aponeurosis. There is, however, little likelihood of the flap sloughing, because it carries the blood-vessels with it. In the same way, even a large flap turned downwards during various intracranial operations is sure of a good blood supply.

Scalp wounds caused by even blunt instruments may be very clean cut, because the tissues are stretched as it were over the firm bone beneath when the violence is applied.

**Abscess of the Scalp.**—A collection of pus above the aponeurosis of the scalp is like a superficial abscess elsewhere, and may be dealt with in the same manner, due attention being paid to the arteries.

An abscess beneath the aponeurosis is much more difficult to treat, and requires many incisions owing to its extent. Further, such abscesses may be long in healing, partly due to imperfect drainage and partly to want of rest owing to the activity of the occipito-frontalis muscle.

The most dependent points for evacuation of the pus vary with the posture of the patient, but the posterior occipital region and the lateral temporal regions are most likely to be the positions that should be selected. Incisions in the frontal region leave very conspicuous scars, in addition to being unsuitable for efficient drainage.

**Hæmatoma of the Scalp.**—Every blow upon the head is liable



to be followed by extravasation of blood, and this may collect in the subcutaneous tissues and form the ordinary bruise so frequently seen.

If the hæmorrhage be in a deeper plane, there is a tendency for the blood to be diffused in the loose areolar tissue beneath the aponeurosis, and a considerable quantity of blood may be poured out, and the scalp may thus be raised so as to rest upon a veritable blood cushion. Provided that sepsis is excluded, all the blood will be eventually absorbed.

In other cases the blood may be poured out between the pericranium and the bone beneath. It is then generally spoken of as a cephalhæmatoma, and is often seen soon after birth as a result of injury during the passage of the child's head along the maternal channel. The pericranium is but loosely attached to the bone except where it is continuous with the tissues at the sutures. Hence it follows that in cephalhæmatoma there may be a complete concealment of a single bone, which has led to the assumption that the bone—most usually the parietal—is absent.

The pericranium is more firmly adherent over the temporal bone than elsewhere, and it is rare to get a hæmatoma beneath it in this region.

#### FRACTURES OF THE CRANIUM.

These may consist of fractures of the vault or fractures of the base, or of a fracture which runs from one into the other.

**Fractures of the Vault.**—The bones of the vault of the skull for the most part are flat bones composed of two tables, an outer and an inner, of compact tissue, and an intervening layer—the diploë—of cancellous tissue.

Four main sutures between these bones may be recognised in the full-grown subject, any one of which may have to be distinguished from a fracture. Their exact position is therefore a matter of some import. (See Fig. 5, p. 104.)

The coronal, or fronto-parietal, has its summit at the bregma, a point in the median antero-posterior plane of the skull where

a line, in the adult, drawn vertically upwards from the external auditory meatus cuts it. From the bregma a line carried downwards and forwards to a spot called the pterion represents the coronal suture. The pterion is placed on a line drawn backwards from the external angular process of the frontal, parallel to the zygoma, at a spot where a second line drawn upwards from the middle of the zygoma cuts the first.

The sagittal suture runs backwards in the middle line from the bregma to the lambda, the latter being approximately placed one-third the distance along the line leading from the bregma to the inion, or external occipital protuberance.

From the lambda down to the base of the mastoid process will be found running the lamboid suture. The remaining suture is the squamo-parietal, which forms a curved line with its convexity upwards, starting from the level of the centre of the zygoma and terminating at the base of the mastoid, the highest point of the curve reaching about one-third the distance along the line passing from the external auditory meatus to the bregma.

Fractures of the vault occur in three forms—the undepressed, the depressed, and the elevated.

In the *undepressed*, there may be a linear or stellate fissure involving the whole thickness of the bone. The presence of either of these as a closed or simple fracture cannot be absolutely diagnosed, but only surmised. An open or compound linear fracture has to be diagnosed from either a mere slit in the pericranium without a break in the bone, or a suture exposed by the scalp wound. A fracture may be diagnosed from a mere slit in the pericranium chiefly by the fact that blood will well up through the fissure from the diploë. From a suture a fracture may be diagnosed by the same fact, and also that most of the sutures exhibit a much greater irregularity because of their serration.

Particular care must be taken in scalp wounds of the temporal region not to mistake the margin of the squamous portion of the temporal bone, overlapping the lower border of the parietal, for a fracture in this region.

CRANIAL LANDMARKS.

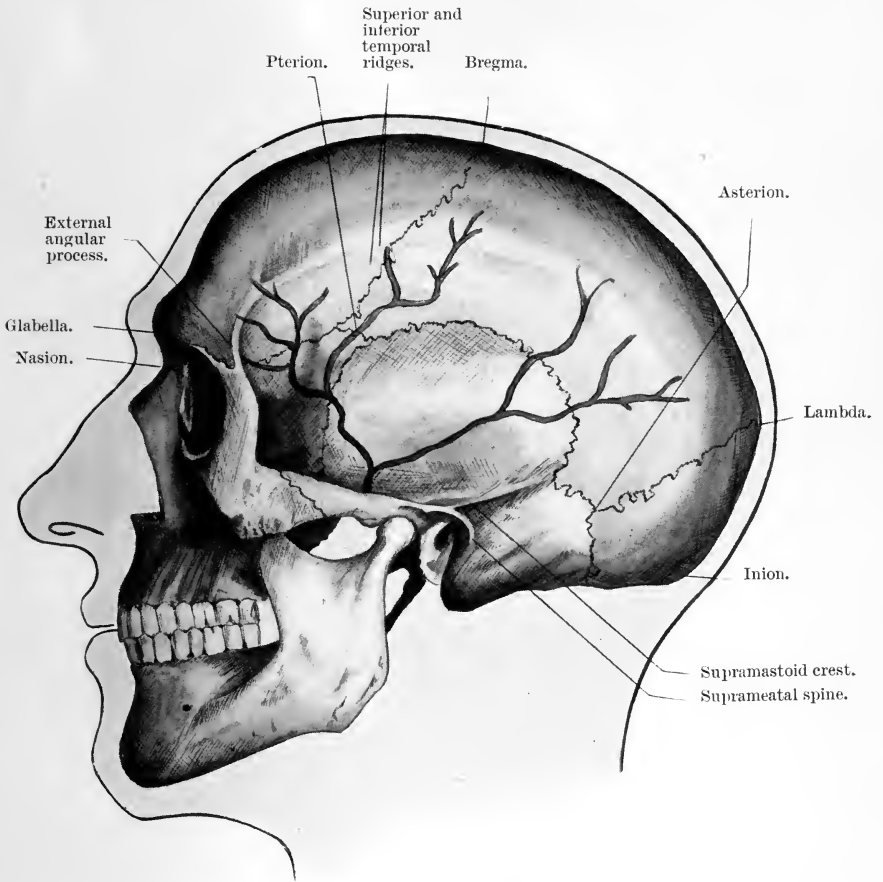


FIG. 5.—View of the left side of the skull, showing landmarks. (After Edward Taylor.)





MIDDLE MENINGEAL ARTERY.

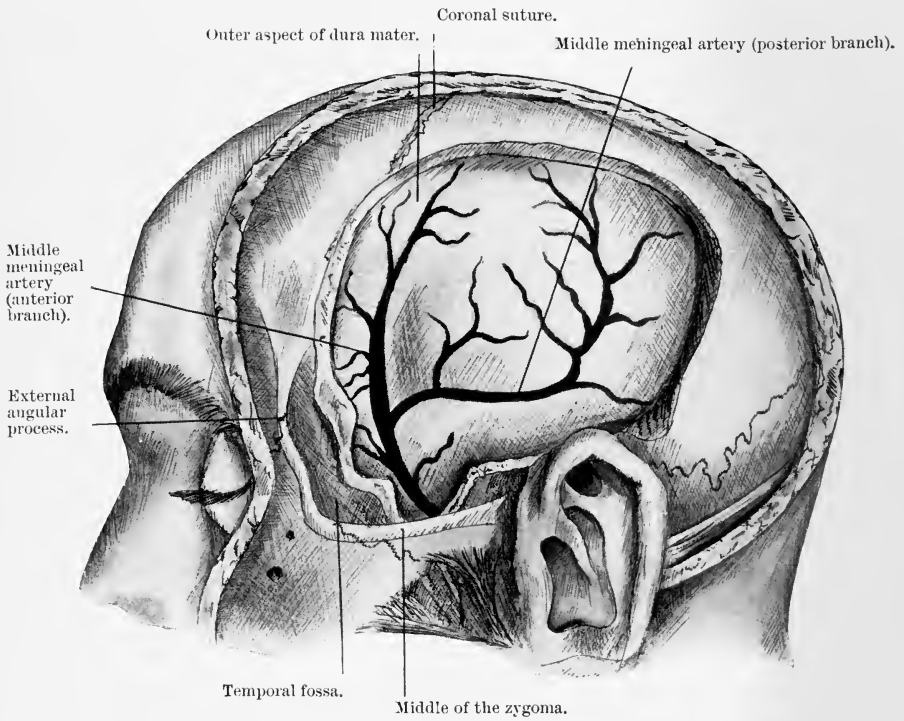


FIG. 6.—View of the left side of skull showing the middle meningeal artery. (After Zuckerkandl.)

Compression of the brain, due to hæmorrhage occurring within the cranium but external to the dura mater, is most commonly associated with a linear fracture across the anterior inferior angle of the parietal bone. At this spot the inner surface of the bone is grooved by the anterior or larger branch of the middle meningeal artery, the main trunk of which having entered the skull through the foramen spinosum soon divides into two branches. (Fig. 6.) It is rare to get hæmorrhage from the trunk itself. Occasionally the anterior branch lies in a complete canal rather than in a groove. When a fracture occurs, the artery lying upon or rather somewhat in the fibrous tissue forming the dura mater may be torn. Blood is then effused between the bone and the membrane, depressing the latter inwards so as to compress the cerebral substance. It will thus be seen that the blood poured out is sub-cranial but extra-dural, and it is better not to speak of it as a meningeal hæmorrhage. The extent to which the blood strips up the dura mater from the inner surface of the bone is limited below by the firm attachment of the membrane to the base of the skull, but above only by the want of force with which the blood is thrown out. The amount of blood, however, which collects immediately internal to the site of the fracture is quite sufficient to cause such compression as in most cases to bring about hemiplegia and later coma.

When the bleeding point has been exposed by the removal of the overlying bone at the pterion, it is as a rule impossible to pick up the artery with a pair of pressure forceps, because of its being somewhat embedded in the tissue forming the dura mater. It is therefore necessary to carry a ligature by means of a curved needle into the membrane and beneath the artery in order to secure it.

When the vessel lies in a complete bony canal, a further difficulty will be encountered in arresting the hæmorrhage, and it will then become necessary either to remove the bone carefully from around the artery, or to plug the canal by means of a spicule of bone or some material such as aseptic wax.

*Depressed fractures of the skull* are as a rule the outcome of

direct violence. The bone which is depressed may be the whole thickness of the vault—that is, the outer table, the diploë and the inner table—and this condition is the commonest. On the other hand the external table alone may be driven in without the internal table being in any way depressed. This occurs most usually and typically where the two tables are separated from one another, not by diploë, but by an air space, as for instance in the region of the frontal sinus.

It is doubtful whether depression of the internal table alone ever occurs.

In quite young subjects, owing to the elasticity of the bones entering into the formation of the vault, it is possible for depression to occur without an actual fracture, and in some cases the depressed bone may become spontaneously restored to its normal position.

In depression of both tables it is usual for the internal to be depressed over a wider area than the external, which is due to the radiation of the force from the point at which it is applied, and the curvature of the bone entering into the formation of the skull.

In the diagnosis it is important to distinguish depressed fractures from natural depressions about the skull. Perhaps the most likely spot at which a mistake may arise is in the anterior part of the temporal region, where the upper border of the zygoma, the external angular process of the frontal bone and the commencement of the temporal ridge together form a raised margin, which, if associated with the history of a blow and the presence of a hæmatoma of the scalp, very closely simulates a depressed fracture. A conclusive diagnosis however can easily be made by comparing the sound with the injured side, the former exhibiting the pseudo-depression even more markedly than the latter. Other natural depressions sometimes mistaken for depressed fractures are found about the occipital region in connection with the spaces between the nuchal lines.

*An elevated fracture* is usually seen at the aperture of exit of a bullet, and is therefore commonly an open fracture, the scalp as well as the bone having been traversed by the missile. In this



case the external table will be elevated over a more extensive area than the internal.

**Fractures of the Base.**—Fractures of the base of the skull may be the outcome of direct or indirect violence. A linear fracture of the vault may run down into the base.

Almost every fracture of the base is open, through the mucous membrane covering a large proportion of the bones entering into its formation. Thus a fracture of the anterior fossa may communicate with the external air through the mucous membrane of the nose; a fracture running across the petrous portion of the temporal bone through the mucous membrane lining the tympanum; and a fracture of the posterior fossa through the mucous membrane lining the roof of the pharynx. It will thus be seen that in many of these fractures the meninges of the brain may be put into direct communication with air that may be septic, and meningitis is therefore apt to follow these fractures.

Further, the same fact of laceration of the soft tissues covering the bones leads to the escape of blood from the nostrils, into the tissues of the orbit, through the ears, down the pharynx, and into the muscles of the back of the neck. Likewise, cerebro-spinal fluid may pass through the same channels. Again, the exit of the cranial nerves through the base of the skull explains the great frequency with which they may be involved when fractures in this region occur. Thus it is, that the three cardinal symptoms or signs of a fracture of the base of the skull, that is to say the loss of blood, the loss of cerebro-fluid and the primary or secondary injury to the cranial nerves, are dependent upon strictly anatomical reasons.

Fractures involving the anterior fossa may give rise to hæmorrhage from the nose when the cribriform plate of the ethmoid is damaged, or the passage of blood into the tissues of the orbit, leading to subconjunctival hæmorrhage or even proptosis, when the orbital plate of the frontal bone is fissured. The nerves which may be involved in this fossa are the olfactory, interference with which it is difficult to demonstrate, and the optic, damage to which is apparent from the blindness which occurs.

When the injury to the optic nerve is primary, that is to say when it is torn coincidentally with the fracture of the bone, the loss of vision is usually permanent, but when the amblyopia is due to pressure on the nerve filaments by inflammatory exudation or callus, it may be only transitory.

In the middle fossa the following cranial nerves may be involved—the third, the fourth, the fifth and the sixth. Interference with the third, fourth and sixth gives rise to squint, and that of the fifth causes failure of movement of the muscles of mastication and loss of sensation in the skin of the whole of the face, except that area supplied by the great auricular over the parotid gland.

When the posterior fossa is involved, particularly where the fracture runs across the petrous portion of the temporal bone and involves the tympanic cavity, blood and cerebro-spinal fluid escape from the external auditory meatus. The nerves that may be injured are the seventh, the eighth, the ninth, the tenth, the eleventh and the twelfth.

Facial paralysis as the outcome of primary injury to the seventh nerve whilst it is passing through the aqueduct is common in fractures involving the petrous bone. Loss of function of the nerve may again be due to secondary inflammation.

Injury to the eighth nerve may induce loss of hearing.

In the case of the nerves passing through the posterior lacerated foramen the damage so usually implicates the tenth that sudden death may result.

The hypoglossal nerve, when torn or pressed upon by fractures running through the anterior condyloid foramen, is deprived of function, and impairment of action, or paralysis, of the muscles of the tongue ensues.

#### FRACTURES OF RIBS.

Ribs are exposed to violence in one of two ways, either by a direct blow, when the bone will break at the spot to which the violence is applied, or by pressure from before backwards—

as in buffer accidents or squeezing in a crowd—when the rib will break at the point of maximum strain, as a rule slightly outside the angle.

In fracture by direct violence there is a tendency for the broken ends to be driven inwards upon the underlying viscera, particularly the lungs, whereas in fracture by indirect violence the broken ends tend rather to be forced outwards, away from the enclosed organs. Vertical displacement of the fragments is prevented by the attachment of the intercostal muscles and membranes.

The adhesion of the parietal pleura to the inner surface of the rib practically necessitates its laceration in every case of fracture, and the passage of the upper division of the intercostal artery along the subcostal groove indicates how rupture of this vessel may occur. The blood from the artery will pass into the tissues of the intercostal space and through the tear in the parietal pleura into the pleural cavity, and may be so much in amount as to give evidence of hæmothorax.

The position of the intercostal nerve explains the reason why pain in a fractured rib may be present at the site of fracture and also referred to the extremity of the nerve.

The close proximity of the lung, covered by the visceral pleura, to the chest wall readily shows how easy it is for this viscus to be injured. It may be bruised by the direct violence which occasions the fracture, or lacerated by the broken ends driven by that force into its superficial parts. The wound of the lung causes hæmorrhage, partly into the lung substance and partly into the pleural cavity. The hæmorrhage into the alveoli and smaller bronchioles of the lung may be evidenced by hæmoptysis, but the expectoration of blood may be delayed for some hours, on account of the length of the respiratory passages through which it has to travel.

During inspiration air may be drawn from the lacerated lung tissue into the pleural cavity, and during expiration this air may pass from the pleural cavity, across the wound in the parietal pleura and between the fractured ends of the bone and torn

muscles into the subcutaneous tissues, there to produce "surgical emphysema." The air thus forced into the tissues may travel through the whole of the subcutaneous tissues of the body, except where the skin is tightly bound down, as for instance in the regions of the palms of the hands, the soles of the feet, the auricles, and to a certain extent the scalp.

The ribs which are most commonly fractured are those of the middle of the series, the fifth, sixth, seventh, eighth, and ninth. Those in the upper part of the thorax are more protected, the first being practically covered by the clavicle, and therefore fracture of the first rib seldom occurs unless accompanied by a fracture of the collar bone. The lower ribs, the eleventh and twelfth, having no attachment through their costal cartilages with the costal cartilages of the other ribs, easily slip out of the way when violence is applied, and thus generally escape a solution of their continuity.

Fracture of any of the ribs below the sixth may involve injury to the diaphragm and the subjacent liver on the right and of the spleen and possibly the stomach on the left, whilst fracture of the tenth, eleventh, and twelfth may be accompanied by damage to the kidneys.

In the union of a fractured rib much external callus is usually in evidence, owing to the fact that it is well-nigh impossible to get complete immobility of the fractured ends, on account of their movement during respiration.

#### FRACTURES OF THE STERNUM.

Fracture of the sternum is a rare fracture, and usually produced by direct violence. It is in most cases associated with such grave injury to the thoracic viscera, particularly the heart, that the sufferer but rarely survives. The sternal fragments are driven backwards, and come into violent contact with the right side of the heart or the great vessels which lie immediately behind the bone. The shock is so severe that, even if no vessel is torn, death nearly always ensues.

## FRACTURES OF THE SPINE.

*Fractures* of the vertebræ may occur without dislocation, although it is rare for a dislocation to occur without an accompanying fracture, owing to the intricate manner in which vertebræ are articulated together. In the dorsal region particularly the spinous processes may be broken off, or the laminae driven towards the spinal cord without displacement of the rest of the bone. This lesion occurs as a rule from direct violence. The enclosed spinal cord is liable to injury either by pressure from the fragments of bone themselves, or from the extravasated blood, which may be found either without or within the thecal sheath.

*Fracture-dislocation* occurs most usually at the more flexible parts of the spine. Hence it is commonly seen at the lower cervical and dorsi-lumbar regions. As a rule indirect violence induces the lesion, the fracture often involving both the centrum and the neural arch as the result of the sudden and forcible wrenching of one centrum from another. The spinal cord lying in the vertebral canal will almost certainly be acutely bent and pressed upon by the dislocated bones. In the case of dislocation of the body of the upper vertebra forwards and downwards, it is the posterior superior edge of the lower vertebra which causes the mischief to the cord. On the other hand, when the body of the upper vertebra is dislocated backwards, often with crushing and fracture of the neural arches involved, it is the lower and posterior edge of this upper centrum which presses upon the spinal cord.

Most of the symptoms are referable to the damage done to the cord itself. As a rule injury to the cord is not recovered from in that there is no power of regeneration, at any rate so far as the grey matter is concerned. It therefore follows that even if death does not take place, restoration of function is very unlikely.

Owing to the fact that the area over which the cord is involved extends some little distance above the actual site of the fracture-dislocation, it will be evident that the peripheral signs will not

accurately correspond to the level at which the dislocation has occurred.

In fracture-dislocation between the fifth and sixth cervical vertebræ, with the usual resulting pressure upon or laceration of the spinal cord, all the parts supplied distally will probably be completely paralysed. There is therefore motor paralysis of every voluntary muscle and anæsthesia of skin and mucous membrane. The patient's respiration is entirely abdominal, that is, it is carried on by the action of the diaphragm alone, this muscle receiving its innervation through the phrenic arising higher than the lesion. Owing to the irritation of the cord close above the actual site of injury, a zone of hyperæsthesia is generally present in the skin supplied by the fifth and sixth cervical nerves, which is roughly over the outer side of the arm and forearm. The centres for defæcation and micturition (and also for parturition) will remain intact in the lumbar segment of the cord. Hence it follows that the rectum will be emptied, though unconsciously, and the bladder will fill and then involuntarily evacuate itself or overflow. Priapism may be present.

Fracture-dislocation involving the third, fourth or fifth cervical vertebra will almost certainly involve the origin of the phrenic nerve from the third, fourth, and fifth cervical roots, and will lead to very rapid death by failure of the respiratory movements, the diaphragm as well as the intercostals being paralysed.

Fracture-dislocation of the first two cervical vertebræ is usually fatal at once owing to the proximity and compression of the vital centres in the medulla. Rarely the displacement is slight and death is not immediate. In such cases incautious voluntary movement may cause further dislocation with fatal consequences.

In the dorsi-lumbar region, fracture-dislocation involves the nerve supply of the lower extremities, causing painful paraplegia, with muscular wasting.

Since the spinal cord terminates at the lower end of the first lumbar vertebra in the adult, fractures below this level can only involve the long roots of the cauda equina and these may escape compression since they occupy a smaller space in the vertebral canal.

## CHAPTER VIII.

### DISLOCATIONS.

A **DISLOCATION** is the partial or complete separation of bony surfaces normally in contact at an articulation.

From this definition it will be apparent that the anatomical relationships of a joint are of importance in determining the frequency, the extent and the treatment of a dislocation. For instance, the imperfect co-aptation of the head of the humerus and the glenoid cavity of the scapula explains to some extent the frequency with which dislocation occurs at the shoulder. On the other hand the great strength of the crucial ligaments of the knee is a reason for the fact that a dislocation at this joint is rarely other than a partial one. And again, the intimate relationship of the bones of the elbow means less likelihood of dislocation and more difficulty in reduction.

In most dislocations there is more or less laceration of the capsule and ligaments of the joint, with but little tearing of the surrounding muscles. Thus the amount of blood extravasated is not as a rule so great as it is in a fracture occurring in the same region. Blood-stained synovial fluid, however, is often secreted so abundantly as to cause considerable swelling of the part.

### SPECIAL DISLOCATIONS.

#### Upper Extremity.

**Dislocations at the Sterno-clavicular Joint.**—This articulation is one of great strength, even in spite of the want of close co-aptation of the articular surfaces, and is the only one connecting the upper extremity with the trunk. The posterior sterno-clavicular ligament is stronger than the anterior. The costo-clavicular ligament holds the clavicle very firmly down to

the cartilage of the first rib, which costal cartilage lies immediately below the sternal end of the clavicle. Because of this relationship, it will be readily understood that dislocation can only take place in one of three directions—forwards, backwards and upwards. In all reduction may be easy, but retention in place difficult, owing to the slight adaptability of the joint surfaces, the laceration of ligaments and the weight of the upper extremity.

Forward dislocation is the most frequent, being permitted by the weakness of the anterior ligament, which is always torn, together with part of the costo-clavicular or rhomboid ligament.

Backward dislocation comes next. In this there is a greater laceration of the capsule and usually a complete tearing away of the rhomboid ligament. The sternal end of the clavicle slips backward and lies behind the manubrium sterni and the origins of the sterno-hyoid and sterno-thyroid muscles, and by posterior pressure on the left side upon the œsophagus produces dysphagia, by pushing the trachea over to the opposite side induces dyspnoea, by direct pressure on the subclavian artery diminution or stoppage of the radial pulse, and upon the innominate vein much congestion of the parts from which this vessel drains the blood.

Upward dislocation is rare, owing to the stability of the rhomboid and the inter-clavicular ligaments, and of the inter-articular fibro-cartilage. When it occurs all these three fibrous structures are usually ruptured.

Downward dislocation can only take place simultaneously with a separation of the first costal cartilage.

**Dislocations at the Acromio-clavicular Joint.**—The joint depends for its strength chiefly upon the coraco-clavicular ligaments. The acromial end of the clavicle may be dislocated upwards upon the acromion process, or downwards beneath it. The former is the more usual owing to the plane of the joint surface, which is from above downwards and inwards. In the upward displacement there is a laceration of the slender capsule, and more or less of the coraco-clavicular ligaments. Reduction may be easy, but retention is difficult.

**Dislocations at the Shoulder-Joint.**—The shoulder-joint is the



most freely movable joint in the body. The head of the humerus is much larger than the glenoid fossa with which it articulates, the capsule is loose and, below and internally, unsupported by muscles. The joint depends chiefly for its stability upon (1) the long tendon of the biceps, which, arising from the supra-glenoid tubercle, passes through the joint above the head of the humerus, a little internal to its middle line, and leaves through an opening in the capsule beneath the transverse humeral ligament, to pass vertically downwards in the bicipital groove: (2) certain muscles, notably the subscapularis, supraspinatus, infraspinatus and teres minor; these muscles are often termed capsular muscles: (3) the coraco-acromial arch, consisting of the coracoid process, the acromion process, and the coraco-acromial ligament thrown between the two: (4) atmospheric pressure. Unless distended with fluid, there is no true cavity in the joint, and its interior practically constitutes a vacuum, so that if the capsule is perforated and air permitted to enter, the laxity of the capsule will allow the articular surfaces to become very distinctly separated.

It will thus be seen that, although there are these four powerful factors maintaining the stability of the joint, they are to a great extent counteracted by the want of co-aptation between the head of the humerus and the glenoid fossa, together with the laxity of the capsule.

The shoulder-joint is the one which is most commonly the seat of dislocation, generally the outcome of falls upon the outstretched hand, upon the elbow, or upon the point of the shoulder. In any of these accidents the head of the humerus tends to be pushed forcibly against the anterior part of the capsule, and if the muscles surrounding the joint are taken off their guard, a rent in the capsule will occur, through which the head of the humerus will be protruded.

The most frequent dislocation is that in which the head is displaced forwards and inwards, so that it comes to lie beneath the coracoid process, and the displacement is therefore termed sub-coracoid. In the majority of such dislocations the head usually reaches a point which is somewhat internal to the line of

the coracoid, the posterior surface of the anatomical neck resting as a rule upon the anterior margin of the glenoid fossa. It will thus be seen that, strictly speaking, the position is rather intra-coracoid than sub-coracoid. The subscapularis muscle is usually lacerated during the displacement of the head, although sometimes it remains untoned, but greatly stretched over the surface of the bone. The supraspinatus, infraspinatus and teres minor are taut over the now-empty glenoid fossa; and in some instances they may drag off the great tuberosity of the humerus.

If the head of the humerus is displaced still further inwards, so as to lie between the pectoral muscles and the serratus magnus—a position which can only be assumed in a wide laceration of the capsule with the muscles attached to it—the dislocation is frequently termed sub-clavicular.

As a rule there is marked evidence from examination of the shoulder-joint that one of these two forms of dislocation has occurred. The first obvious sign is that, owing to the displacement of the head inwards, the deltoid muscle will pass vertically from its attachment to the acromion to the outer side of the shaft of the humerus, its fibres being no longer raised and curved by the head articulating with the glenoid fossa. The affected shoulder thus appears distinctly flattened. For the same reason a straight line, such as the edge of a splint, can now touch the tip of the acromion process and the external condyle of the humerus at one and the same time, whereas in the normal condition the axis of the humerus will prevent this.

Further the anterior wall of the axilla may be bulged, and the infraclavicular fossa obliterated. Thus it comes about that the vertical measurement round the shoulder and axilla will be greater on the affected than on the sound side.

Again, the range of movement at the dislocated shoulder is decidedly limited, and it will be found that the sufferer is unable to place the palm of the hand of the affected limb upon the opposite shoulder with the elbow applied to the side of the chest, owing to the long axis of the humerus being now directed downwards and a good deal outwards.

The displacement of the head of the bone inwards will necessarily bring about pressure upon the structures lying internal to it. The axillary vein is frequently the first to suffer, and œdema of the limb distally, from obstruction to the return of venous blood, is a common sign. If the compression is great and is maintained too long, there is a possibility of gangrene occurring. It is well also to remember that in dislocations which have remained unreduced for some length of time, adhesions may have formed between the vessels and the protruded head, and there is some likelihood of their being torn in too energetic attempts at reduction.

Pressure upon the nerves of the brachial plexus gives rise to referred pain, and in some cases paralysis, particularly of the deltoid, as a result of interference with the function of the circumflex nerve.

In the reduction of this form of dislocation by the common method of manipulation, it is desired first to stretch the opening in the capsule by bringing the elbow to the side and firmly rotating the humerus outwards; secondly, to bring the head of the bone well under the acromial arch by raising the elbow and carrying it somewhat inwards across the chest. The last step in the manipulation, namely rotating the arm suddenly inwards and carrying the elbow still further across the chest, will cause the head to slip through the rent in the capsule and over the margin of the glenoid fossa into its natural position.

There are two other varieties of dislocation of the shoulder. In one the head also leaves the capsule at its unprotected area, namely below and somewhat in front, and rests, hitched as it were, beneath the glenoid fossa: this displacement is in consequence styled sub-glenoid. In this way some lengthening will be observed on the affected side, if a measurement is taken from the acromion process to the external condyle of the humerus.

In the last variety the head is displaced backwards through a rent in the posterior and lower part of the capsule, and, tearing up the infraspinatus muscle, it comes to lie beneath the spine of the scapula, the dislocation is therefore termed sub-spinous.

**Dislocations at the Elbow-Joint.**—The elbow-joint is anatomically a fairly secure articulation. The greater sigmoid cavity of the ulna very closely embraces the trochlear surface of the humerus, which is limited internally by a prominent margin. While the shallow cup-shaped facet on the upper surface of the head of the radius cannot be said to have much co-adaptation with the capitellum of the humerus, yet the ligaments adjacent, particularly the orbicular and the external lateral, together with the supinator brevis muscle, serve to keep the bony surfaces securely in contact.

The smallness of the lip of the coronoid process, the liability of the upper extremity to severe violence, and the complicated movements of pronation and supination all tend to neutralise the anatomical peculiarities making for safety. Thus it comes about that the elbow is more frequently the site of dislocation than any other joint except the shoulder, and the temporo-mandibular joints.

The bones of the forearm entering into the elbow-joint may be dislocated together or separately. There are, however, only two common dislocations, one where both the ulna and radius are displaced backwards, and the other where the radius alone has its head dislocated forwards. The backward displacement is probably most usually brought about by a fall on the extended and pronated hand, with the elbow slightly flexed. The anterior ligament and the anterior portions of the lateral ligaments are suddenly put upon the stretch, they snap, and allow the coronoid process to slip around beneath the trochlea into the olecranon fossa, dragging the head of the radius surrounded by the orbicular ligament with it. At the same time the lower end of the humerus is forced forwards, completing the laceration of the anterior ligament and the displacement of the bones. Occasionally the tip of the coronoid process is broken off, but it does not carry with it the attachment of the brachialis anticus. The fracture of the process however leads almost necessarily to a recurrence of the displacement after reduction, owing to the want of an anterior lip to the sigmoid cavity to hold the ulna in position.

The diagnosis is very simple, for the alteration in the anatomical relationship of bony points is very great.

There are four prominences of bone about the elbow-joint, the tip of the olecranon process, the internal epicondyle, the external epicondyle and the head of the radius. Their exact relations to each other and in all positions of the forearm are of the greatest import, and a comparison of the two limbs should always be made.

In the fully extended position of the bones of the forearm, the olecranon sinks into its proper fossa and therefore becomes less prominent. Its upper margin will then be on a level with a line drawn across the back of the arm from the most prominent points of the epicondyles. As flexion occurs the olecranon becomes more and more prominent, and sinks to a level below the intercondylar line, and when the arm and forearm are at right angles the olecranon is the lowest point of the elbow. With full flexion the olecranon is carried forwards, and comes to rest anterior to the lower articular extremity of the humerus. The coronoid process cannot be felt, being thickly covered by the brachialis anticus muscle.

The head of the radius lies immediately below the dimple caused by the line of the radio-humeral articulation, that is, a short distance below the external epicondyle. If the finger be placed over it and the hand pronated and supinated, the radial head is readily felt to rotate, while if the finger lies upon the epicondyle this is found to remain stationary.

In a backward dislocation, the normal relationship of the olecranon to the other bony points is altogether lost. Further, seeing that the bones of the forearm are carried backwards, the length of the forearm measured from the external epicondyle to the styloid process of the radius will necessarily be diminished.

Forward displacement of the head of the radius is often caused when a child is caught up by the hand. The sudden strain thus thrown upon the ligaments of the wrist-joint, through the metacarpus and the carpus, acts upon the head of the radius rather than the ulna, and jerks the head of the radius out of the orbicular ligament, with or without a laceration of the latter.

The immediate consequence of this displacement is a pronounced alteration in the relationship of the head of the radius to the external condyle of the humerus when compared with the opposite, normal, side. The prominence of the head of the bone in front of the capitellum explains the reason why flexion is so limited, seeing that the head soon comes in contact with the soft tissues of the arm.

The bone can usually be comparatively easily reduced into position, but the difficulty is to maintain it there. This difficulty is entirely due to anatomical reasons. If the orbicular ligament is lacerated, there is nothing to keep the head of the radius in place, while if this ligament remains intact, it is impossible by manipulation to reinsert the head of the radius within it.

**Dislocations at the Wrist Joint.**—These dislocations are of extreme rarity, probably owing to the strength of the ligaments of the radio-carpal articulation and the manner in which the strain of a fall upon the palm of the hand is shared by the several articulations of the carpus. When, however, one does occur it is usually backward. In these dislocations it will be noticed that the styloid processes retain their normal anatomical relationship, namely, that of the radius being at a somewhat lower level than that of the ulna, and further that the hand has little or no tendency to a radial displacement. Both of these facts serve to distinguish this dislocation from fracture of the lower end of the radius or separation of its distal epiphysis.

**Dislocations at the Metacarpo-phalangeal Joints.**—These are most frequently seen in the case of that of the thumb, in which the proximal phalanx is displaced backwards.

The anterior ligament is stretched and the head of the metacarpal bone tears it transversely away from its attachment, and when the base of the phalanx is displaced backwards the sheet of glenoid ligament is carried with it so as to come to lie on the dorsal aspect of the metacarpal bone. Sometimes, however, the anterior or glenoid ligament is split vertically, when one half will lie embracing either side of the neck of the metacarpal bone when the phalanx is dislocated dorsally. In all the backward

dislocations the two tendons of the flexor brevis pollicis, together with their enclosed sesamoid bones, will occupy a similar position. At the same time one or other lateral ligament, generally the outer, is lacerated.

It is probable that the difficulty in reduction is due chiefly to the fact that when extension is applied to the proximal phalanx, the torn glenoid ligament is drawn taut, and forms a sheet which prevents the return of the base of the phalanx forwards into its natural position. At the same time the tendons of the flexor brevis, together with the sesamoid bones, grasp the neck of the metacarpal bone much in the same way as a button-hole does the shank of a button, and thus still further interfere with reduction.

In order to overcome this difficulty in reduction, an anæsthetic may be required, and a series of particular manipulations should be carried out. The first manœuvre is to adduct the metacarpal bone towards the palm of the hand; the second is to acutely dorsi-flex or hyper-extend the first phalanx, so as to bring its base up on to the head of the metacarpal bone, and thus open up the sides of the "button-hole" and displace the torn glenoid ligament forwards. Then a sudden flexion of the phalanx towards the palm of the hand, whilst its base is steadied by the surgeon's thumb, placed behind it, causes the head of the metacarpal bone to slip through the button-hole formed by the tendons of the flexor brevis and the sesamoid bones and the base of the phalanx to pass into its normal position.

Should these manipulations fail, operative interference is necessary. This is best carried out by the open method. A median incision over the protruding head of the metacarpal bone on the palmar aspect will readily show the encircling tendons with their sesamoid bones. If these are hooked on one side and the glenoid ligament drawn forwards, it is usually easy to press the head of the bone backwards through the "button-hole," and so bring the base of the phalanx into position. If a few stitches are inserted so as to hold the torn parts of the ligament together, there is but little chance of a recurrence of the displacement.

**Dislocations at the Interphalangeal Joints.**—The dislocation most commonly seen is a backward displacement of the distal phalanx of the little finger, and as a rule the anterior ligament is fully torn. Reduction, however, by extension is fairly easy, and a dorsal splint will prevent recurrence.

### Lower Extremity.

**Dislocations at the Hip-Joint.**—The conformation and strength of the hip-joint, including the depth of the acetabular cavity and the perfect way in which the head of the femur fits into it, the thickness of the capsule and the strength of the muscles that surround the joint, explain the comparative rarity of traumatic dislocations at this articulation. On the other hand, it must be remembered that the head of the femur is at the end of a long lever even when violence is applied to the knee, and much more so when it is applied to the foot, a factor which it might be thought would predispose to displacement of the head from its socket, but as a matter of fact fracture of the neck of the femur is much more common than dislocation at the joint. The probable explanation of these two lesions, both arising from the same kind of violence, is to be found in the position of the limb at the time when the force is applied. In order that fracture of the neck may occur, the limb should be in the fully extended position, without abduction or adduction; in order that dislocation should occur, the hip-joint must be flexed and the limb abducted.

The only dislocations at the hip-joint which are seen with any comparative frequency are those which are termed "regular," in which the ilio-femoral or  $\Lambda$ -shaped ligament remains intact. The lower portion of the acetabulum is shallowest and the margin is notched, and it is at this position as a rule that the head of the bone slips out of the socket.

The most usual dislocation is a dorsal one, in a direction upwards and backwards. The capsule is torn posteriorly, and as a rule the ligamentum teres is ruptured. As soon as the head of



the femur leaves the acetabulum, it passes most commonly above the tendon of the obturator internus muscle. Many of the muscles about the joint are torn, but to a different degree in various cases. The gluteus maximus often has its deeper fibres lacerated, and frequently the gluteus medius, pyriformis and even the obturator internus and gemelli are damaged. The ilio-femoral ligament, as has been mentioned, remains intact, although its external or upper fibres will be found very tense.

The head of the bone being displaced backward, the great trochanter comes to look forwards, and there will be marked shortening of the limb, the actual amount depending upon the distance to which the head of the bone has ascended upon the dorsum ilii. The foot is inverted so that the ball of the great toe rests against the instep of the sound foot, the long axis of the dislocated femur being directed across the lower third of the sound thigh.

In the reduction of this form of dislocation by manipulation, flexion of the thigh upon the abdomen relaxes the ilio-femoral ligament, and some abduction still further induces this condition. Slight internal rotation disengages the head from behind the acetabulum, then traction upon the limb in the line of the femur brings the head of this bone over the socket, into which it will generally slip.

**Dislocations of the Patella.**—Again, dislocation of the patella is a rare displacement, the only comparatively common variety of the accident being a dislocation outwards. The line of contraction of the quadriceps muscle has a tendency to drag the patella outwards, seeing that the direction of the large bulk of the fibres of this composite muscle is downwards and inwards. This obliquity of action is naturally considerably increased in cases of genu valgum, in some instances of which the patella may slip outwards over the external condyle whenever the knee is flexed.

**Dislocations at the Knee.**—The great strength of the ligaments entering into the knee joint explains the rarity of dislocation at this articulation, and even when it occurs, it is usually but partial.

In dislocation backwards of the tibia, the posterior ligament of the knee joint is as a rule torn, and many of the other ligaments, particularly the crucial, must partially or completely give way. The vessels and nerves of the popliteal space are severely pressed upon by the posterior margin of the head of the tibia, in backward dislocation of this bone, or are tightly stretched in the popliteal notch in forward displacement of the tibia. In both cases, the pulse in the tibial arteries may be obliterated.

**Dislocations of the Semilunar Cartilages.**—Displacement of the internal semilunar cartilage is undoubtedly more common than that of the external. As a rule, the fibro-cartilage is torn away from its anterior attachment to the front of the spine of the tibia, and from the anterior third of its marginal attachment. The anatomical explanation of the frequency of the displacement of the internal cartilage may be the following:—First, that this cartilage is perhaps more insecurely attached than the external, particularly so far as its marginal attachment to the head of the tibia is concerned; secondly, because in the position and movement of the joint in and during which a dislocation of the cartilage occurs, namely, flexion at the knee with rotation outwards of the leg, the lower end of the femur is driven as it were against the circle of the cartilage and tears it from its attachment, forcing it away from the interior of the joint. Sometimes the prominence formed by it after dislocation on the inner side can be distinctly palpated and the joint is locked, so that extension cannot be brought about until the cartilage has been forced into position.

If dislocation of the cartilage constantly recurs, its removal operation as a rule brings about a very satisfactory result, the loss of the cartilage in no way affecting the function of the joint even when a small portion near the posterior attachment is left behind.

**Dislocations at the Ankle-Joint.**—Owing to the conformation of this joint, dislocation of the whole foot including the astragalus can only occur backwards or forwards, apart from an accompanying fracture, lateral dislocations of necessity requiring a solution of continuity of one of the bones.

**Dislocations of the Astragalus alone.**—Dislocation of the astragalus alone from the malleolar arch as a rule takes place in a forward direction, and generally results from the weight of the body coming upon the upper surface of the astragalus when there is ventro-flexion at the ankle joint, as in alighting upon the toes when jumping from a height. The bone when it is shot forwards as a rule passes somewhat outwards and comes to lie beneath the tendons of the extensor longus digitorum, which together with the skin are tightly stretched over it, and unless the bone is reduced or excised by the surgeon, the integument will slough because it is deprived of its blood supply by the tension to which it is subjected.

### Skull.

**Dislocations at the Temporo-mandibular Joint.**—The condyle of the mandible may be displaced out of the glenoid fossa of the temporal bone on one or both sides. A hollow will appear in the place where the condyle should naturally be, and the condyle will lie in front of the eminentia articularis. The external pterygoid muscle draws forwards the condyle and with it the meniscus. The temporal, internal pterygoid, and the masseter muscles pull the mandible upwards and constitute the main obstacle to reduction, since they tend to prevent the condyle from passing beneath the eminentia articularis and so back into the glenoid fossa.

## CHAPTER IX.

### DISEASES OF BONES AND JOINTS.

#### DISEASES OF BONES.

**Inflammation of Bone.**—Because of close continuity, it is highly improbable that inflammation attacks any one constituent part of a bone. Thus, in periostitis there is always a certain amount of osteitis, and in osteomyelitis it is usual for the periosteum to be sooner or later involved.

*Periostitis* may be of a simple or a specific origin. Simple traumatic periostitis occurs most frequently in those bones which are chiefly exposed to injury. Hence it is common in the tibia, which is so liable to meet with blows or kicks. Moreover, acute infective periostitis also as a rule attacks those bones which may meet with traumatism; therefore the femur, the tibia, the radius, the clavicle and the lower jaw are most generally affected. The micro-organisms inducing the disease are generally conveyed to the injured portion of the diaphysis by the blood stream, the patient not infrequently having a small focus of suppuration somewhere in the body, such as a boil or patch of impetigo contagiosum. The inflammation as a rule starts in the diaphysis not far from an epiphysis, and most usually near to that epiphysis from which the bone chiefly grows in length, as for instance, the proximal end of the tibia, the distal end of the radius and the distal end of the femur, possibly owing to the greater vascularity of the part. The inflammation is limited to the diaphysis because of the close adhesion of the periosteum to the epiphyseal cartilage. Therefore while the whole of the shaft of the bone may perish from the fluid effused beneath the periosteum depriving it of its blood supply, the epiphysis retains its vitality because its enveloping periosteum is not stripped off, and its

arterial twigs are so numerous. For the same reason, the adjacent joint is comparatively rarely involved. The separated diaphysial periosteum may, if the patient lives, throw down fresh bone at some distance from the necrosed shaft, and it will be this newly-formed bone which will support the limb after the removal of the sequestrum. Therefore it follows that if that portion of the extremity is attacked which only contains a single bone, it is absolutely necessary that a large quantity of ensheathing bone should be thrown out before the necrosed portion is removed, otherwise great and permanent shortening of the limb will occur.

*Chronic Abscess of Bone.*—Whether due to tuberculous or staphylococcic infection, it is the cancellous tissue at the epiphyseal end of a long bone which is usually the seat of a chronic abscess. Enlargement occurs, and a tender spot is often present evidencing the line along which the pus is endeavouring to obtain an exit, and indicating the spot at which the abscess should be attacked. It is interesting to note that the pus finds its way out through the bone to the skin much more frequently than through the articular cartilage into the joint, the non-vascular cartilage being a highly resistant structure.

*Caries of the Spine.*—The deposit of tubercle bacilli apparently takes place most usually in the newly formed bone deep to the epiphyseal discs of the centrum. The regions commonly affected are those where most movement occurs, hence the dorsi-lumbar and cervico-dorsal junctures are frequent sites of the disease.

As destruction of the bone forming the body progresses, the weight of the superincumbent parts causes the centra to be pressed together, whereby the spinous processes become prominent in an angle, and give rise to the typical kyphosis or angular curvature.

The spinal nerve roots issuing from the intervertebral foramina are liable to be irritated by the inflammation associated with the tuberculous granulation tissue, hence pain, referred to the peripheral distribution of these nerves, is a very common occurrence. Consequently a child suffering from lower dorsal

caries may complain of pain in the abdominal wall, which may be attributed to intestinal colic, rather than spinal disease.

Tuberculous disease involving the occipito-atloid joint has referred pain over the mastoid process, on account of the termination of the suboccipital or first cervical nerve in this region; and again the same lesion in the atlo-axoid joint is associated with pain in the occipital region of the scalp, from the distribution of the great occipital branch of the second cervical nerve. In atlo-axoid disease the transverse ligament behind the odontoid process may be eaten through, and in consequence the head, together with the atlas, slips forward, and the odontoid crushes the spinal cord and induces instant death.

Paraplegia may occur in the course of tuberculous disease of the spine, occasioned by the pressure of tuberculous granulation tissue derived from the diseased centres of the vertebræ. The posterior common ligament, so long as it remains intact, offers a barrier for the extension backwards of the inflammatory products, but when it is eroded, or is rendered flaccid by the falling together of two or more vertebral bodies, pressure is allowed upon the anterior columns of the spinal cord through the thecal sheath, and paraplegia results. It is seldom that the actual bony tissue in curvature of the spine induces the pressure upon the cord.

It is obvious that from its position it is a difficult matter to reach this mass of granulation tissue in order to remove it and thus relieve the pressure upon the cord. If it is approached from behind by laminectomy, the whole thickness of the cord lies between the operator and the tuberculous material which is producing the deleterious pressure.

#### DISEASES OF JOINTS.

**Synovitis.**—In synovitis there is secreted an increased amount of synovial fluid, causing the joint cavity to be filled and the synovial membrane to bulge. The actual space within a joint is relatively small. Thus, in the case of the largest joint in the body—that of the knee—it requires only two ounces of fluid to

fully distend it in the adult. In consequence of the increase of fluid, all the natural depressions about a joint become obliterated, or even converted into elevations, and thus the outline of the limits of the synovial membrane becomes apparent.

**Joints of the Upper Extremity.**—*Sterno-Clavicular.* Owing to the weakness of the anterior ligament, fluid in this joint shews itself by a distinct prominence over the front of the joint.

*Shoulder-joint.*—When distended with fluid the bony surfaces may be appreciably separated, owing to the laxness of the capsule, the rotundity of the deltoid is increased, the elbow is carried a little backward and humerus rotated internally, and thus a slight lengthening of the arm may be demonstrated. The hollow groove between the pectoralis major and the deltoid is obliterated, and fluctuation may be determined by manipulation of that part of the capsule which may be felt in the axilla.

There are three diverticula of the synovial membrane of the shoulder, all in connection with tendons. Beneath the subscapularis in front and to the inner side, around the long tendon of the biceps for some distance down the bicipital groove anteriorly, and beneath the infraspinatus near its insertion behind. All of these become distended with fluid in synovitis.

In suppurative synovitis pus may track along any one of these pouches. If it escapes by the subclavicular route, it may spread between the subscapularis and the venter of the scapula. When it leaves the joint cavity by the bicipital groove, it may ultimately appear at the lower and anterior border of the deltoid. Escaping behind the joint, purulent fluid nearly always progresses forwards towards the anterior aspect of the limb, this being the path of least resistance owing to the denseness of the fascia covering the deltoid.

*Elbow-joint.*—The elbow-joint when filled with fluid, as the result of inflammation, loses most of its natural contour, and all the guiding bony points become greatly obscured. The obliteration of the fossæ on either side of the olecranon process is very marked, and readily indicates the existence of synovitis.

Pus within the joint as a rule finds its way to the surface about

the posterior and outer aspects of the articulation, this being again the line of least resistance, since the front of the joint is well protected by the brachialis anticus muscle.

*Wrist-joint.*—In acute inflammatory conditions of the wrist and carpal joints, not infrequently seen in acute rheumatism and gonococcic infection, the actual distension of the joint cavities themselves is frequently masked by the swelling in the tissues around, particularly in the extensor tendon sheaths. It is well therefore to be careful not to overlook the joint lesion, which, as a rule, may be diagnosed by the pain produced by jarring the joint surfaces together without simultaneously throwing into action the flexor or extensor muscles.

In suppurative synovitis abscesses will make their appearance chiefly about the dorsal aspect which is only covered by a slight amount of soft tissue.

**Joints of the Lower Extremity.**—*Hip-joint.* Owing to the great depth at which this joint is placed, distension of its cavity in synovitis is not readily detected, but on comparing the two groins, especially in children, and in thin persons, there will appear to be some fulness on the affected side. Occasionally a similar condition may be made out behind and internal to the great trochanter.

As a rule the position in which the limb is placed is that of flexion, abduction and external rotation. This is the position of rest, because it is that in which most of the ligaments are relaxed.

It has also been thought that the position occurs owing to a reflex contraction of the muscles about the joint, together with the fact that the flexors, abductors, and external rotators are stronger than their opponents.

Pain in synovitis of the hip, and particularly in that form dependent upon infection by tubercle, is felt not only in the region of the joint itself but is often referred to the knee, and peculiarly to the inner and posterior aspect of the distal joint. This fact is due to the anatomical reason that at least two nerves supply twigs to both of the articulations, namely the obturator and the anterior crural. It is possible that the referred knee joint pain is



most marked in those instances in which the (so-called) ligamentum teres is diseased, for the obturator nerve sends a special branch into the substance of this structure.

It is possible also that pain from inflammation of the anterior part of the hip may be even referred, through the internal saphenous nerve, to the inner side of the ankle, and also that pain may be felt in the heel or foot owing to the fact that the twigs supplying the posterior part of the hip-joint and the nerve fibres constituting the great sciatic and its divisions are all derived from the same source, namely the sacral plexus. It is well to bear in mind however that pain referred to these various places is by no means always present in morbus coxæ, and because of its absence inflammation of the hip must not be overlooked.

In suppurative synovitis (arthritis), the purulent material as a rule perforates the capsule posteriorly where it is thinnest, and gives rise to an abscess behind. Subsequently, however, the pus, following the line of least resistance, tracks forward beneath the gluteus minimus and medius, and at the anterior border of these muscles it passes internal to the tensor fasciæ femoris and deep to the upper part of the sartorius, forming a swelling in the upper and outer part of Scarpa's triangle.

When the acetabulum is perforated, as it may readily be in children in whom the Y-shaped cartilage is still in existence, pus will collect within the pelvis between the bones and the obturator internus. Hence a rectal examination should not be neglected in any case of suppuration in connection with the hip-joint.

*Knee-joint.*—Probably synovitis of the knee-joint occurs almost more frequently than inflammation of any other joint except the ankle. The synovial cavity of the knee-joint is the largest in the body and yet it only requires an ounce and a half to two ounces of fluid to completely distend it in the normal adult articulation.

The limits of the synovial cavity are readily seen when fluid is poured out as the result of inflammation. All the natural depressions around the joint are obliterated, and elevations appear in place of them. Thus, bulging will be seen on either side of the highest part of the ligamentum patellæ and laterally to the

rectus tendon. The patella also is raised on the surface of the fluid and can be made, by a sharp tap on its anterior surface, to pass backwards through the fluid and to strike the anterior surface of the condyles of the femur. In order to obtain this latter sign satisfactorily, it is well to have the quadriceps thoroughly relaxed and to press the fluid from the upper part of the synovial cavity into that portion which lies immediately behind the patella.

Suppurative synovitis is always an extremely serious matter, particularly when depending upon staphylococcic infection, because of the large absorbing surface bathed with purulent fluid. The various recesses of the synovial cavity of this joint make it an extremely difficult one from which to get efficient drainage; there are two condylar pouches which terminate above and behind the condyles, beneath the heads of the gastrocnemius; a synovial prolongation of the outer pouch which lies along the tendon of the popliteus; and the great subcrureus bursa. (Fig. 7.) It is well to remember, in making lateral incisions for drainage, that the one placed on the outer side will probably be the more efficient, because the patient naturally tends to externally rotate the limb—hence the external surface becomes the most dependent part—and because fewer muscular fibres have to be cut through, from the fact that the fibres of the external vastus do not descend so low as those of the internal vastus. Opening the condylar pouches of the joint on the posterior aspect needs great care on account of the structures lying in the popliteal space.

*Ankle-joint.*—Inflammation of the ankle-joint is extremely common, generally as the outcome of traumatism, such as a sprain. Again, all the natural depressions about the joint will become obliterated, and a puffiness or swelling will appear on either side of the tendo Achillis and about the anterior surface of the joint deep to the extensor tendons.

**Tuberculosis of Joints.**—Infection of a synovial membrane with the tubercle bacillus is very frequent, and probably so because it is well supplied with blood, its capillaries are somewhat smaller than in other tissues, and from movement and

KNEE-JOINT.

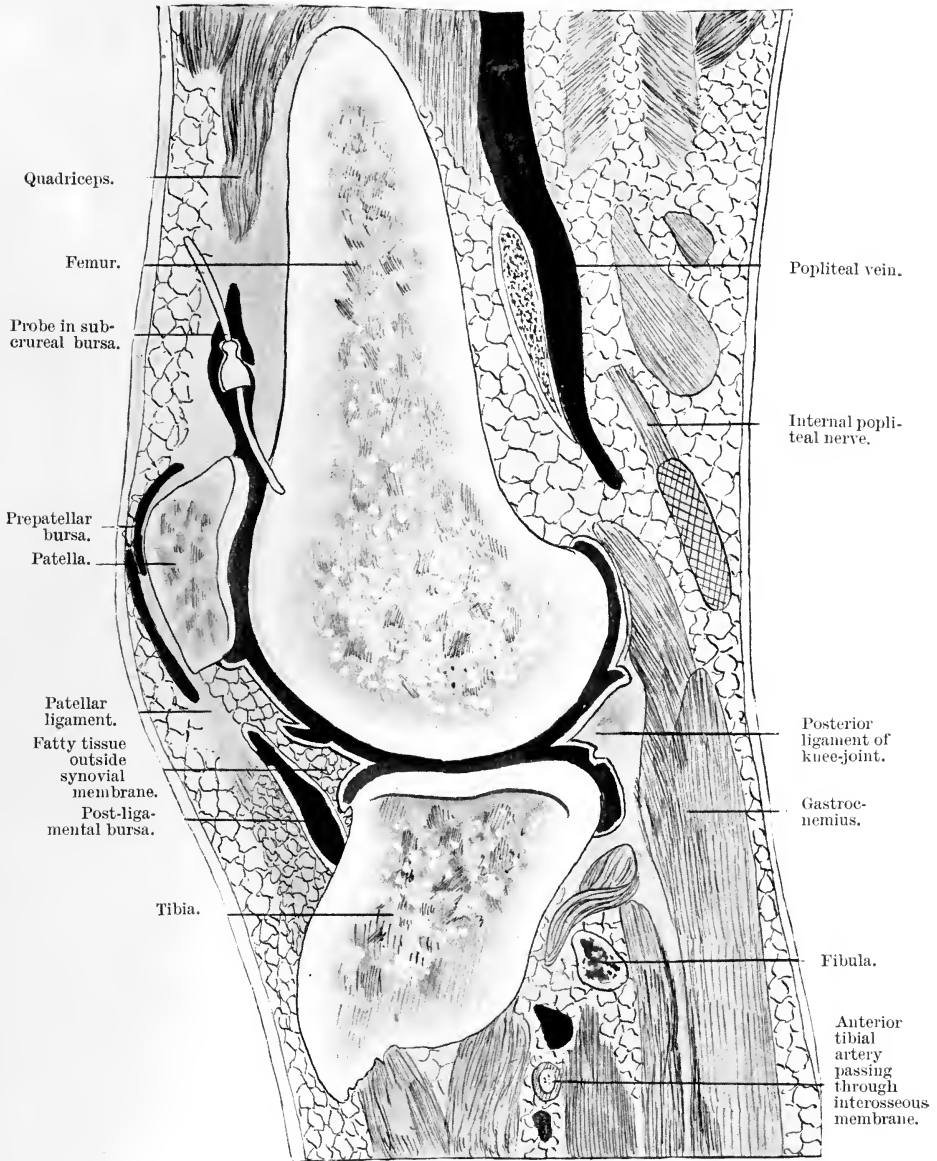


FIG. 7.—Sagittal section of a knee-joint, showing extent of the synovial cavity, and some of the adjacent bursæ. (Modified from Bardeleben.)



external violence slight amounts of traumatism are constantly occurring, leading to inflammation and a favourable soil for the growth of the bacilli.

It is well to remember that the synovial membrane itself only lines the inner surface of the ligaments up to the margin of the articular cartilage. It is only in cases of disease that the membrane tends to creep over and to veil the articular cartilage—a condition so common in tuberculous arthritis.

Tuberculosis leads to the destruction of ligaments and to subsequent pathological dislocations. The direction in which the bones are displaced depends greatly upon the comparative strength of the muscles acting upon them. Thus, for instance, in the case of the knee-joint the tibia is drawn backwards and flexed by the ham-strings which are stronger than the extensors, and is rotated outwards by the biceps, the action of which is more powerful than that of the internal rotators of the leg, namely the sartorius and popliteus.

## CHAPTER X.

### DISEASES OF MUSCLES, TENDONS, FASCIÆ, AND BURSÆ. TALIPES.

#### MUSCULAR DYSTROPHIES.

THE distribution of the wasting in the muscular dystrophies does not correspond to that met with in infantile paralysis or in progressive muscular atrophy. In both the latter diseases there is reason to believe that the distribution is determined by the segmental representation of muscles in the spinal cord. Neither does the wasting in the dystrophies correspond to the groups of muscles supplied by individual peripheral nerves. It has been assumed, on slight evidence certainly, that the muscles which are the first to be developed in the fœtus are the first to become degenerated when muscular dystrophy supervenes.

A knowledge of the influence of various muscles in producing the normal contour of the body, and also of their actions, is essential for the investigation and discrimination of the various types of muscular dystrophy. The annexed table shows the muscles which need investigation in the different forms, but it must not be forgotten that intermediate varieties occur, and that sometimes the disease is exceedingly widespread.

The *deltoid* abducts the humerus to a right angle, and in so doing tends to rotate the scapula so that the acromion points downwards. This tendency is counteracted by the contraction of the acromial fibres of the trapezius. The anterior fibres of the deltoid carry the arm forwards in a horizontal plane. The fibres forming the posterior third of the muscle are said to adduct the arm to the body. When the deltoid is paralysed the shoulder becomes flattened and the head of the humerus tends to sink

away from the acromion, a tendency which is much accentuated when the supraspinatus is also affected.

The *infraspinatus* rotates the arm outwards. The muscle being comparatively superficial, alteration in the way of wasting or of hypertrophy is easily recognised.

**Types of Muscular Dystrophy.**

	Pseudo-hypertrophic (Duchenne). Atrophic Form (Leyden and Möebius).		Juvenile (Erb).		Facio-scapulo-humeral (Landouzy and Dejerine).
Face.	Normal.		Normal.		Zygomatici Lev. Labii Superioris Orbicularis Oris Orbicularis Palpebrarum
					} Wasted.
Arm and Shoulder.	Deltoid Supraspinatus Infraspinatus (especially) Triceps? Biceps?	} Enlarged.	Deltoid Supraspinatus Infraspinatus Muscles of Fore- arm	} Escape.	}
	Pectoralis Major (lower part) Latissimus Dorsi Biceps Supinator Longus? Intrinsic Muscles of Hand?		Triceps Biceps Brachialis Anticus Supinator Longus		
			Whole Trapezius Pectoralis Major (lower part espe- cially) Serratus Magnus Latissimus Dorsi (lower part espe- cially) Rhomboids Sterno-mastoid	} Also wasted.	} As in Erb's Juvenile Type.
Pelvis, Thigh, and Leg.	Glutei Quadriceps (espe- cially Vastus Externus) Calf Muscles	} Enlarged.	Glutei Flexors of Hip Quadriceps and Muscles above Knee	} Wasted.	
	Flexors of Hip Flexors of Knee Peronei Anterior Tibial Group		} Wasted.		

The *triceps* extends the elbow. Its long head also adducts the arm. Paralysis of the triceps renders supination of the extended forearm impossible, for the biceps which supinates is also a flexor and will flex the forearm when supination is attempted unless it is antagonised by simultaneous contraction of the triceps.

The *pectoralis major* consists of two portions, clavicular and

sternal. One of these may be wasted without the other. The action of the two parts of the muscle is demonstrated as follows. If the arm is advanced in the horizontal plane and then carried towards the mid-line of the body against resistance, both parts of the muscle stand out. If, in this position, the arm is raised against resistance, only the clavicular fibres contract; if it is depressed against resistance only the costo-sternal fibres are in action. The sternal part is frequently involved in the muscular dystrophies, and wasting of the latissimus dorsi is often associated. The clavicular part is usually affected together with the serratus magnus. Wasting of the clavicular fibres of the pectoralis major gives rise to subclavicular depressions.

The *latissimus dorsi* retracts and adducts the humerus, the arm being carried across the back as in the action of placing the hand in the coat-tail pocket. The muscle can be made to stand out by executing this movement against resistance. By palpating the posterior axillary fold the muscle can also be felt to contract during coughing or sneezing. Its contracture during these expiratory efforts is probably for the purpose of fixing the lower ribs in order that the external oblique muscle, which rises from them, may compress the abdomen.

The *biceps* is a flexor and supinator of the forearm. When supination without flexion is required this muscle is antagonised by the triceps.

The *brachialis anticus* is a flexor of the elbow.

Wasting of the triceps, biceps and brachialis anticus produces the slender cylindrical arm seen in muscular dystrophy.

The *supinator longus* or *brachio-radialis* flexes the elbow and is usually said to always bring the thumb uppermost from any position. It is, however, a pure flexor without any pronator or supinator action. Its fleshy belly at the outer side of the ante-cubital fossa is easily recognised when the forearm is flexed against resistance.

The *intrinsic muscles of the hand*.—The dorsal interossei abduct and the palmar interossei adduct the extended fingers away from or towards a line running through the middle finger.



In addition the interossei and lumbricales, by means of their attachments to the long extensor of the fingers as that passes over the proximal phalanges, flex those phalanges and extend the two distal ones. The long extensor of the fingers is practically responsible only for the extension of the proximal phalanges to which, paradoxically enough, it is not attached.

When the interossei and lumbricales are paralysed, the action of the long extensor on the first phalanges, and of the long flexor on the two terminal phalanges of each finger are not antagonised and a claw hand results. The intrinsic muscles of the hand are but rarely involved in the muscular dystrophies.

The *trapezius*.—The clavicular and the lower portions of this muscle must be considered separately.

The clavicular portion. When the arm is to be abducted laterally from the body the scapula must lie applied to the back of the thorax in such a way that it can become tilted in a transverse vertical plane. This allows the glenoid cavity to be directed upwards and outwards. To permit the scapula to glide backwards into the appropriate position, the outer end of the clavicle is carried directly backwards. This is the function of the clavicular fibres of the trapezius. The deltoid then abducts the arm and the serratus magnus, by tilting the scapula, acromion upwards, completes the elevation.

The lower portion. This part keeps the scapula in apposition with the thorax during the first part of the action of advancing the arm. When the humerus has been moved by the deltoid through 45 degrees the serratus magnus comes into action and performs a similar function to that of the trapezius, at the same time tilting the scapula. Consequently a slight starting of the vertebral edge of the scapula away from the thoracic wall, or "winging," at the commencement of the advance of the arm may be caused by paralysis of the lower part of the trapezius. If the serratus magnus be intact the deformity will disappear when the arm reaches the horizontal plane, whereas the deformity due to serratus paralysis is at its maximum in this position, especially if a pushing effort be made at the same time. A

similar sequence of events is noticed when the arm is abducted laterally instead of advanced. But now, instead of becoming winged, the inferior angle of the scapula moves a short distance towards the spine. As soon as the serratus commences to act the bone moves in the opposite direction towards the axilla.

The *serratus magnus* is best put in action by causing the patient to push directly forwards against resistance. The part played by the muscle in elevating the arm has already been alluded to. Without the aid of the serratus magnus in tilting the glenoid cavity upwards the deltoid cannot raise the humerus above the horizontal line.

The *rhomboids*.—When the *teres* muscles are in action, depressing and adducting the humerus, they at the same time tend to draw the inferior angle of the scapula into the axilla, and this actually happens if the rhomboids are paralysed, since the latter, with the lower part of the trapezius, normally counteract this displacement.

The *gluteus maximus* is a powerful extensor of the hip. It is not employed in standing or walking on the level, but comes into action in such movements as going upstairs or rising from a seat or stepping up on a chair. Paralysis of this muscle causes the pelvis to remain tilted when standing and so produces a lordosis of the lumbar spine. This lordosis disappears when the patient sits, since the tilting of the pelvis is then counteracted. Weakness of the glutei also gives rise to a peculiar method of extending the trunk by pressure of the hands on the thighs. This is usually known as climbing up the thighs.

The *gluteus medius* and *gluteus minimus* are important in the act of walking. Their function is to draw the trunk over towards their own side so that the centre of gravity of the body is brought over the head of the femur. The muscles of the two sides act alternately, swaying the pelvis and trunk from one side to the other as the corresponding foot is planted on the ground.

The *ilio-pectineus* is a flexor of the hip. There is a divergence of opinion as to its rotatory action, but it is perhaps correct to regard

it as an internal rotator, its point of attachment being outside the axis of rotation of the femur.

The *calf muscles* are plantar flexors of the foot, and when weak there is inability to stand on tip-toe. Contracture of the interstitial tissue of the enlarged calf muscles in the pseudo-hypertrophic form of the disease produces talipes equinus.

The *quadriceps* is an extensor of the leg. Weakness of this compound muscle causes difficulty in extending the leg to gain the erect position. It also allows the knee to come forwards in advance of the foot in walking. The vastus internus portion when enlarged is easily recognised owing to the increase of the fleshy portion of this muscle which lies just above the inner side of the patella.

The *erector spinæ* muscle of each side is sometimes affected. If at the same time the glutei are intact the resulting lordosis is distinguished from that due to paralysis of the latter muscles by the fact that the pelvis is not tilted forwards.

#### OTHER DISEASES AND INJURIES OF MUSCLES.

A muscle may be said to consist of a mass of contractile tissue, enclosed within a fibrous sheath. As a rule, a muscle has a free portion or belly between two or more attachments, termed the origin and the insertion of the muscle. The anatomical disposition of certain muscles renders them peculiarly liable to disease or injury.

**Inflammation of Muscle (Myositis).**—This is usually of a chronic nature, and especially due to tubercle or syphilis. The disease may be primary, or, more usually, in the case of tubercle, secondary.

Muscles surrounding joints, the seat of tuberculous mischief, are very apt to become involved in the infection, and subsequently the spread of the purulent material is greatly influenced by the sheath of the particular muscle it has invaded, or by the inter-muscular planes it reaches. This influence is particularly well seen in the case of the *psoas magnus* muscle. This muscle is

attached to the sides of the bodies of the last thoracic and all the lumbar vertebræ. When the lower thoracic or the lumbar centra become infected with tubercle, and caseation or suppuration occurs, the tuberculous material is prevented from passing forward by the anterior common spinal ligament, is hindered from invading the spinal canal by the posterior common ligament, and therefore tracks laterally, and enters the substance of the psoas muscle. The muscular tissue gradually becomes displaced or absorbed, and the sheath filled with the tuberculous material. This is then conducted by the sheath downwards along the brim of the pelvis, behind Poupart's ligament to point in Scarpa's triangle not far from the insertion of the muscle into the lesser trochanter.

When such a collection of caseous matter is opened in the lumbar region, and the finger introduced, it is often found that the cords of the lumbar nervous plexus have been dissected out, and remain stretched across the cavity.

**Injuries of Muscles.**—Laceration of muscular fibres is a very common accident. A muscular belly may be completely ruptured within the sheath. The sheath of a muscle may be lacerated without the contained muscular fibres being torn, in which case a protrusion, or hernia, of the fibres may occur. A muscle and its sheath may both be ruptured simultaneously. Certain muscles from their attachments, position or functions are more liable to injury than others. Thus it is that some of those muscles which have their attachments widely separated and are subjected to sudden and violent strain, are often the site of laceration or rupture. The great stretching which the *sterno-mastoid* from its position must necessarily undergo during the birth of the after-coming head, by no means infrequently leads to some laceration, resulting in the formation of a hæmatoma, which constitutes the so-called tumour or gumma of the sterno-mastoid. Torticolis may result from the subsequent contraction.

The physiological action of the *rectus abdominis* in parturition, or even defæcation, may occasion some laceration of its fibres, and the consequent extravasation of blood which occurs within its

sheath may be widespread. Should the hæmatoma suppurate, the pus will at first be confined by the sheath of the rectus, but may track for a considerable distance within it, leading sometimes to most troublesome sinuses.

The *biceps cubiti* arises by two heads, the long from the upper border of the glenoid fossa, the short from the tip of the coracoid process. The long tendon is a part of the muscle which not infrequently gives way, and its passage through the joint over the head of the humerus conduces to its laceration, particularly when the tendon has been thinned by friction in cases of osteo-arthritis. It may then acquire an attachment lower down in the bicipital groove. The short head is rarely torn. The belly of the muscle may be ruptured in very forcible flexion against much resistance. The muscle is inserted by means of a tendon into the posterior part of the bicipital tubercle of the radius. In some cases of strong action, the tendon may be torn away from its attachment, and may even drag a piece of bone off with it.

The *plantaris*, arising from the back of the femur just above the external condyle, has a small belly, terminating in a long, slender tendon which is inserted into the tendo Achillis or into the inner side of the posterior surface of the os calcis. The tendon may be snapped in wrestling, slipping on the stairs, tennis, etc.

The *quadriceps extensor cruris*.—Sometimes, and for the same reasons that the patella is transversely fractured by muscular action, the tendon of the quadriceps may be torn across close above its insertion, the patella remaining intact.

#### DISEASES OF TENDONS.

Many tendons, especially when long as those of the flexors and extensors of the fingers and toes, are surrounded by a fibrous sheath lined by a synovial membrane. Such tendons from their anatomical position are liable to considerable action, and often to external violence.

**Tenosynovitis.**—Inflammation of the tendon sheaths of an acute character occurs either from simple over-exertion, as in the flexor sheaths of the wrist from rowing, or from infection by a punctured wound, as in many cases of whitlow.

*Tendon Sheaths of Wrist and Hand.*—The flexor tendons as they pass deep to the anterior annular ligament of the wrist are covered with a synovial sheath of a large size which is common to all except the flexor longus pollicis. This sheath extends upwards for two fingers' breadth above the upper border of the annular ligament. Downwards it tends to spread out upon the tendons, terminating in three diverticula about the middle of the metacarpal bones of the index, middle and ring fingers, but in the case of the little finger, it is prolonged downwards as far as the base of the terminal phalanx. In addition, the tendons belonging to the three outer fingers have additional and separate synovial linings to their sheaths, starting opposite the heads of the several metacarpal bones, and ending at the bases of the last phalanges. It will thus be seen that there is an interval between the large common synovial sheath in the palm and the special sheaths in these digits.

The flexor longus pollicis tendon has its own sheath lined by synovial membrane, which extends from about one-and-a-half finger's breadth above the annular ligament continuously to the base of the second or terminal phalanx of the thumb. Sometimes this special synovial sheath communicates with the larger common sheath near the annular ligament.

Pricks of the pulp of the digits, as with a needle or pin, are very apt to be followed by septic inflammation, leading to the varieties of whitlow. It is easy to see how a synovial sheath may become secondarily infected from this adjacent inflammation. It is possible that at certain spots, particularly opposite the interphalangeal joints where the fibrous sheaths are thin or even fenestrated, the synovial membrane may bulge and become primarily inoculated with bacteria.

If the theca of the flexor tendons of the little finger is involved, the inflammation may spread directly into the common synovial

sac. If the theca of the tendons of the index, middle or ring finger is the site of the mischief, it may be confined to the digital part, but sometimes it may pass over the gap and involve the common sac. If the theca of the flexor longus pollicis is infected, suppuration may be limited to it, though occasionally pus may find its way into the large palmar sac.

If a thecal abscess is present, flexion of the terminal phalanx of the digit becomes impossible, when the intermediate phalanx is prevented from being flexed by the pressure of the surgeon's finger against its palmar aspect. If the pus is superficial to the sheath, this function of flexion of the terminal phalanx is preserved. An incision to evacuate the pus in a theca should be so planned as to cause the least possible damage to the surrounding structures. It is best to approach the sheath between the head of the metacarpal bone and the first interphalangeal joint on the palmar aspect, and in the median line of the digit.

When incisions are needful higher in the palm of the hand, they must not be carried nearer the wrist than the level of the abducted thumb for fear of wounding the superficial palmar arch, and should be in a line continued upwards from the middle line of the finger.

#### DISEASES OF FASCIÆ.

**Contraction of the Palmar Fascia (Dupuytren's).**—The deep fascia of the palm consists of three portions, the dense central part or the palmar fascia proper, and the lateral portions, much less important, covering the thenar and hypothenar muscles.

The central portion consists of a strong, triangular layer of white fibrous, almost ligamentous, tissue. The apex is attached to the lower edge of the anterior annular ligament, and is continuous with the termination of the palmaris longus tendon, when this is present.

A very large number of fine bands pass from its superficial aspect to become attached to the deep aspect of the skin. When these are shortened, as in Dupuytren's contraction, they tend to

drag inwards small islets of skin and so to form deep narrow pits, into which often nothing larger than the head of a pin can be passed. The deep surface of the fascia is quite smooth, and overlies the tendons.

As the fascia proceeds towards the digits it broadens out, and at the heads of the four inner metacarpal bones it divides into four slips, one of which passes to the root of each finger. Here it is connected with transverse fibres, constituting the superficial transverse inter-metacarpal ligament; it then divides into two, passing on either side of the digits to blend with the fibrous sheaths of the flexor tendons.

Contraction of this central part of the fascia and particularly its inner portion gives rise to "Dupuytren's contraction of the palmar fascia." One or more of the digital processes of the fascia becomes for some reason contracted, and the resulting deformity is characteristic. First, the finger connected with the shortened process is flexed more or less markedly at the metacarpophalangeal joint, with often the terminal phalanx slightly flexed upon the second. Greater flexion of the affected digit can be brought about, but active or passive full extension is impossible. The little and ring fingers are the most frequently and the first to be contracted. Secondly, when forcible efforts are made to extend these fingers, very distinct bands of fascia stand out under the skin of the palm, and the pits which have already been mentioned become increased in depth.

Operative treatment is generally indicated, and may consist of either multiple subcutaneous division of the contracted bands or their exposure and removal by dissection. Strict asepsis is essential for the success of such operative measures, whether open or subcutaneous. The flexion of the fingers, sometimes extreme, and the creases and pits in the skin of the palm render cleansing peculiarly difficult. Thorough scrubbing of the opposed surfaces and the depths of the depressions is almost impossible, so that it is desirable to soak the hand in an antiseptic solution for a lengthened period before operation is undertaken, in order that all parts may be reached by the germicide.



## DISEASES OF BURSÆ.

A bursa is strictly speaking a closed sac wholly lined with synovial membrane. Some sub-tendinous so-called bursæ have direct communications with joint cavities, and are then in reality extensions from the joint and are lined with a prolongation of synovial membrane continuous with that of the joint itself.

**Enlargement of Bursæ.—Bursitis.**—A bursa may become enlarged, usually by over-distension with bursal fluid, without any obvious signs of inflammation, though probably inflammatory influences are the ultimate cause of the increase in secretion. There are certain bursæ which from their anatomical position are peculiarly liable to intermittent pressure or irritation, and are consequently those which are most frequently enlarged.

*Pre-patellar bursa.*—The bursa which is found in the tissue between the skin and the anterior aspect of the patella is more commonly over-distended than any other in the body. This bursa lies over the lower part of the patella and the upper part of the ligamentum patellæ, and is usually called the prepatellar bursa. It is the largest bursal sac which normally exists. Often its interior is divided by crossing bands of fibrous tissue and it is not infrequently multilocular.

Inflammation of this bursa with a large excess of bursal fluid is very apt to occur from kneeling, and particularly in that form of kneeling and leaning forwards with a certain amount of movement of the parts knelt upon, as is occasioned in the process of washing a floor. It is thus that the enlargement is often seen in housemaids, carpet layers and others whose occupation entails such a position. Sometimes the walls of the bursa become very much thickened, and the tumour formed may be practically a solid one, but its position indicates its probable origin.

Excision of the bursal sac is the most satisfactory treatment. This can be accomplished without difficulty or danger, though perhaps warning should be given on two points. The first is that the skin over the front of the knee is not infrequently much

thickened and furrowed in those who kneel, and it requires a considerable amount of care to render it thoroughly aseptic. The second is that when the bursa is very much enlarged it may laterally overlap the capsule of the knee-joint, and dissection on the deep surface of the sac must be carried out with due caution not to open the joint cavity.

Owing also to the exposed position of this pre-patellar bursa, it is liable to be the site of punctured wounds, such as from needles, which may carry infection into the sac. An acute septic bursitis results. This may also follow infection by the blood stream. Pus within the sac of the pre-patellar bursa will be prevented from finding its way through the overlying skin owing to its denseness. Nor will it be able, fortunately, to pass easily into the cavity of the knee-joint because of the resistance of the capsule. It will, therefore, when it transgresses the wall of the sac, become extravasated as it were into the loose subcutaneous cellular tissue about the joint. Sometimes this condition has been mistaken for septic arthritis, but the fact that the patella is buried in the inflammatory material instead of being prominent, and riding on the surface of the fluid, should easily lead to a correct diagnosis. Free evacuation of the pus is necessary, otherwise the overlying skin may slough, being to a great extent cut off from its blood supply.

*Post-ligamental bursa.*—This bursa lies between the posterior aspect of the patellar ligament and the front of the upper part of the head of the tibia. It may be sometimes enlarged from the irritation produced by repeatedly using the knee to fix a piece of wood against a carpenter's bench. The bursa does not communicate with the knee-joint, but in spite of this fact distension may simulate a collection of fluid within the joint. The swelling, however, caused by the bursa only obliterates the lower natural depressions, namely, those on either side of the ligamentum patellæ and not those seen laterally above the patella, all four of which concavities are lost in synovitis of the knee-joint.

*Semi-membranosus bursa.*—This sac is a so-called bursa situated between the tendon of the semi-membranosus and the inner head

of the gastrocnemius, or in some cases more directly between that head and the posterior ligament of the knee-joint. In by far the greater number of cases the bursa communicates with the knee-joint, and there is a great tendency for it to be distended with fluid when there is inflammation of the synovial membrane of this joint. In the flexed position of the knee the bursa becomes less tense, and it is possible to squeeze some of its contents into the cavity of the articulation. In this way the enlarged bursa is said to disappear in this position of the limb. In extension fluid is forced from the interior of the joint into the bursa, which now becomes palpable as a tense cyst lying on the inner side of the posterior aspect of the joint, just external to the tendon of insertion of the semi-membranosus muscle.

The bursa is probably the starting point in many instances of a synovial cyst which later on may be entirely disconnected with the joint, and may enlarge peripherally for some considerable distance. Such synovial cysts are frequently tuberculous in nature.

*Sartorius bursa.*—The bursa placed between the expanded tendinous insertion of the sartorius muscle and the insertions of the gracilis and semi-tendinosus is sometimes the seat of enlargement. The bursa does not communicate with the knee joint.

*Sub-crural bursa.*—This bursa lies between the quadriceps tendon and the anterior surface of the lower part of the shaft of the femur, and is in reality a prolongation upwards of the synovial membrane of the joint. Hence it follows that a distension of the synovial cavity is usually accompanied by a filling-up of this sac with fluid, which may readily be pressed out of the sac into the joint, so as to make the sign of riding of the patella easily obtainable.

It follows from the position of this bursa and the two others which communicate with the knee joint, namely the semi-membranosus and the popliteal, that a punctured wound involving any one of them, although appearing to be at some

little distance from the joint proper, may yet give rise to septic arthritis.

*Ischial tuberosity bursa.*—From prolonged sitting, and particularly when associated with a certain amount of movement upon the seat, the bursa superficial to the tuber ischii becomes chronically inflamed. Hence coachmen, omnibus drivers, weavers with a hand loom, and others with similar occupations are liable to this affection. It is, moreover, not an uncommon site for gummatous disease.

*Trochanter bursa.*—The bursa lying over the trochanter major of the femur may also become the seat of inflammation, particularly in persons who are thin and who have to lie upon hard substances, or who carry a bag which intermittently rubs over the region of the bursa.

*Psoas bursa.*—This lies on the front of the hip-joint beneath the psoas and may communicate with the articular cavity.

*Olecranon bursa.*—The bursa normally found over the lower part of the olecranon process is apt to become irritated and thus inflamed in persons following certain occupations, such as packers and miners. Effusion into this bursa produces a swelling in the middle line posterior to the bone, and obscures the olecranon, instead of the swelling on either side of this process as in synovitis of the elbow. But it is perhaps more commonly the site of acute septic inflammation from punctured wounds, the outcome of falls upon the point of the elbow. In these cases long-continued suppuration, and perhaps even necrosis of the bone, may result unless free discharge of the pus is provided for.

*Sub-deltoid bursa.*—This bursa is situated between the deltoid and the capsule of the shoulder joint. It may become enlarged as the result of chronic inflammation, and the swelling thereby caused may be mistaken for inflammatory mischief in the shoulder-joint. The position of the bursa, however, indicates that it is unlikely that any fulness can be felt or seen in the axilla, whilst the deltoid appears much more stretched and tense. It should also be remembered that distension of the

shoulder-joint, even when inflammation is present, is rarely well marked.

In addition to the above natural bursæ, certain exposed bony points which are prone to receive intermittent pressure may have adventitious bursæ developed over them. The most common example of this is the bursal sac which forms over the inner side of the head of the first metatarsal bone and the first metatarso-phalangeal joint, particularly in cases of hallux valgus, constituting what is ordinarily called a bunion. Chronic or acute inflammation of this bursa is by no means infrequent. If suppuration occur, pus may find its way into the subjacent joint and lead to acute septic arthritis.

**Hæmorrhage into Bursæ.**—Bursal sacs may become distended with blood as the result of falls or blows upon them. This is particularly liable to occur in the case of the pre-patellar and the olecranon bursæ.

#### TALIPES.

**Flat-foot (Talipes valgus).**—Flat-foot is a very common complaint. The longitudinal arch of the foot is made up of a posterior and an anterior pillar. The posterior is the shorter and more vertical of the two, and consists of the whole of the os calcis and all of the astragalus except the anterior part of its head, namely that portion which is received into the cup-like depression on the posterior surface of the navicular. The anterior pillar consists of the tarsus and metatarsus, but readily falls into two portions, an inner, made up of the navicular, the three cuneiforms and the three inner metatarsals, and an outer composed of the cuboid and the two outer metatarsals. The phalanges take no share in forming the arch.

The conformation of the bones, particularly the articulating surfaces of the head of the astragalus and the posterior facet of the navicular, is one of the factors in the preservation of the longitudinal arch.

In close contact with the bones are the ligaments, the second

factor in the maintenance of the arch. Of these the important are,—(1) The inferior calcaneo-navicular, attached behind to the front of the sustentaculum tali and in front to the under and inner surface of the tubercle of the navicular. This ligament underlies and therefore supports the head of the astragalus, the only part of that bone which can in any way be termed the keystone of the arch. The ligament contains much elastic tissue, and is often termed the “spring” ligament. (2) The long plantar ligament, attached behind to the under surface of the os calcis and in front to the margins of the groove on the under surface of the cuboid, in front of the tuberosities, and the bases of the third, fourth and fifth metatarsals. (3) The short plantar ligament, attached behind to the front of the under surface of the os calcis and in front to the under surface of the ridge forming the posterior boundary of the groove on the inferior aspect of the cuboid. (4) The anterior part of the internal lateral ligament of the ankle joint.

The third factor in maintaining the arch is formed by the tendons of certain of the leg muscles. Of these the most important are those of the tibial muscles.

The tibialis posticus tendon passes in a groove behind the internal malleolus, then across the inner surface of the sustentaculum tali to the under and inner surface of the tubercle of the navicular, superficial to the inferior calcaneo-navicular ligament. The tendon also sends expansions backwards to the sustentaculum, outwards to the cuboid and forwards to the bases of the second, third and fourth metatarsals. It will thus be seen that this muscle is a most powerful supporter of the longitudinal arch of the foot, particularly from its position in reference to the spring ligament, and through it to the head of the astragalus. The tibialis anticus tendon lies anterior to the lower end of the tibia, and crossing inwards on the dorsum of the foot runs to be inserted into the inner and under surface of the internal cuneiform and base of the first metatarsal. Both of these tendons come out into prominent relief when a person stands over on the outer edge of the foot. In addition to these two

FLAT-FOOT.

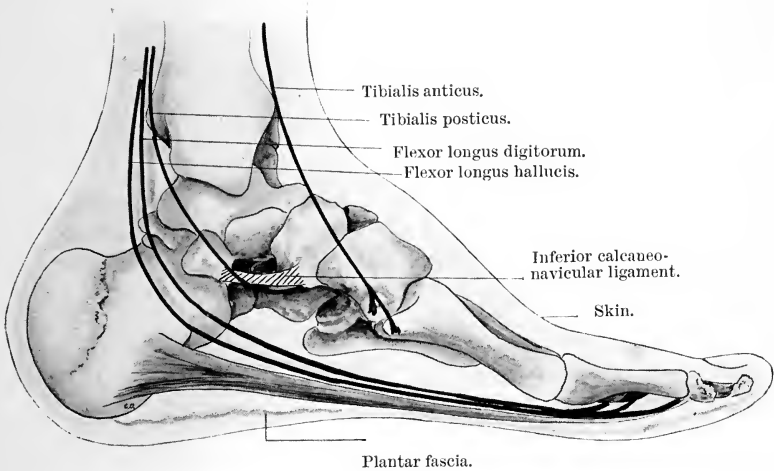


FIG. 8.—Diagrammatic representation of structures maintaining the longitudinal arch of the foot.





tendons, those of the flexor longus digitorum and the flexor longus hallucis aid in supporting the arch.

The next factor is constituted by certain of the short muscles of the sole of the foot, especially the abductor hallucis on the inner side and the flexor brevis digitorum in the middle. After this the strong plantar fascia bears its share in the support; and lastly, the skin also takes a part.

To recapitulate the factors which maintain the longitudinal arch, they are:—the bones, certain ligaments, the tendons of some of the long muscles of the leg, certain short muscles of the sole of the foot, the plantar fascia and the skin. (Fig. 8.)

The pain felt in many cases of flat-foot is mostly due to the pressure exerted on the nerve-terminations in the ligaments which are subjected to stretching, it being a well-known fact that although ligaments and tendons may be cleanly divided with but little discomfort, yet longitudinal tension of the same leads to severe pain. When the bones constituting the longitudinal arch have become so displaced as to no longer cause tension of the ligaments, rigidity results and pain subsides almost entirely.

In flat-foot an extra bony point becomes prominent on the inner side of the sole of the foot. Normally the internal malleolus, the tuberosity of the navicular, and the base and head of the first metatarsal are the bony points readily discernible. When the arch is lost the head of the astragalus forces its way into prominence between the internal malleolus and the tuberosity of the navicular, the front of the foot being abducted and, as it were, partially dislocated outwards at the astragalo-navicular articulation.

The transverse arch is most marked at the line of the tarso-metatarsal joints, and is chiefly caused by the wedge shape of the three cuneiform bones and of the bases of the metatarsals. The tendon of the peroneus longus, and possibly the adductor obliquus hallucis and the transversus pedis (adductor transversus hallucis) muscles help to maintain the transverse arch.

**Congenital Talipes Equino-varus.**—Equino-varus is the most

common compound or secondary form of club-foot occurring congenitally. The heel is raised, and the foot, particularly in front of the transverse tarsal joint (that is, the combination of the two joints between the astragalus and os calcis posteriorly and the navicular and cuboid anteriorly) is turned inwards. The tendons which are taut are the tendo Achillis behind, inducing the equinus, and the tibialis anticus, tibialis posticus, together with the flexor longus digitorum and flexor longus hallucis on the inner side, inducing the varus. On the outer aspect of the foot the anterior end of the os calcis becomes very evident, and not infrequently the external surface of the neck of the astragalus can be readily felt. The head of the astragalus looks somewhat inwards, because the neck is markedly elongated on its outer aspect and shortened on its inner. The external malleolus is prominent, while the internal malleolus may be somewhat buried. A well marked vertical furrow may be seen on the inner side of the foot, indicating the transverse axis at which adduction and internal rotation has occurred, while on the outer side after the child has commenced to walk, a callosity and even an adventitious bursa may be formed.

It will thus be seen that in the treatment of these cases, if the tendons which are contracted can be stretched and the anterior part of the foot abducted and externally rotated, a cure may be effected. Forcible manipulation and massage commenced immediately after birth will in a large number of cases lead to a restoration of position by obtaining the above results. The equinus as a rule is more difficult to correct than the varus by these manipulative procedures.

Should the case, however, be one in which the correction of the deformity has been imperfect, or, if the patient is not seen until the parts have become considerably fixed, certain tenotomies are needful. The tendons most commonly requiring division are those of the tibialis anticus and posticus and the tendo Achillis. It is desirable that the division of these tendons should be done in the following order:—the tibial tendons first and the tendo Achillis last, so that after division of the tibials the tendo Achillis

may still act as a point from which manipulation may be undertaken to cure the varus.

The place at which division of the three tendons should be carried out must be carefully noticed. The tibialis anticus may be conveniently divided close above its insertion into the inner and plantar aspect of the internal cuneiform and base of the first metatarsal and therefore below the ankle joint. The tibialis posticus can be satisfactorily attacked immediately behind the internal border of the tibia, at the base of the internal malleolus. The tenotome placed between the bone and the tendon cuts, backwards and inwards, and may easily at the same time divide the flexor longus digitorum. There is not the least disadvantage in so doing, provided the knife does not go further and injure the posterior tibial artery immediately posterior to the tendon of the long flexor of the toes. Behind this again will be found the posterior tibial nerve and posterior to it the flexor longus hallucis tendon; but none of these structures should ever really be in danger in tenotomy of the posterior tibial tendon.

The section of the tendo Achillis is best performed where that structure is rounded, namely, a little below the level of the tip of the internal malleolus. A puncture is made with a sharp tenotome from the inner side in front of the tendon, care being taken not to insert the point of the instrument into the substance of the tendon, which is decidedly thick at this spot. A blunt tenotome is then introduced, and its cutting edge directed towards the anterior surface of the tendon. Whilst the last fibres are being divided, and particularly the moment when they give way and the foot is released, the skin lying posterior to the tendon is very apt to be cut through, an accident which may lead to disastrous results.

Even after the division of these tendons it may be found that full correction is impossible, mainly owing to the contraction of other tissues on the inner side of the foot, including skin, fascia, the abductor hallucis and even the ligaments. It may, therefore, become necessary to divide all these constricting structures down to the bones themselves, but as a rule such

a proceeding should not be carried out without an accompanying tarsectomy.

A cuneiform tarsectomy for varus has the base of the wedge looking outwards and somewhat upwards. The section as a rule passes through the anterior part of the os calcis and the head of the astragalus posteriorly; through the joint between the cuboid and the fourth and fifth metatarsals, across the base of the external cuneiform and through the navicular anteriorly. Therefore the parts removed will consist of portions of the anterior part of the os calcis and the astragalus, the whole of the cuboid and parts of the external cuneiform and the navicular. In the young subject it must be remembered that all this mass is to a very great extent cartilaginous.

**Acquired Talipes Equino-varus.**—Infantile paralysis is the most common cause of acquired varus or equino-varus. In this condition the deformity is brought about by the active contraction of the unopposed non-paralysed muscles and the subsequent shortening of the muscular tissue between the approximated points of their origin and insertion. Hence it comes about that a deformity will result with rigidity of the neighbouring joints. In order that equino-varus should occur, paralysis of the anterior tibial group is necessary—that is to say, paralysis of the tibialis anticus, extensor proprius hallucis, extensor longus digitorum and peroneus tertius, supplied by the anterior tibial nerve. In some cases, moreover, the peroneus longus and brevis may be likewise affected. Seeing that anterior tibial paralysis is the most common form met with as a result of anterior poliomyelitis, it follows that equino-varus is the usual variety of deformity that ensues.

In cases, however, where paralysis involves both the anterior and posterior tibial groups, it is extremely unlikely that contraction and rigidity will occur, but laxity and flail-like joints will result.

In those cases in which one group of muscles alone is paralysed, an interesting anatomical question arises in connection with the method of treatment by tendon-grafting or tendon-transplantation. In cases of equino-varus resulting from paralysis

of the anterior tibial group, it may be possible to bring a part of the tendon of the active tibialis posticus forwards around the inner side of the tibia, and to graft it into the tendon of the paralysed tibialis anticus. Supposing that the peroneus longus retains its activity, part of its tendon may be carried across the antero-external surface of the leg above the external malleolus, and fixed to some or even all of the tendons of the paralysed extensor group. On the other hand, should the peronei have lost their function, the outer part of the tendo Achillis may be fixed to them.

**Talipes Calcaneo-valgus.**—It is uncommon to get pure talipes calcaneus or pure talipes valgus (if flat-foot is excluded), and not very common even to see the combination of the two.

The position of the foot in calcaneo-valgus is that the anterior portion of the foot is dorsi-flexed, abducted and somewhat externally rotated.

In acquired cases from infantile paralysis, the contracted tendons are those of the anterior tibial and peroneal group, particularly the peroneus longus and brevis and the extensor longus digitorum. In the operative treatment by tendon-grafting, a portion of the active tibialis anticus tendon may be grafted into the paralysed tibialis posticus, and the whole of the peroneus longus spliced into the tendo Achillis.

## CHAPTER XI.

### DISEASES OF THE NERVOUS SYSTEM.

#### THE BRAIN AND ITS MEMBRANES.

##### MENINGITIS.

**Suppurative meningitis** may be local or diffuse. The common form is a diffuse leptomeningitis involving the base or the convexity of the brain or even the whole area of the subarachnoid space. The wide extent and loose meshwork of this space especially favour the diffusion of the suppurative process. On the convexity of the brain the pus at first lies in the sulci where the spaces between pia and arachnoid are larger than over the convolutions.

The meningitis is always associated with encephalitis because the lymphatic sheaths of the pial vessels carry the infection to the superficial layers of the brain substance. The interior of the brain may also be invaded by extension along the pia and arachnoid where they become invaginated into the cerebral ventricles at the great transverse fissure.

The infection of the meninges may be direct or through the medium of arteries, veins, or lymphatics.

Direct infection is the result of open fractures of the vault of the skull, of perforating wounds or of basal fractures which involve the cavities of the nose, the naso-pharynx or the ear. Infection through the medium of the arterial stream occurs in certain general infective diseases of which pneumonia may be taken as an example, and also in pyæmia. The emissary and some of the diploic veins of the skull may carry suppurative organisms to the sinuses and so to the membranes. In some cases the lymphatic sheaths surrounding the arteries which enter

the skull or the sheaths which surround the cranial nerves may serve as channels of invasion.

Acute meningitis is often secondary to disease of the ear or nose ; it may also follow disease of the air sinuses of the skull, the orbit, globe of the eye or brain itself.

From the ear there are several paths of invasion. The canal of the facial nerve affords a direct channel from the tympanum, when its bony casing is incomplete. In disease of the cochlea the sheath of the auditory nerve may convey the infection. The veins of the tympanum may infect the superior petrosal or lateral sinuses, and in infants and young persons the petro-squamous suture may be permeable. Defects in the tegmen tympani may afford a direct path for infection of the overlying membranes, but here a subdural or temporo-sphenoidal abscess occurs rather than leptomeningitis, since the dura, arachnoid and pia all lie in the closest contact in this region.

The free communication of the perineural lymphatics of the olfactory nerve filaments with those of the nasal mucous membrane explains the occurrence of leptomeningitis in connection with nasal disease. Caries of the ethmoid will afford a direct path to the membranes or the bone may be perforated during operations in this region. Disease of the frontal or ethmoidal sinuses may spread directly to the meninges in the neighbourhood.

Suppurative disease of the orbit may extend to the interior of the skull along veins or lymphatics and suppuration in the globe of the eye may spread back to the membranes along the sheath of the optic nerve.

Abscesses near the surface of the brain can infect the pia and arachnoid directly, and a localised leptomeningitis also may be found overlying superficial cerebral new growths.

**Posterior basic meningitis** is due to infection of the walls of the great subarachnoid cisterns which lie at the base of the brain and around the pons and medulla. The inflammation extends through the transverse fissure to the interior of the ventricles and may also involve the membranes of the cord by continuity. The infective agent is probably carried to the meninges

in the first place by the blood stream, but it has been suggested that the primary invasion may be from the nasopharynx along the perineural and perivascular lymphatic sheaths or by way of the Eustachian tube to the tympanum and thence to the meninges by veins which traverse the petro-squamous suture if that be still open. If this suture be closed there are still other efferent veins from the tympanum which may carry infection.

The subarachnoid space communicates directly with the tubular investment of the auditory nerve and so with the perilymph spaces of the internal ear, but it does not communicate with the endolymph spaces or with the tympanum. Consequently it is improbable that infection travels to the meninges from the middle ear along the auditory nerve sheath. Infection, however, may and does travel in the reverse direction, and in this way otitis interna sometimes develops secondarily to the meningitis, but the otitis media of posterior basic meningitis is more commonly due to infection along the Eustachian tube from the nasopharynx of a stuporous patient.

In early cases of posterior basic meningitis the subarachnoid cisterns are filled with a greenish exudation so that their extent and connections become quite obvious. These cisterns are formed by the wide separation of the pia and the arachnoid at certain spots, whilst in other positions these membranes are closely connected by a very small meshed areolar bond.

At the base of the brain a sheet of arachnoid passes between the under surfaces of the temporal lobes near their inner margins, and by bridging over the interpeduncular space forms the cisterna basalis. This basal cistern is prolonged laterally along the Sylvian fissures and anteriorly round the front of the corpus callosum into the great longitudinal fissure. Posteriorly the cisterna basalis is continuous with the cisterna pontis, which at its fore part has lateral prolongations around the crura cerebri leading to the large angular interval which lies dorsally between the front of the cerebellum and the corpora quadrigemina. The cisterna pontis is continued behind into the tubular investment of the medulla. This investment when viewed from below is





MENINGITIS.

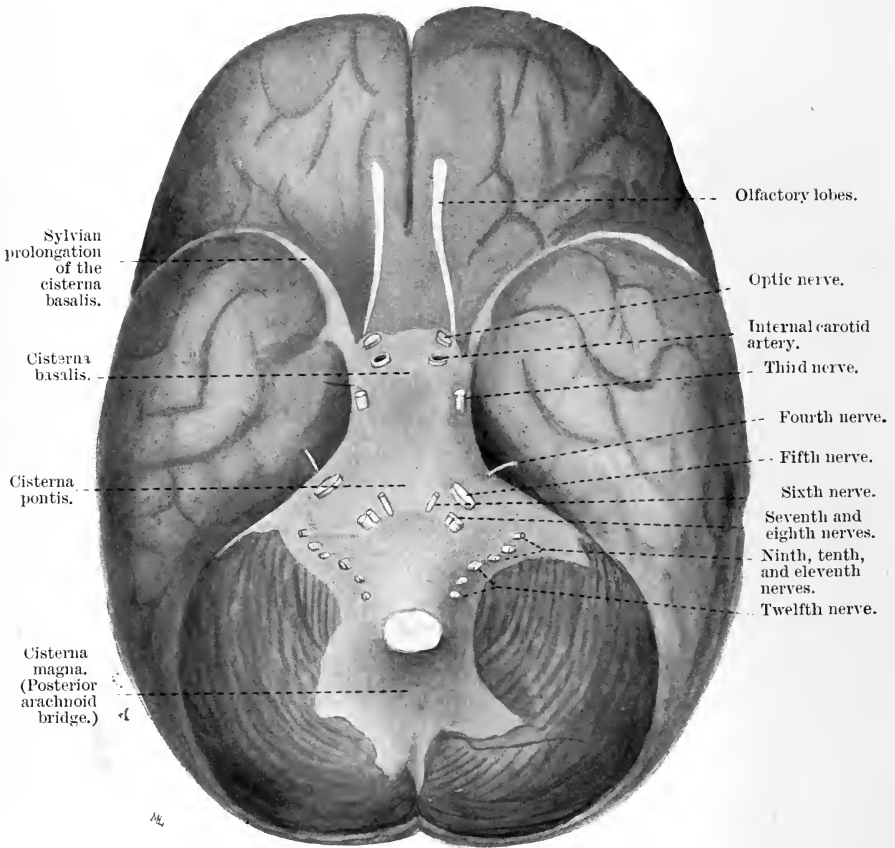


FIG. 9.—Suppurative meningitis, showing the outlines of the infected arachnoid cisterns and the positions in which the cranial nerve trunks appear on the surface of the arachnoid mater.

seen to be limited by fusion with the pia on the under surface of the cerebellum some distance away from the sides of the medulla and pons, whilst it becomes abruptly narrowed at the lower part of the medulla where it is continued as the arachnoid sheath of the spinal cord. The dorsal part of the arachnoid sheath of the cord is continued over the dorsal part of the medulla as far as just within the foramen magnum and then turns back rather abruptly to fuse with the under surface of the cerebellum. It thus forms the posterior boundary of the cisterna magna, which occupies the angular interval between the medulla and lower part of the cerebellum. The sheet of arachnoid just described as passing from the medulla to the cerebellum is sometimes called the posterior arachnoid bridge, and by careful removal of this the openings in the pial covering of the fourth ventricle can be investigated. This pial covering forms the anterior boundary of the cisterna magna which, like the other cisterns, is a space between the pia and arachnoid. (See Fig. 9.)

The great blood-vessels of the brain penetrate the arachnoid and then lie in the subarachnoid cisterns, whilst sheaths of arachnoid tissue are prolonged along the issuing nerves. Irritation of the first cervical nerve where it traverses the subarachnoid space just below the medulla may account for the rigidity of the neck and retraction of the head which are such a marked feature in posterior basic meningitis. This nerve supplies the short muscles which pass between the atlas and axis and the occiput, and also the overlying complexus, whilst the next one or two cervical nerves supply the splenius and the trachelo-mastoid. It is said that the rotatory movements between the head and spine are usually retained, *c.* at all events not resisted. It is also possible that inflammatory irritation of the superior vermis of the cerebellum and the region of the corpora quadrigemina accounts for the retraction and opisthotonos which may occur. At the transverse fissure of the brain, which lies immediately above the corpora quadrigemina, a fold of pia mater with a basis of arachnoid tissue, forming the velum interpositum, becomes invaginated; this affords a track by which inflammation spreads to the cavities of the ventricles.

When the spinal theca is invaded, symptoms due to spinal nerve irritation may be met with. One such symptom is an increased irritability of the hamstring muscles, and is the basis of Kernig's sign. Even under normal conditions the length of the hamstrings is insufficient to allow the knee to be fully extended when the thigh is flexed on the abdomen, but in spinal meningitis and some other conditions the range of extension is still further limited. Care must be taken, when eliciting the sign, that the other thigh is kept in contact with the bed, lest flexion of the pelvis on the spine conceal an imperfect flexion of the thigh on the abdomen and so allow fuller extension of the knee. If the greatest possible angle made by the back of the leg with the back of the fully flexed thigh be less than a right angle and a half, Kernig's sign may be considered to be present.

**Hydrocephalus** is usually secondary to posterior basic inflammation. Sometimes it can be definitely attributed to obstruction of the channels of communication between different parts of the ventricular system. Such obstruction has been found at the foramina of Monro causing distension of one or both lateral ventricles; in the iter, when the third ventricle is also distended; completely closing in the fourth ventricle, when the foramina of communication between the ventricles and the subarachnoid space are of necessity blocked; or in the cervical region, just below the foramen magnum, completely cutting off the spinal from the cerebral subarachnoid space. The velum interpositum may be matted to the edges of the transverse fissure, but the veins of Galen are rarely occluded, so their influence in causing hydrocephalus is problematical.

Hydrocephalus often exists without any obstructions such as have been mentioned. It is then due to inflammations of the ventricular cavities and can be drained by lumbar puncture, the fluid passing from the fourth ventricle into the space between the arachnoid and pia of the cord. The spinal cord terminates in the adult at the lower border of the first lumbar vertebra, but in the child extends to the third. The subarachnoid and subdural spaces reach to a point opposite the middle of the second

sacral vertebra. Hence at all ages lumbar puncture may be effected between the laminae of the third and fourth or fourth and fifth lumbar vertebræ without risk of injury to the spinal cord. The drainage is usually facilitated by flexion of the head during the tapping since this movement opens up the angle between the lower aspect of the cerebellum and the fourth ventricle, and so allows free exit of the fluid. The subdural space has no communication with the subarachnoid space and the ventricular cavities, but in the treatment of hydrocephalus an artificial communication is sometimes made in the hope that the vessels of the dura may dispose of the excess of fluid which has accumulated in the ventricles and subarachnoid space.

**Tuberculous meningitis** is in most instances a secondary infection, the bacilli being carried to the leptomeninges in the blood stream. A primary focus is commonly found in the mediastinal glands, but sometimes the infection is derived from the mesenteric glands, the lungs, bones, joints, or generative organs. Occasionally direct infection of the brain substance can be traced from the petrous bone, or the bones of the nasal cavity.

The meningeal tubercles are found in the pial sheaths of the cerebral vessels; they chiefly accumulate in the walls of the basal cistern of the subarachnoid space and in its Sylvian prolongations, and so follow the course of the middle cerebral arteries. A moderate degree of hydrocephalus with consequent flattening of the convolutions co-exists, the ventricles being invaded along the velum interpositum which is invaginated into the brain at the great transverse fissure. The inflammation is carried from the meninges to the surface of the brain along the perivascular sheaths of the small vessels passing into the cortex from the pia, and a moderate amount of diffuse softening is thus produced. Similar inflammatory softening is also present around the ventricular cavities. In some instances the inflammation of the pia and arachnoid is sufficiently intense to cause occlusion of larger arterial branches which lie in the subarachnoid spaces and then definite areas of softening may be found in the central ganglia and sometimes in the cortex. In such cases the ganglia

suffer most because their circulation is absolutely terminal and without anastomosis, whilst the amount of cortical anastomosis varies in different individuals.

The symptoms of tuberculous meningitis correspond, in the main, to the visible distribution of the lesions. The cranial nerves of that part of the base affected usually suffer fairly early in the disease; the third, sixth and seventh are most commonly picked out; and squints, ptosis, and facial paralysis of the complete nuclear type result. Partial facial paralysis of the cortical type is due to extension of the inflammation along the Sylvian fissures and towards the convexity of the hemispheres. The prodromal aphasias and mental disturbance are attributed to cortical œdema which may be transitory. Later on convulsions, coarse tremors and rigidities point to cortical invasion, as also do paralysees of hemiplegic or monoplegic distribution.

Head retraction is usually moderate and caused by extension of inflammation to the posterior part of the base. The coma may be explained by increasing ventricular distension. Thrombosis of the cerebral veins and sinuses is rare; since the veins of the cortex, unlike the arteries, communicate freely.

Tuberculous spinal meningitis is usually secondary to or associated with meningitis of the cerebral meninges, the membranes of the brain and cord being directly continuous. Like the cerebral form, the spinal variety is a leptomeningitis accompanied by effusion into the space between the pia and the arachnoid. Owing to the dorsal posture assumed in the later stages of the cerebral lesion, the morbid changes are best marked on the dorsal aspect of the cord, and the posterior sensory nerve roots are especially apt to suffer. The peripheral zone of the cord itself may be invaded along the paths afforded by the pial septa, the anterior and posterior roots, and the vessels which enter the margin of the cord from its pial investment.

Diffuse **syphilitic meningitis** usually originates in the pia and arachnoid as a rich cellular infiltration. The lesion, however, is not limited to the leptomeninges, but spreads along the vessels of the pia to the brain tissue on the one hand and to the adjacent

dura on the other. The disease, therefore, presents itself as a diffuse gummatous meningo-encephalitis and the resulting cranial nerve paralysees may be associated with symptoms referable to lesions of the crura, pons or medulla. These parts of the cerebro-spinal axis may be compressed by the infiltrated membranes or their nutrient vessels may become occluded and softening result. Under these circumstances various forms of true "crossed-paralysis" may present themselves, the cranial nerve paralysis being on the opposite side to the hemiplegia. Crossed paralysis may also be simulated; the paralysis of the cranial nerve and the hemiplegia being caused by two separate and distinct foci of disease, indeed syphilitic meningitis is characterised by its erratic distribution as well as by its tendency to spontaneous remission and relapse.

The vertex is sometimes the seat of the disease and the exposure of the head to blows may appear to favour manifestations in this locality. If the meningitis lies over the precentral convolution, Jacksonian epilepsy may be expected. Syphilitic meningitis is, however, most characteristically seen at the base of the brain, especially in the region of the optic chiasma and between the crura cerebri. The relations of the cranial nerves to each other as they lie in the membranes and to the three fossæ of the base of the skull are of importance in the diagnosis and localisation of the mischief. The relations which different nerve trunks bear to each other on their course through the membranes to their foramina of exit is in several instances very different from the relations of the nuclei of the same nerves in the cerebro-spinal axis, and thus disease in the meninges may be differentiated from disease within the brain. An accurate knowledge of the relations of the nerves to the different fossæ of the skull is of further assistance in localisation of the lesion.

In the anterior fossa lie the olfactory nerves; to the middle fossa belong the fifth and sixth nerves, with those above them, except the olfactory; to the posterior fossa belong the fifth and sixth nerves and those below them. The fifth nerve may be said to lie at the junction of the middle and posterior fossæ, its stem of origin

from the pons belongs to the fore part of the posterior fossa, whilst the Gasserian ganglion and the three subdivisions of the nerve belong to the middle fossa.

In the middle fossa the third, fourth and sixth nerves may be involved where they are concentrated in the cavernous sinus or at the sphenoidal fissure, and in such case the optic nerve is also involved as a rule. Affection of the optic chiasma may cause hemianopia.

The fifth nerve may be picked out by itself as also may one of the ocular nerves. Since the sixth nerve where it pierces the dura is nearer to the fifth than to any other cranial nerve the two may suffer together.

In the posterior fossa the fifth and seventh nerves may be paralysed together; or the fifth with the ninth, tenth, eleventh and twelfth; or the seventh and eighth. Paralysis of the vago-accessory and hypoglossal nerves, producing unilateral paralysis of palate, tongue and soft palate, is also characteristic of a lesion at the base, outside the medulla and in the region of the jugular and hypoglossal foramina. Or the hypoglossal nerve and the spinal portion of the spinal accessory nerve may be damaged in the membranes near the foramen magnum, producing an associated paralysis of the trapezius, sterno-mastoid and corresponding half of the tongue.

Special combinations of nerves pointing to basic as opposed to pontine lesions are fifth and sixth, seventh and eighth, eleventh and twelfth. The nuclei of origin of these pairs of nerves are more widely separated than their trunks are where they lie at the base of the brain. But the intimate relation of the seventh and eighth nerves in the petrous bone renders exclusion of disease of the ear necessary before the inference of a meningeal affection is drawn from their associated paralysis.

#### CEREBRAL HÆMORRHAGE.

The corpus striatum and its immediate neighbourhood are by far the most common situations of hæmorrhage into the substance





VASCULAR SUPPLY OF CENTRAL GANGLIA AND INTERNAL CAPSULE.

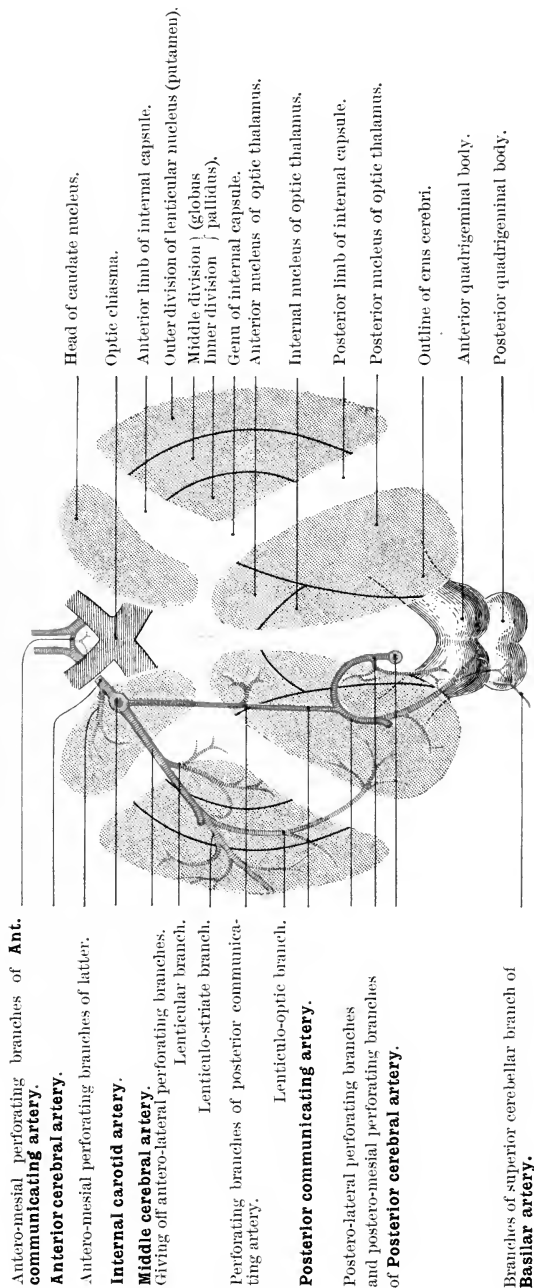


Fig. 10.—Diagrammatic representation of the central distribution of the antero-mesial, antero-lateral, postero-mesial and postero-lateral perforating arteries.

of the brain. Hæmorrhage may also occur in the centrum ovale, the cortex, the pons, and the medulla. The right and left cerebral hemispheres are equally liable, the vascular arrangements being identical. There are several reasons why the arteries of the central ganglia should rupture more frequently than those of the other parts. The arteries are short and come off directly from the large main stem, they are terminal vessels with no collateral circulation to relieve the pressure in them, and they receive poor support from the surrounding grey matter. The pressure in these central vessels is probably nearly as great as the pressure in the internal carotid at their point of origin. (See Fig. 11, p. 166.)

The actual starting point of the hæmorrhage in the *corpus striatum* depends on the vessel which gives way. A clot in the head of the caudate nucleus is derived from the perforating branches of the anterior cerebral artery; hæmorrhage into the middle part of the nucleus comes from the lenticular or lenticulo-striate branches of the middle cerebral artery; hæmorrhages in the posterior part are derived from the lenticulo-optic branches of the same main trunk. The lenticulo-striate artery from the frequency with which it is the source of the bleeding has been called the artery of cerebral hæmorrhage. This vessel passes upwards from the anterior perforated spot through the putamen or outer zone of the lenticular nucleus to terminate in the internal capsule and the body of the caudate nucleus. Extravasations of blood from this artery lie immediately outside the motor section of the *internal* capsule on which they exercise pressure or into which they plough their way, producing hemiplegia. They also tend to separate the putamen from the *external* capsule since anatomically there is but little adhesion between these two structures. Hæmorrhage from the lenticulo-optic branch of the middle cerebral artery is in a position to cause pressure on the posterior or sensory portion of the capsule. (Fig. 10, p. 165, Fig. 12, p. 179.)

The origin of cerebral hæmorrhage may be in the *optic thalamus* instead of in the *corpus striatum*. The lenticulo-optic artery may be responsible for hæmorrhage here also, and its

relation to the sensory portion of the capsule has just been indicated. Branches derived from the posterior cerebral artery help to supply the outer part of the thalamus, and these may give rise to bleeding which also causes pressure on the sensory part of the capsule, and may extend either towards the third ventricle or downwards into the crus. The posterior cerebral and posterior communicating arteries supply branches to the ventricular portions of the optic thalamus, and hæmorrhage from these may easily break into the ventricles. (Fig. 10, p. 165.)

A large hæmorrhage in the *white matter* of the hemisphere has usually spread into it from the corpus striatum. When primary hæmorrhages occur in the white matter they are usually small and of oval outline, being compressed into this form by the general direction of the nerve fibres among which they lie. Branches of the calcarine arteries from the posterior cerebral trunk are the sources of hæmorrhages in the white matter of the occipital lobe close to the visual centre.

Large hæmorrhages in the hemispheres tend to burst into the ventricular cavities rather than on the surface. They travel in the direction of least resistance. Having gained access to the lateral ventricle the blood passes from one cavity to another through the natural communications, and may ultimately reach the fourth ventricle and exercise pressure on the important bulbar nuclei or induce glycosuria. Blood from the ventricles may appear outside the brain by traversing the transverse or the choroidal fissures, or may burst through the floor of the third ventricle, or in some cases invade the pituitary body. The blood when it appears on the brain surface infiltrates the subarachnoid space and may form a thin coating over the cerebellum and other parts.

Branches of the posterior cerebral trunk and of the posterior communicating artery supply the *crus*, but are seldom responsible for primary hæmorrhage into its substance. The crus may also be infiltrated from above by a hæmorrhage which has ploughed up the basal ganglia or from below by a hæmorrhage extending from the pons. Crossed paralysis or alternate hemiplegia

CEREBRAL ARTERIES.

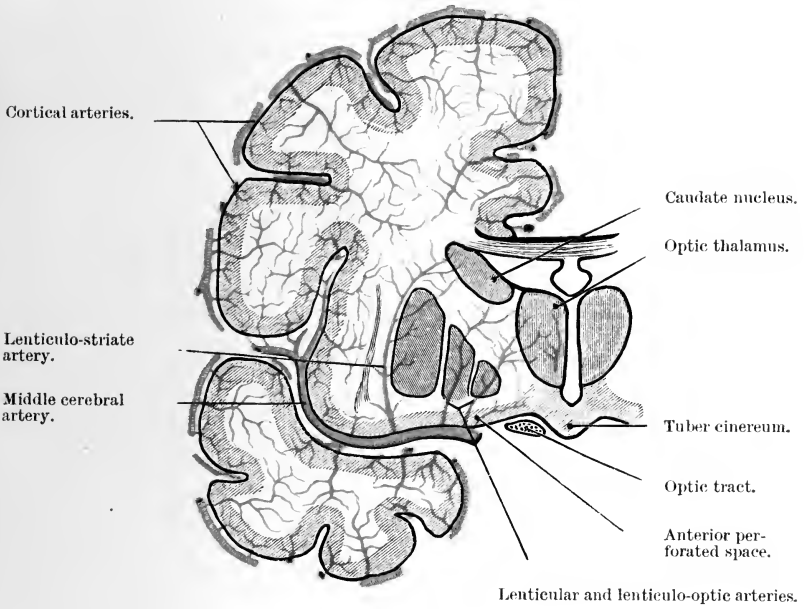


FIG. 11.—Diagram showing the manner of distribution of the cortical and central branches of the cerebral arteries. (From Morris's Anatomy, after Arthur Robinson.)



involving the third nerve of one side and the opposite half of the body is characteristic of a lesion in the crus. This form of paralysis may, however, be mimicked by multiple lesions elsewhere, and also produced by pressure on the crus from without by tumours.

The substance of the *pons* is dense and resistant. This part of the cerebro-spinal axis consists of interlacing fibres, some longitudinal and some transverse, moreover, it is provided with a central raphe. Hæmorrhages are therefore small, and may be confined to one side. The extravasated blood may travel out in the middle cerebellar peduncle towards the cerebellum, or as mentioned before, up in the lower portion of the crus. Each half of the pons is supplied by median branches of the basilar artery and by radicular branches which pass in along the nerves. The radicular branch which is most liable to give way is the branch to the large trunk of the fifth nerve. The proximity to the pons of the ocular nuclei, the presence in it of the nuclei of the fifth and seventh nerves as well as the fact that it transmits the motor and sensory tracts account for the important symptoms which are associated with pontine hæmorrhage. Hæmorrhages which encroach on the dorsal part of the pons may rupture into the fourth ventricle.

Hæmorrhage into the *cerebellum* is said to come from that branch of the superior cerebellar artery which supplies the dentate nucleus. The superior cerebellar artery is itself a branch of the basilar. Cerebellar hæmorrhage may rupture into the fourth ventricle or spread over the surface of the cerebellum beneath the pia and arachnoid.

Hæmorrhage into the *ventricles* is usually secondary to hæmorrhage into other parts of the brain. Occasionally primary hæmorrhage occurs, and may be derived from the arteries which supply the wall of the ventricles. These arteries are mainly branches of the posterior cerebral vessels, but the anterior choroidal branches of the internal carotid also take part. It is possible that ventricular hæmorrhage is sometimes venous, being derived from the striate veins.

## MENINGEAL HÆMORRHAGE.

Meningeal hæmorrhage is called extra-dural when the blood is effused between the skull and the dura mater ; sub-dural when it lies between the dura and the arachnoid ; sub-arachnoid when it lies between the arachnoid and the pia.

*Extra-dural* hæmorrhage is usually due to traumatic rupture of the middle meningeal artery, and from its position gradually induces compression of the adjacent motor cortex and by gravitation also comes to compress the nerves of the middle fossa on the side of the hæmorrhage.

*Sub-dural* hæmorrhage may exist by itself or in association with the other varieties. This form of hæmorrhage may spread widely in the subdural space and even surround the medulla or spinal cord. A subdural collection of blood may come from the meningeal arteries or veins, or from the great sinuses as a result of their rupture. A particular variety is termed sub-dural hæmatoma : it arises in connection with head injuries and is also often associated with insanity.

*Sub-arachnoid* hæmorrhage may result from rupture of aneurysms at the base of the brain since the trunks on which these aneurysms occur are contained in the sub-arachnoid spaces. Hæmorrhage from the interior of the brain may also make its way into the same spaces in the manner already described.

## CEREBRAL EMBOLISM.

A constricted or ulcerated mitral valve is the commonest cause of cerebral embolism. Emboli are sometimes derived from clots in the recesses of the left auricle or from the aortic valves, or from an aneurysm of the arch of the aorta.

Clots from the systemic veins, large enough to produce embolic softening of the brain, cannot traverse the pulmonary capillaries, so when embolism accompanies thrombosis of the veins of the uterus, lower extremities, or other parts, there is usually a left-sided endocarditis or an abscess of lung to account for it. A



patent foramen ovale or a defect in the ventricular septum might conceivably allow a clot from one of the systemic veins to reach the brain, but such an occurrence must be exceedingly rare. In certain cases microbial emboli may be carried from the lungs by the pulmonary veins and set up suppuration in the brain.

Emboli are frequently arrested at the points where vessels bifurcate or become suddenly narrowed by giving off large branches. As examples may be cited the internal carotid where it breaks up into its branches near the base of the brain and the division of the middle cerebral artery into a leash of cortical vessels. Branches which form a more or less direct continuation of the main trunk from which they spring are more likely to receive plugs than those which come off at sharp angles. The left common carotid is in a direct line with the first part of the arch of the aorta, whilst the innominate trunk from which the right common carotid is derived comes off from the aorta at a considerable angle, consequently emboli are rather more frequent in the left middle cerebral artery than in the right. Further, an embolus travelling in the internal carotid artery is much more likely to enter the middle cerebral artery than the anterior cerebral, since the former is a direct continuation of the main trunk.

The left vertebral artery, arising from the left subclavian artery where that is distinctly ascending, is a more direct continuation of the subclavian than is the right vertebral artery which comes off the subclavian nearly at a right angle, so the left vertebral artery is much more likely to receive an embolus. Since the vertebral arteries unite to form the basilar trunk, and that in turn bifurcates symmetrically, each of its posterior cerebral branches is equally liable to be plugged.

The cerebellar arteries come off their parent trunks at acute angles and are rarely the sites of embolisms. The basilar artery too is rarely occluded since a clot which can traverse the vertebral artery is unlikely to be arrested in the larger basilar trunk formed by the union of the two vertebrals, but such clots are sometimes entangled at the point of bifurcation of the vessel.

The results of embolism differ in different regions of the brain

owing to variations in the degree of collateral circulation which is possible. When perforating arteries are occluded, softening of the parts they supply is inevitable; on the other hand the anastomosis of cortical branches in the pia mater may be sufficient to maintain the integrity of the cortex.

The position of the anterior communicating artery at the commencement of the anterior cerebral arteries explains how softening of the head of the caudate nucleus may be the only result of a plug in the first part of one of these vessels. When the middle cerebral artery is occluded near its origin softening may be expected in the central ganglia and at the anterior perforated spot. The amount of cortical softening is rarely co-extensive with the distribution of the vessel, and in some cases there may be none. The cortical collateral circulation appears to vary within considerable limits. The distribution of the cortical branches of the middle cerebral artery to Broca's convolution, to the precentral or ascending frontal convolution which contains the motor centres, to the higher visual cortex and to the auditory centre render this artery of great importance. The central or perforating branches of the same vessel supply the neighbourhood of the internal capsule. It may here be noted that the leg centre is in part supplied by the anterior cerebral artery and the posterior part of the internal capsule receives some branches from the posterior cerebral trunk.

The collateral circulation of the cortical branches of the posterior cerebral trunks is usually sufficient to prevent much softening in their area of distribution. These important vessels supply the optic radiation and the occipital visual cortex. They also supply the visual portion of the thalamus, the anterior and, in part, the posterior corpora quadrigemina. The relation of their branches to the posterior limb of the internal capsule has been mentioned. The crus cerebri receives part of its supply from the same source.

The optic tract is supplied by the anterior choroidal branch of the internal carotid. The cerebellar arteries anastomose so freely that, even if they are occluded, any extensive softening is rare.

The size of the communicating branches of the circle of Willis materially influences the results of blocking of the main feeding trunks. If the communicating trunks are healthy and of fair size and the blood pressure remains high, the integrity of the brain may be maintained by an adequate collateral circulation. If the vessels are diseased or the blood pressure is low, as after severe hæmorrhage, serious cerebral symptoms may be expected. Thus cerebral thrombosis, a condition in which the vessels as a rule are extensively diseased, is more serious than embolism of a healthy trunk; also ligature of the carotid after severe hæmorrhage is more likely to induce hemiplegia than when there has been no loss of blood.

The posterior communicating artery may be able to supply sufficient blood to the middle and anterior cerebral arteries of the same side when the carotid artery is blocked in the cavernous sinus, and in this connexion it is important to remember that the right posterior communicating artery is almost invariably larger than its fellow. Abnormal arrangements of the arteries at the base of the brain may be responsible for anomalous distribution of cerebral softening.

#### THROMBOSIS OF CEREBRAL ARTERIES.

Thrombosis of the cerebral arteries is usually the result of pre-existing vascular disease. The arterial disease is in most cases due to syphilis, but occasionally other forms of arteritis produce the same result. Rarely the pressure of tumours or spread of inflammation from the meninges causes vascular occlusion. The large vessels at the base of the brain, such as the basilar artery and the middle or posterior cerebral arteries, are most likely to suffer. These vessels lie in the subarachnoid cisterns and their prolongations, have but little external support, and the pressure in them is high. The perforating arteries to the interior of the brain are given off from the large basal vessels and do not anastomose, hence cerebral softening is an inevitable consequence of the occlusion of these small branches. Owing to the proximity

of the vital centres of the bulb to the vertebral and basilar arteries, thrombosis of these vessels is a more serious menace to life than occlusion of the middle or posterior cerebral trunks. Thrombosis of cortical branches may produce slight or transient symptoms, since there is usually a fairly free anastomosis of vessels in the pia mater. Sometimes the vascular disease at the base is so situated as to occlude the perforating branches without materially obstructing the main trunk, softening of the pons Varolii, and of the central ganglia of the brain may be brought about in this way.

The grey matter of the brain is much more richly supplied with vessels than is the white matter, hence softening in the grey substance has a red appearance in its earlier stages, whilst in the white substance the red coloration is wanting. Thrombosis in a main trunk may spread by continuity to the branches which the trunk gives off. Thus a clot in the internal carotid artery may spread into the anterior and middle cerebral vessels or into the ophthalmic artery.

It is obvious that thrombosis of the *middle cerebral artery* can produce hemiplegia from either capsular or cortical softening, since it supplies the former by perforating and the latter by cortical branches. Aphasia may result from the cortical softening, and such softening is nearly always thrombotic, for in embolism of the same trunk the healthy anastomosing vessels aided by the higher blood pressure are usually sufficient to carry on the circulation. In the ordinary type of hemiplegia the leg suffers less than the arm and recovers more rapidly, but when the focus of softening is in the posterior part of the capsule producing hemianæsthesia and hemianopia, the motor weakness may be expected to be most marked in the lower limb, since the motor fibres of the leg lie nearer the sensory portion of the capsule than do the motor fibres for other parts of the body. (Fig. 12, p. 179.)

The area of distribution of the *posterior cerebral artery* has already been indicated in the section on cerebral embolism.

Symptoms pointing to occlusion of the *basilar artery* are almost always due to thrombosis, for the anatomical conditions here do

not favour embolism as has previously been explained. Since the occlusion is not at first complete, remissions and exacerbations of symptoms may be expected, and these fluctuations in the amount and distribution of paralysis may help in the distinction of thrombotic from embolic softening. The lower part of the basilar artery is held to supply the median or chief branches to the vagus centre; its upper part is directly continuous with the posterior cerebral trunks into which the clot may extend. If unilateral softening occur in the pons above the decussation of the facial fibres the resulting hemiplegia will not differ from the capsular type; face, arm and leg all being affected on the side opposite to the lesion. If the softening be in the lower half of the pons, an alternate, or crossed, paralysis may occur and the face suffer on the same side as the lesion, whilst the limbs are paralysed on the opposite side. Of course both halves of the pons may become softened and so produce bilateral paralysis. The sixth nerve nucleus is some distance below the third and fourth nuclei, so in lesions of the lower half of the pons the sixth may be paralysed on the same side as the facial nerve, *i.e.*, opposite to the limbs. As the sixth nucleus is the centre for conjugate deviation to its own side the patient will look away from the lesion owing to the unantagonised action of the muscles of the other side.

The *vertebral artery* is a variable vessel; the left is usually larger than the right, indeed the basilar artery may appear to be chiefly formed by the left vertebral. The vertebral is responsible, near its termination, for the blood supply of the hypoglossal and spinal accessory nuclei, and its occlusion produces unilateral labio-glosso-laryngeal paralysis on the side of the lesion as well as interference with the motor and sensory tracts of the medulla.

#### THROMBOSIS OF CEREBRAL SINUSES.

Thrombosis of the cerebral blood sinuses may be primary, traumatic, or inflammatory. Primary thrombosis is met with in certain debilitating conditions at the two extremes of life.

Traumatic thrombosis is usually associated with fracture, and is frequently inflammatory when it ranks with the next group. Inflammatory thrombosis may be due to direct extension of contiguous inflammation to the sinus walls, or the infection may be carried to the sinus by veins which drain an inflammatory focus.

The *lateral sinus* is involved more frequently than any of the others. Its intimate connection with the ear accounts for this, an inflammatory process being set up by extension of caries from the antrum or tympanum to the bony groove in which the sinus lies. The sinus may also be infected by certain of the tributary veins which it receives from the ear. The petrosquamous vein, which receives radicles from the tympanum and attic, joins the lateral sinus behind, and also communicates with the middle meningeal vein in front. The blood from the labyrinth, and from the cochlea, is returned by the internal auditory veins, which open into the inferior petrosal sinus, which in turn a tributary of the lateral sinus.

Thrombosis of the lateral sinus may spread to the sinuses which communicate with it, or to the opposite lateral sinus across the torcular by venous communication. The right lateral sinus usually communicates chiefly with the superior longitudinal sinus, which drains the cortical regions of the brain, whilst the left is continuous with the straight sinus which receives blood from the ventricles and basal ganglia. Sometimes the thrombosis extends along the petrosal sinuses to the cavernous sinus or to the veins of the hemisphere.

The efferent veins of the lateral sinus are the internal jugular, the mastoid, and the posterior condylar. Extension of the infection to the jugular vein gives rise to a tender cord-like swelling under the sterno-mastoid; the surrounding lymphatic glands are also enlarged, and rarely the inflammation attacks the nerves which pass out through the jugular foramen. The jugular blood stream may also convey infection to the lungs, giving rise to infarctions and abscesses. Theoretically it is possible for the jugular bulb to be directly infected from the inner ear by means of the

vein which passes out through the aqueductus cochleæ without participation of the lateral sinus. The mastoid emissary vein is responsible for the production of mastoid œdema and superficial suppuration, but the œdema may occur as a result of mastoid disease without sinus thrombosis. The posterior condylar veins which pass to the suboccipital and thence to the vertebral veins may become involved, and associated with this the posterior cervical venous plexus which lies at the upper part of the posterior triangle of the neck may be the site of thrombosis. The vertebral vein, by its connection with the lateral sinus through the posterior condylar vein, sometimes affords another path for infection of the lungs, when the internal jugular has been ligatured. Suppurative meningitis and extradural abscess are often associated with thrombosis of the lateral sinus, and infection may also spread from the sinus to the cerebrum or cerebellum, giving rise to abscesses in these structures. The external suture between the squamous and petrous bones lying behind the external auditory meatus affords a track by which inflammation in the ear may spread to the surface and so may save the sinus, but this suture closes in the second year of life.

Thrombosis of the *cavernous sinus* is uncommon, although its free venous communications render it liable to infection from many places. Lateral sinus thrombosis caused by ear disease, may spread along the petrosal sinuses to the posterior end of the cavernous sinus. The ophthalmic veins which enter the front of the sinus may carry infection to it from the territories of the angular, facial, or supra-orbital veins. Disease of the nose may extend to the sinus by way of the sphenoidal air cells, or by the medium of the ethmoidal veins which enter the ophthalmic trunk, or of veins in the sphenomaxillary fossa which enter the pterygoid plexus, which in turn communicates freely with the cavernous sinus. Ulcerations of the tonsil and pharynx, retropharyngeal abscesses and nasopharyngeal diphtheria have all occasionally caused cavernous thrombosis; infection being carried by the veins of the pterygoid plexus, which enter the sinus from below through

the foramen lacerum medium, the foramen of Vesalius, if present, and the foramen ovale. Alveolar abscesses, and necroses of the jaw and orbit may have the same result.

Owing to the serious obstruction to the return of blood from the orbit and the intimate relation of the third, fourth, fifth and sixth nerves to the walls of the sinus, the signs of cavernous thrombosis resemble those of cellulitis of the orbit combined with ophthalmoplegia. The lids are swollen and œdematous, and discoloured, the eye is prominent, and there is much chemosis. At the same time the movements of the eye are restricted or lost, and there is considerable pain in the territory of the supraorbital branch of fifth nerve.

Since the sinus communicates freely by means of the circular sinus with the cavernous sinus of the opposite side the thrombosis sooner or later becomes bilateral. As nearly all the veins of the sinus drain into the internal jugular veins, these are the vessels which convey pyæmic infection to the lungs and other parts of the body. Any attempt to exclude the cavernous sinus from the circulation calls for a ligature of the internal jugular, very low in the neck.

The *superior longitudinal sinus* is usually affected in marantic thrombosis, but is sometimes thrombosed as the result of fractures and injuries to the vertex of the skull, and rarely as a sequel to lateral sinus disease. There are several reasons why, with failing circulation, thrombosis should appear in this sinus. The blood flow in it is retarded because the stream has to pass backwards whilst the blood in the veins of the cerebral hemispheres is projected into it in the opposite direction; again, the blood from the ascending arteries of the cortex is carried on into the sinus by veins which also ascend, and so the flow is not assisted by hydrostatic pressure, and lastly the lacunæ laterales form as it were small backwaters in which thrombosis may commence.

As a rule the symptoms of thrombosis of the superior longitudinal sinus are indefinite, but sometimes œdema of the scalp occurs and the veins of the parietal, frontal, and occipital regions



are turgid. Epistaxis may be present in childhood and interference with the circulation of the cerebral cortex may induce convulsions or paralysis. In early life the foramen cæcum transmits to the sinus a small vein from the nose and this may be the source of the epistaxis. The communication with the sinus of the parietal emissary vein may account for venous congestion and œdema in this part of the scalp.

## ABSCESS OF THE BRAIN.

The intimate relations of the tympanic cavity and of the mastoid cells to the temporo-sphenoidal lobe of the brain and to the cerebellum account for the frequency with which ear disease is the cause of temporo-sphenoidal and of cerebellar abscesses. In like manner the proximity of the upper part of the nasal cavity and of some of the accessory sinuses of the nose to the frontal lobe of the brain may give rise to frontal abscess when the nasal cavities are diseased.

Brain abscess may also be a sequel of disease or injury of other bones of the skull, and in such cases the abscess as a rule lies in close relation to the diseased or injured bone. Sometimes the abscess is in a part of the brain remote from the site of injury, but at the same time in a direct line with it; such a position appears to be determined by the bruising due to *contrecoup*. In this way a frontal injury has been known to produce an occipital abscess.

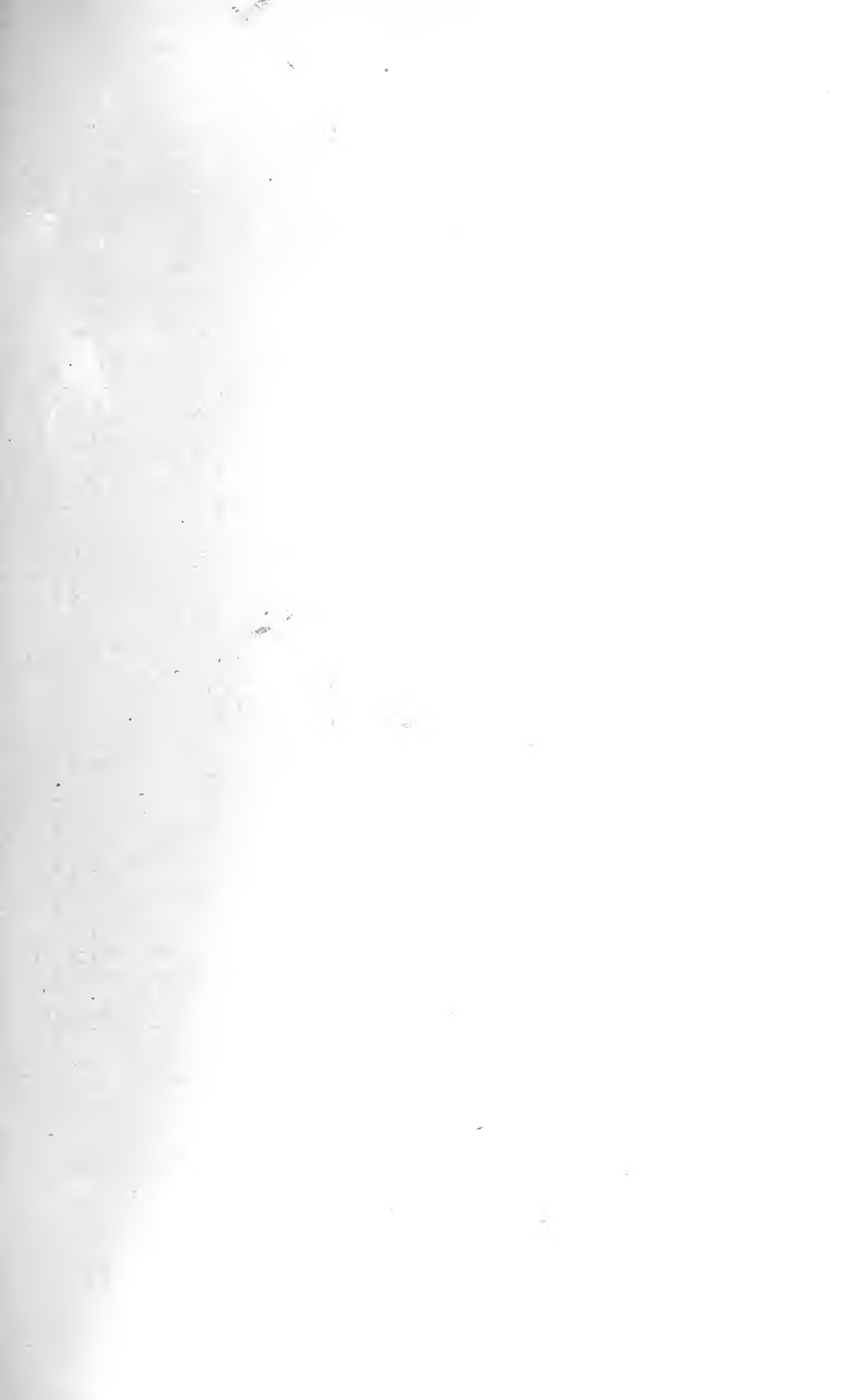
Blood-borne infection from a distance may also be a cause of cerebral abscess. The focus of such infection is usually in the lung, and the infective agent is carried by the pulmonary veins to the left side of the heart, and thence by the arterial system to the brain. Bronchiectasis, empyema, and sometimes phthisis, produce abscesses in this way. Ulcerative endocarditis occasionally leads to embolic abscesses of the brain, but is more likely to induce hæmorrhage or cerebral softening. Brain abscesses may also occur in general pyæmia. Abscesses due to blood-borne infection are usually multiple.

Temporo - sphenoidal and cerebellar abscesses are nearly always caused by suppurative ear disease. Caries of the tegmen tympani or of the tegmen antri may give rise to extradural abscess or subdural abscess in the middle fossa, but the brain membranes often become adherent to the lowest temporo-sphenoidal convolution which lies directly over the tegmina, and a temporo-sphenoidal abscess results.

Extension of inflammation through the labyrinth to the internal auditory meatus produces similar results in the neighbourhood of this opening. The membranes may become adherent and the front part of the lateral lobe of the cerebellum be found to contain an abscess. But temporo-sphenoidal and cerebellar abscesses may occur without caries or perforation of the bone and without adhesion of the brain membranes. In such cases the veins or the perivascular lymphatics are the tracks along which infection travels. The abscesses in the brain or in the cerebellum may be multiple, as many as three having been found in the temporo-sphenoidal lobe and the same number has been known to occur in the cerebellum. The superior petrosal sinus receives veins from the tympanum and from the temporal lobe, so this sinus may provide the link between the two. The lateral sinus receives veins from the mastoid region and the cerebellum, and may allow the transference of the infective agent from one to the other. It is difficult to conceive that such transference can occur unless the sinuses in question are thrombosed.

At birth there is a petro-squamous suture in the roof of the tympanum, but this is obliterated in the second year and has no influence in the causation of temporo-sphenoidal abscesses, since these are not met with at this period of life.

Disease of the nose and of its accessory sinuses may lead to abscess in the *frontal* lobe of the brain. The cribriform plate of the ethmoid forms a very fragile septum between the brain and the roof of the nose. The frontal air sinuses when of average size extend about an inch upwards in the adult, from the upper orbital margin, and nearly an inch backwards along



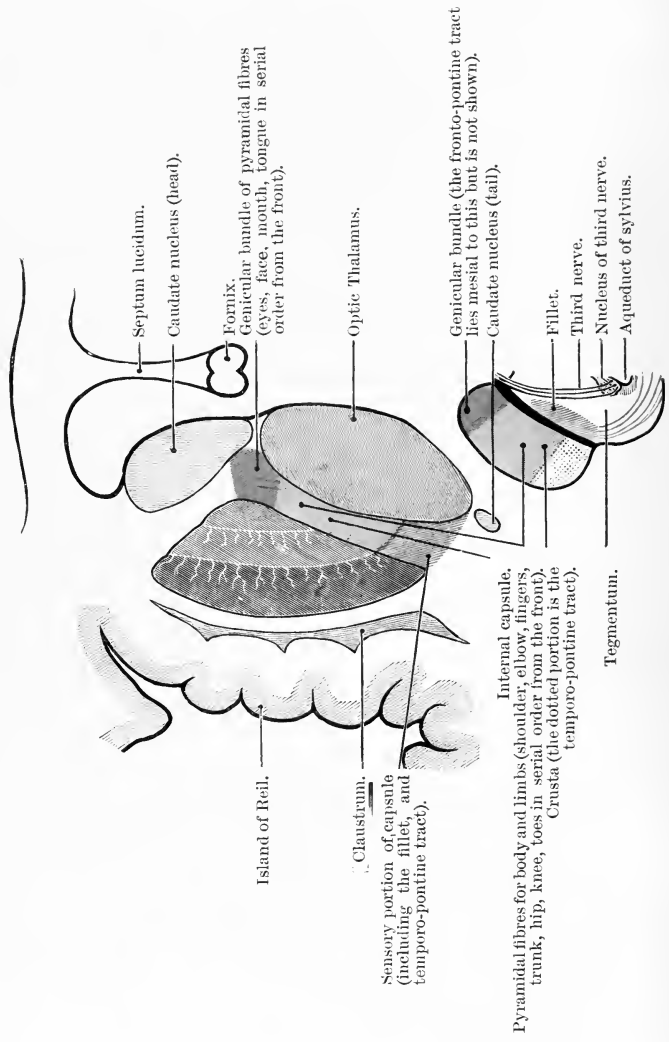


FIG. 12.—Diagram showing the positions of the motor and sensory fibres in the internal capsule and mid-brain. (From Morris's Anatomy, after Arthur Robinson.) The auditory radiation and the optic radiation which lie immediately behind the posterior limb of the capsule are not shown.

the orbital roof. These sinuses are of insignificant size until the sixth or seventh year of life. A very thin plate of bone separates the fully developed sinus from the frontal lobe of the brain, so that caries and perforations of its inner wall may cause extradural, subdural or intracerebral abscess. In the latter case the brain membranes are often closely adherent to the perforated bone, but in some instances there is no perforation and no adhesion, the infection having been carried by the lymphatics or veins of the diploë.

The anterior and posterior ethmoidal cells are separated from the anterior fossa of the skull by the ethmoidal edge of the frontal bone, but occasionally a posterior ethmoidal cell, extending backwards, intervenes between the roof of the sphenoidal sinus and the base of the brain. It is easy to see that infection of the brain membranes and the brain might originate from these cells.

The roof of the sphenoidal air sinus lies in contact with the olfactory peduncles, the optic commissure, the pituitary body and the front part of the pons Varolii. Infection is not often transmitted through it.

An abscess in the *temporo-sphenoidal* lobe is so situated that it may cause motor paralysis of either cortical or capsular type. By pressure on the adjacent lower end of the ascending frontal convolution or by the spread of inflammatory œdema to that region, convulsions or paralysis may be produced, and in each instance the face may be expected to suffer first, then the arm, and lastly the leg, owing to the arrangement of motor centres in that order from below upwards. (Fig. 14, p. 183.) Any anæsthesia which is present will be of the patchy and incomplete cortical type.

By pressure exerted inwards, and by the inward spread of inflammation, the internal capsule becomes involved in its posterior part. The motor paralysis is then of capsular type, and the leg, the fibres of which lie most posterior of the motor fibres, will suffer first. (Fig. 12.) Since the posterior part of the capsule is involved, hemianæsthesia and hemianopia may occur.

The auditory centre lies in the posterior part of the upper temporo-sphenoidal convolution and the angular and supra-marginal visual centres are adjacent. Hence alexia, agraphia and word-deafness may accompany extensive or acute temporo-sphenoidal abscesses of the left side. The deafness which accompanies these abscesses and usually also the occasional complete facial paralysis are accounted for by the lesion in the ear on the side of the abscess. Rarely, cortical deafness occurs in the opposite ear, but frequently this ear is also diseased.

Temporo-sphenoidal abscesses have a great tendency to rupture into the ventricles of the brain and naturally break into the descending cornu of the lateral ventricle since this traverses the lobe in which they lie.

The third cranial nerve on the same side as the abscess often shows signs of weakness in the form of ptosis or paralysis of the muscles of the globe or inactive pupil. The nerve lies in the front part of the tentorium cerebelli, and is possibly subject to pressure from the swollen temporo-sphenoidal lobe, for the uncus of this lobe is often deeply grooved by the edge of the tentorium.

The knowledge of the localisation of *cerebellar* functions is scanty, so the application of anatomical facts is difficult. Cerebellar abscesses are subtentorial, and may produce pressure symptoms in the posterior fossa, such as occipital headache radiating down the neck, and distension of the ventricles of the brain.

From the dentate nucleus of the cerebellum fibres stream out to the superior peduncle and so reach the opposite cerebral hemisphere. Interference with these fibres is supposed to produce a particular type of paralysis which consists in considerable brachial monoplegia on the side of lesion, coupled with this may be some weakness of the lower extremities. This paralysis is explained by the assumption that the cerebellar hemisphere normally augments the action of the cerebral hemisphere of the opposite side. Since the cerebellar abscess

is usually in the outer and fore part of the hemisphere, the dentate nucleus and its fibres escape and paralysis of the above type is then absent. Cerebellar lesions do not produce any anæsthesia.

Rarely the abscess lies in the flocculus and then the cranial nerves in the proximity may be paralysed. The nerves in immediate relation are the seventh, the eighth and the pars intermedia; but the fifth, sixth, ninth and tenth are not far off. Unfortunately for localisation, the seventh and eighth nerves may be affected in the diseased petrous bone and indeed are more likely to suffer here than in the neighbourhood of the flocculus.

#### INTRACRANIAL TUMOURS.

After obliteration of the fontanelles, a process which is complete before the second year of life, the cranium is practically a closed cavity. The majority of intracranial tumours occur some time after this closure, *i.e.*, between puberty and middle adult life, hence the pressure effects produced by these growths are more marked and more extensive than those produced by new growths in any other part, save perhaps the vertebral canal. Tumours growing in the thorax and bony pelvis afford the nearest approach to similar conditions elsewhere in the body. Tuberculous tumours sometimes occur before the period of closure of the anterior fontanelle, but even then the mass often lies below the tentorium cerebelli, and so the patency of this fontanelle has but little modifying influence on the direct pressure symptoms, although it may permit distension of the ventricles of the brain.

The strength and firm attachment of the tentorium cerebelli, which forms a fibrous dome over the posterior fossa of the skull, account for the marked pressure symptoms associated with tumours which grow beneath it. The parts of the brain which lie in the posterior fossa are the cerebellum, medulla, pons, and cranial nerves below the fifth. The veins of Galen and the straight sinus may be occluded by subtentorial tumours, and

the exit of fluid from the third and lateral ventricles may be obstructed by the pressure these tumours exert on the iter or behind it.

The sagittally directed falx cerebri, although firmly fixed at its two extremities and above, has only a slight influence in restricting pressure signs to one or other hemisphere of the brain.

Tumours are occasionally found to compress certain large arterial trunks at the base of the brain, and then areas of softening may occur in addition to the new growths.

Tumours which interfere with the outlets of the ventricles produce a peculiar train of symptoms, amongst which are apathy, drowsiness, headache, and coma. Such symptoms may accompany tumours of the corpus callosum or of the structures forming the boundaries of the third ventricle or tumours beneath the tentorium.

Of the general symptoms of **cerebral tumour**, headache is of some localising value if accompanied by local tenderness, and this is most likely to occur if the tumour is on the convexity of the brain. Speaking generally, frontal headache accompanies tumours of the frontal lobes; occipital headache tumours of the occipital lobes; and similar headache extending into the nape of the neck, and often accompanied by retraction of the head, tumours of the subtentorial region. With a cerebellar tumour headache may occur in the opposite frontal region; the crossed connexion of each cerebellar hemisphere with the frontal lobe of the opposite side is held by some to account for this anomaly.

Vomiting, another general symptom, is most severe and most common with tumours below the tentorium, presumably on account of the proximity of the vagus centre.

The fact that the motor tract traverses the brain from medulla to cortex explains how tumours in widely different situations can produce motor paralysis. The fan-like radiation of the corona radiata from the genu and anterior part of the posterior limb of the internal capsule to the cortex of the Rolandic region allows cortical and subcortical tumours to cause paralysis, which may



THE MOTOR TRACTS.

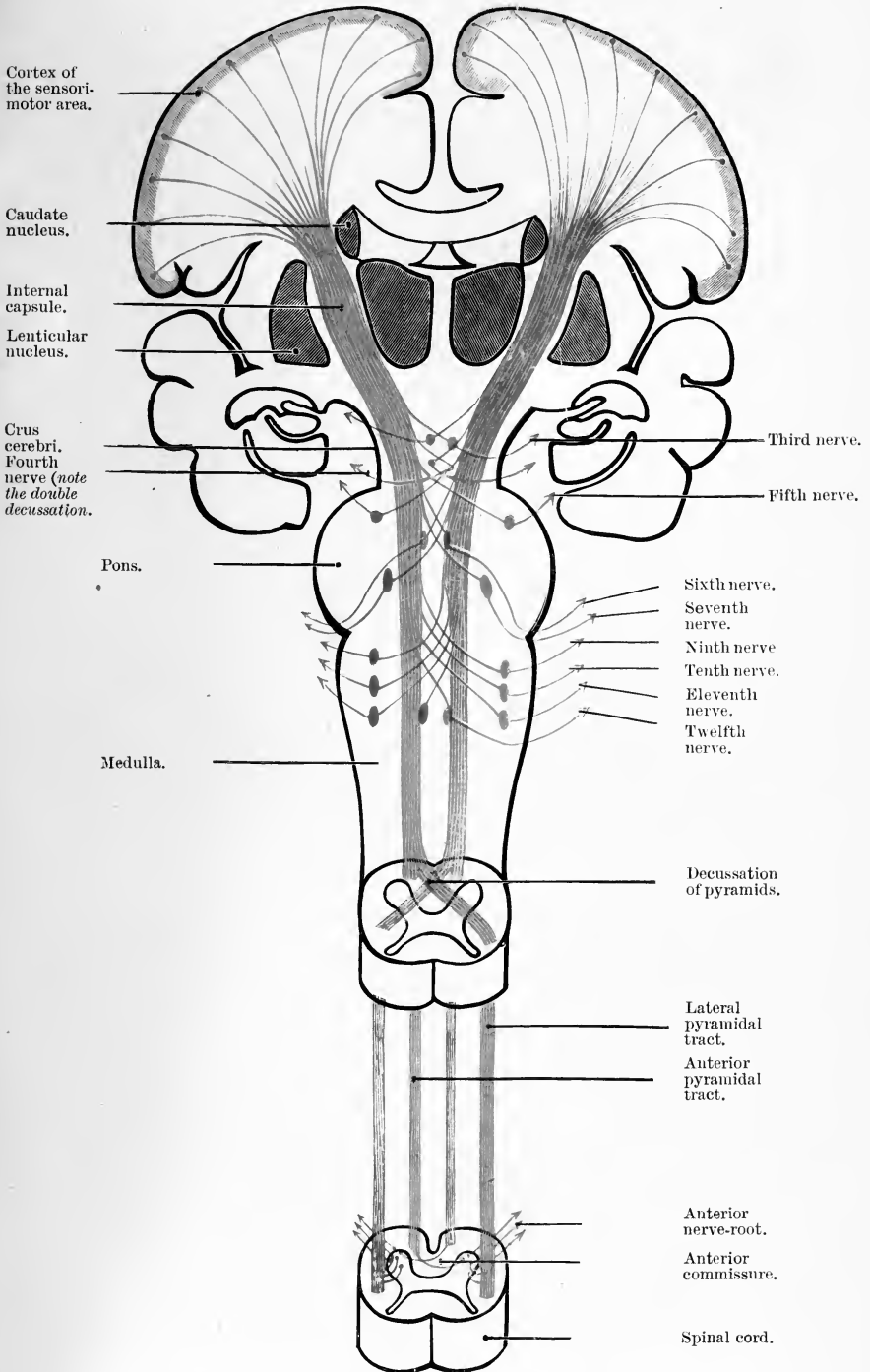


FIG. 13.—Diagram of the pyramidal fibres and the nuclei of the cranial and spinal motor nerves. (From Morris's Anatomy, after Arthur Robinson.)





MOTOR CORTEX.

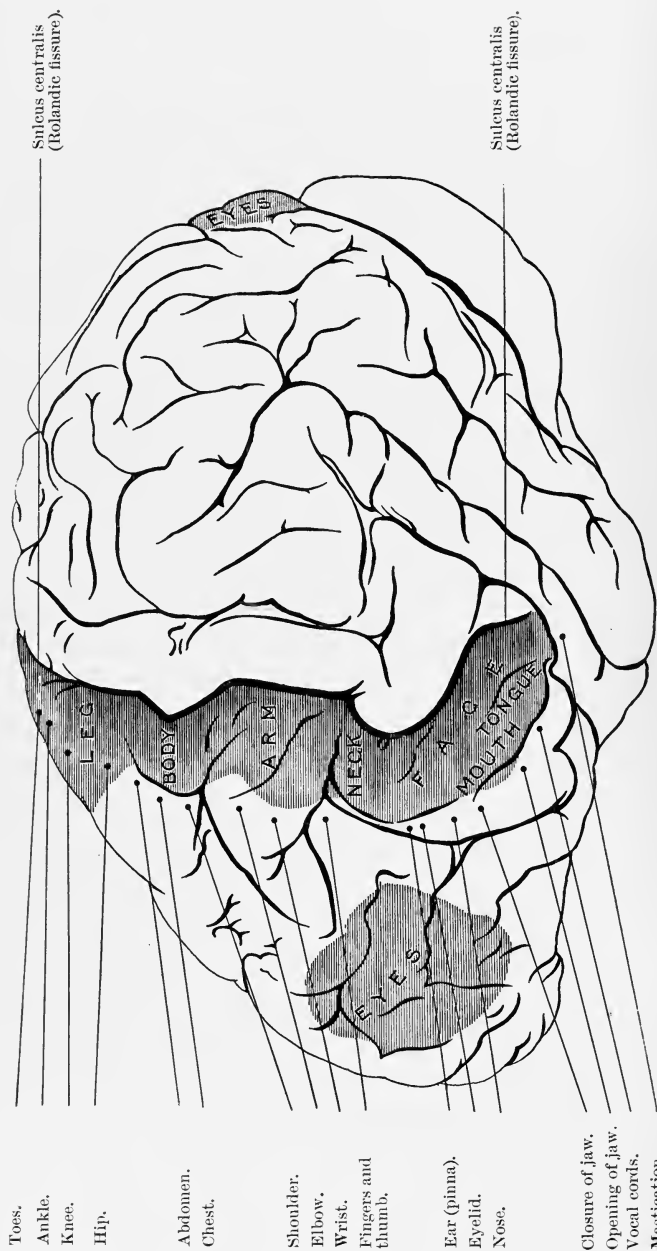


FIG. 14.—The left hemisphere of the brain of the chimpanzee showing the motor areas of the cortex. (After C. S. Sherrington and A. S. F. Grünbaum.)

be at first limited to one limb or to parts of a limb on the opposite side of the body. Jacksonian epilepsy may be associated. If such a tumour happen to be at the spot where the leg centre, which stands highest in the precentral convolution, adjoins the trunk and arm centres which lie next below it, weakness of the hip and shoulder on one side may result. (Fig. 14.) A tumour in the neighbourhood of the internal capsule will tend to produce hemiplegia of gradual development, and so also will a tumour in the upper part of the crus compressing the motor tract in the crusta. In the pons and medulla, where the motor tracts of the two sides of the body approximate, a bilateral paralysis may result, but symmetrically placed tumours in the hemispheres may have the same effect. (Fig. 13.)

When a tumour is so situated as to paralyse a cranial nerve on one side of the body and the limbs on the opposite side, the so-called "crossed paralysis" is produced. Tumours in the crus, the pons or the medulla may do this. A tumour of the crus may involve the third nerve on the same side, and the rest of the body on the opposite side. (Fig. 13.)

In the upper part of the pons, the fifth nerve may be paralysed on the side of the lesion, and the face, tongue, and limbs on the opposite side. The facial tract crosses the mid line at the junction of the upper and lower halves of the pons to reach the facial nucleus, so a tumour in the lower part of the pons will completely paralyse the face on the same side and involve the opposite half of the tongue and opposite limbs as well, by pressing on the motor tracts to these parts above their decussation. (Fig. 13.)

A tumour on one side of the medulla, above the decussation of the motor tracts in the pyramids may paralyse the tongue on the same side, and the limbs on the opposite side, but this is uncommon.

The presence of certain varieties of cranial nerve paralysis is important in fixing the locality of a tumour. The nucleus of the third nerve extends from the back part of the floor of the third ventricle to the groove between the anterior and posterior corpora

quadrigemina, the nucleus lies in the front part of the floor of the iter, and the nerve emerges from the inner side of the crus. Tumours of the crus therefore tend to paralyse this nerve as already indicated. (See Fig. 17, p. 191.)

The fourth nerve nucleus also lies in the floor of the iter, immediately behind the nucleus of the third, and corresponds to the fore part of the posterior corpora quadrigemina. The nerve emerges dorsally from the valve of Vieussens, and winds round the outside of the crus. It may suffer in association with the third when tumours occupy the quadrigeminal bodies or be compressed by a tumour near the anterior end of the cerebellar vermis.

The sixth nerve nucleus lies beneath the eminentia teres in the anterior part of the floor of the fourth ventricle; the nerve fibres emerge in the groove between the top of the pyramid and the lower border of the pons. Owing to its long intracranial course, paralysis of this nerve has little localising value, but the nerve lies between the ventral surface of the pons and the basilar process, and is likely to be compressed in subtentorial lesions. Paralysis of all the ocular muscles of one eye, often associated with blindness of the same eye, is likely to be caused by a tumour of the sella turcica, which lies adjacent to the cavernous sinus, and contains the pituitary body.

The sixth nerve nucleus may be considered to be the centre for conjugate deviation of the eyes towards its own side, its crossed connection with the nucleus of the opposite third nerve accounts for this. The communication is effected through the posterior longitudinal bundle. A tumour in the pons or corpora quadrigemina affecting the sixth nucleus will render impossible conjugate deviation of the eyes towards the same side, since it will paralyse the associated actions of the external rectus of its own side and the internal rectus of the opposite eye. If both sixth nuclei be involved the eyes will remain fixed in the mid position. When the facial nerve is paralysed by tumours within the pons, the nucleus of the sixth nerve around which the genu of the facial root turns, may also suffer. Outside the pons facial paralysis is more likely to be associated with paralysis of the auditory



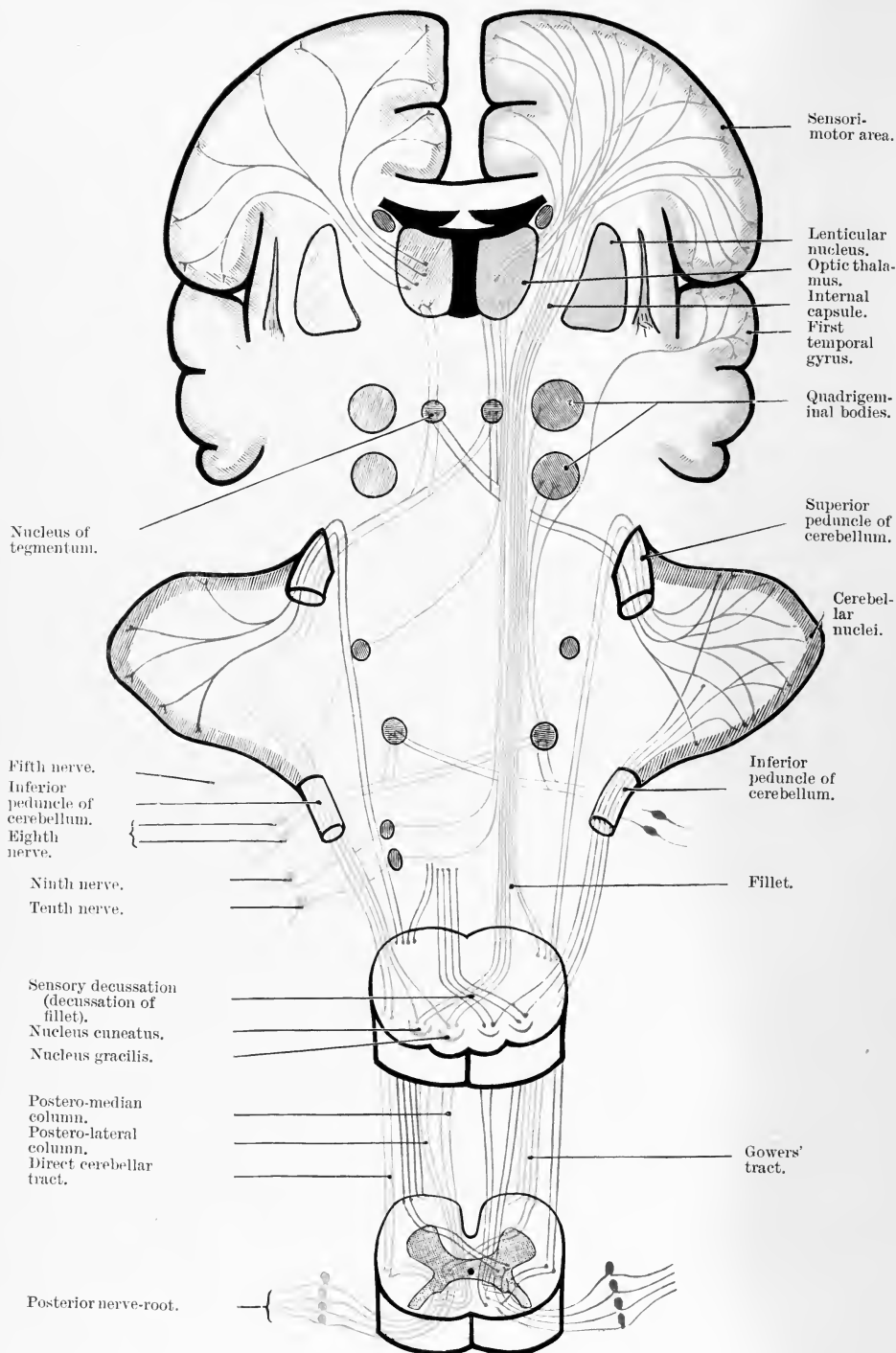


FIG. 15.—Diagram showing some of the paths by which a sensory impulse may ascend from the spinal cord to the brain. (From Morris's Anatomy, after Arthur Robinson.) The fibres from the nucleus gracilis and nucleus cuneatus to the opposite side of the cerebellum are not shown.



nerve which lies contiguous to facial trunk in the membranes and internal auditory meatus.

The spinal accessory nerve may be paralysed by tumours in the medulla and its vicinity. Isolated paralysis of the sterno-mas-toid and trapezius points to tumour outside the medulla, as these muscles are innervated by the purely spinal portion of the nerve.

The tract of the sensory fibres in the brain is not so well known and not so well localised as is the motor tract. The fibres traverse the posterior part of the posterior limb of the internal capsule, and a tumour here may produce complete hemianæsthesia. Tumours of the gyrus fornicatus (callosal gyrus) have the same effect. Even tumours of the so-called motor cortex produce a blunting of sensation in the part most paralysed, and, what is more striking, loss of power to accurately localise sensory impressions on the part. The sensory fibres reach the internal capsule by traversing the tegmental region of the crus, but lesions here seldom produce marked anæsthesia. Hemianæsthesia may be produced by tumours of the pons and of the medulla. (Fig. 15.)

Intracranial tumours may be so situated as to involve the sense of sight or the sense of hearing.

The visual fibres may be involved anywhere between the eye-ball and the occipital cortex. Bilateral homonymous hemianopia will result when a tumour involves the optic tract or optic radiation or the occipital visual cortex of one side, for these structures are in relation with a half of each retina, since only the fibres from the nasal half of the retina decussate.

When a tumour presses on the central part of the optic chiasma bitemporal hemianopia results, since the decussating fibres from the nasal halves of the retina are affected, and the nasal halves of the retina correspond to the temporal halves of the visual fields. Frequently a central defect of vision suggestive of tobacco amblyopia precedes the full development of the hemianopic defect. If the concentration of light on the blind half of the retina in hemianopia still produces a pupil reflex, the afferent fibres in the optic tract must be able to convey impulses as far as the point

where a connection is established with the third nerve, *i.e.*, the lesion producing the hemianopia must be above the external geniculate body, and somewhere between it and the occipital cortex.

Hearing may be involved as the result of tumour pressure on the auditory nerve. Tumours in the pons or at the base of the brain can do this, and the deafness is on the same side as the lesion. Tumours of the auditory centre in the temporo-sphenoidal lobe may also produce subjective noises or deafness and then the deafness is of the opposite ear owing to the decussation of the tract.

Intracranial tumours which spring from the base of the skull are usually either sarcomatous or carcinomatous. Their usual seats are the petrous portion of the temporal bone, the sella turcica or the occipital bone in the neighbourhood of the foramen magnum. Growths in the region of the sella turcica tend to compress all the nerves of one eyeball and may press on the optic chiasma with results already mentioned; they may also cause loss of smell by pressure on the olfactory tract. Growths of the petrous bone are adjacent to the Gasserian ganglion and the fifth nerve, hence they may produce facial neuralgia, anæsthesia in the territory of the fifth nerve, paralysis of masticatory muscles, and even keratitis. These tumours may also invade the posterior fossa, since the petrous bone is one of its boundaries. The nerves of the posterior fossa may thus be involved and occlusion of the lateral sinus also result. In the substance of the petrous bone the growth may erode the carotid, and so produce bleeding from the ear, whilst masses of glands may appear beneath the mastoid deep in the upper part of the neck. Tumours in the neighbourhood of the foramen magnum may implicate any of the nerves behind the fifth, but chiefly the lower ones. They also exercise pressure on the motor tract for the opposite side of the body, but cannot produce conjugate deviation of the eyes, since for this to occur the nucleus of the sixth nerve within the pons must be involved.

Tumours of the middle and anterior fossæ of the skull may

invade the orbit through the sphenoidal fissure and cause proptosis. Tumours of the middle fossa sometimes involve the roof of the nasopharynx, and tumours of the posterior fossa may give rise to masses in the neck behind, or under cover of, the mastoid process.

## CRANIAL NERVES.

### THE OPTIC NERVE AND TRACT.

The accessibility of the optic disc to ophthalmoscopic examination renders possible the detection of slight degrees of inflammation which probably pass unrecognised in other cranial nerves.

The optic nerve is invested by tubular sheaths prolonged from the pia, arachnoid, and dura, therefore inflammation of the meninges can produce optic neuritis by extension along these nerve sheaths. Effusions of blood and serum may also extend in the same direction or local hæmorrhage may occur within the firm dural sheath as the result of injury; injurious compression of the nerve fibres may thus be set up.

At the optic foramen the periosteum of the orbit is continuous with the dural sheath of the nerve, and thus periosteal inflammation of the orbit, due to bone disease or even secondary to dental caries, may extend to the nerve and induce neuritis.

As the nerve passes through the optic foramen it is only separated from the sphenoidal air sinus by a thin bony lamina, which is sometimes imperfect, and inflammation of this air sinus may secondarily involve the nerve. Fractures in the neighbourhood of the foramen may compress or actually sever the nerve fibres, and bony growths in this situation may also cause pressure upon the nerve.

The optic foramen is really a narrow canal and is traversed by the ophthalmic artery as well as the optic nerve, consequently inflammatory swelling of nerve or periosteum may give rise to severe compression of the visual fibres.

Broadly speaking, the fibres from the periphery of the retina lie at the periphery of the nerve trunk and in a lesion near the

foramen are the first to suffer, giving rise to concentric diminution of the visual fields.

The ophthalmic artery lies below and to the outer side of the nerve in the intracranial portion of its course, but leaves the dural sheath to curl over the nerve when the orbit is reached. Aneurysms may compress the nerve in either situation. The central artery of the retina which is sometimes the site of embolism, passes into the nerve a short distance behind the eyeball.

The **optic chiasma** practically forms part of the floor of the third ventricle, and so may be compressed by effusions in the ventricular system. Immediately behind the chiasma lies the pituitary body, tumours of which may give rise to a characteristic form of visual defect, bitemporal hemianopia. The internal carotid arteries occupy the outer angles of the chiasma and nasal hemianopia may be produced by aneurysms or calcification of these vessels, but is rare. The sphenoidal air sinuses are in the floor of the middle fossa below the chiasma, and the latter lies in the cisterna basalis so that it is liable to invasion by gummatous and tuberculous tumours, and inflammation of the meninges.

Disease processes which affect the chiasma act chiefly on its central portion and accordingly the characteristic symptom of a chiasma lesion is bitemporal hemianopia due to interference with the decussating fibres from the nasal halves of the retina, which in turn receive impressions from the temporal halves of the visual fields. The temporal fibres are uncrossed, and chiasma lesions often extend irregularly to one side or the other and so may involve temporal fibres also in varying degree. (Fig. 16.)

A lesion limited to the non-decussating (temporal) fibres of one side should produce nasal hemianopia, but this is exceptional.

The adjacent motor nerves of the eye are liable to be involved in affections of the chiasma.

The **optic tract** extends backwards from the chiasma to terminate in the anterior corpus quadrigeminum, the external geniculate body, and the pulvinar of the optic thalamus. In part of its course it is closely applied to the fore part of the crus



THE VISUAL PATHS.

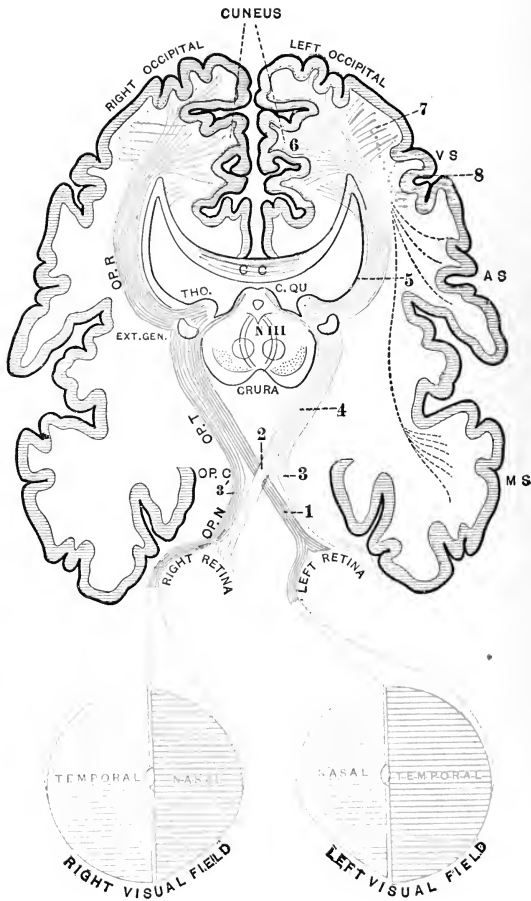


FIG. 16.—Diagram of the visual paths (from Violet, modified by Osler). OP.N., optic nerve; OP.C., optic chiasma; OP.T., optic tract; OP.R., optic radiation; GEN., geniculate body; THO., optic thalamus; C.QU., corpora quadrigemina; C.C., corpus callosum; V.S., visual speech centre; A.S., auditory speech centre; M.S., motor speech centre. A lesion at 1 causes blindness of that eye; at 2, bi-temporal hemianopia; at 3, nasal hemianopia. Symmetrical lesions at 3 and 3' would cause bi-nasal hemianopia; a lesion at 4 hemianopia of both eyes, with hemianopic pupillary reaction; at 5 and 6, hemianopia of both eyes, pupillary reflexes normal; at 7, amblyopia, especially of the opposite eye; at 8, on *left* side, word blindness.

cerebri, and both may suffer together as the result of tumour infiltration. The tract, bending round the crus, lies under cover of the temporo-sphenoidal lobe and may be compressed by new growths in this situation. It is also liable to injury by tumours springing from the base of the skull.

The visual defect which results from a lesion of the tract is homonymous hemianopia since the tract contains uncrossed fibres from the temporal half of the retina of its own side and crossed fibres from the nasal half of the retina of the opposite side; hence the blindness involves the nasal half of the field on the side of lesion and the temporal half of the field of the opposite side. (Fig. 16.)

If no light reflex can be obtained from the blind halves of the retina, or if the reflex from these halves is very sluggish, the hemianopic pupillary reaction is said to be present. This is of value in that it localises the lesion in the chiasma or tract as distinguished from the optic radiation in the hemisphere. This indication is based upon the fact that the fibres which carry the impulse from the eye to the pupil nucleus in all probability leave the optic tracts at their partial termination in the anterior corpora quadrigemina, passing thence to the third nerve nucleus. Consequently a lesion which produces the hemianopic reaction must involve the visual fibres not further back than the corpus quadrigeminum.

The anterior choroidal branch of the internal carotid artery closely follows the course of the optic tract and distributes branches to it.

The apparent termination of the optic tract in the superior quadrigeminal body, the external geniculate body, and the pulvinar, might lead to the expectation that disease of these structures should produce defects of vision, but clinical confirmation of this is still needed.

The **optic radiation** consists of fibres which pass from the external geniculate body and the pulvinar to the cortex. These fibres lie in the retrolenticular part of the posterior limb of the external capsule; sweeping thence round the posterior cornu of

the lateral ventricle they terminate in the occipital cortex. Some of the fibres possibly pass to the angular region, but the optic radiation lies immediately subjacent to this part of the parietal lobe, and may be indirectly injured by lesions in this situation even if it do not in part terminate here. (Fig. 16.)

The optic radiation may be damaged by softening, hæmorrhage, tumour or injury of the optic thalamus, or the posterior part of the internal capsule. In such cases hemianæsthesia or hemiplegia may be associated with homonymous hemianopia. Lesions in the occipital lobe, the occipital cortex, and possibly in the parietal lobe, may also damage the tract.

The blood supply of the occipital visual cortex is derived from the occipital branch of the posterior cerebral artery. This branch also supplies the optic radiation. Sight may be impaired as the result of embolism or thrombosis of this vessel, hemianopia resulting. The parieto-occipital and the calcarine branches of this occipital artery lie in the superior and inferior limiting sulci of the cuneal lobule and the parieto-occipital branch supplies a secondary cuneal branch which lies in a fissure in the cuneus parallel to the calcarine artery.

Since the posterior cerebral artery supplies the temporal lobe in part, lesions of this lobe may be associated with lesions of the occipital visual centres.

The angular region of the cortex lies in the area of distribution of the middle cerebral artery.

The visual portion of the thalamus is supplied by the posterior perforating branches of the posterior cerebral artery. (Fig. 10, p. 165.)

The superior quadrigeminal and the external geniculate bodies are also supplied by the posterior cerebral artery.

#### OPHTHALMOPLÉGIA.

Anatomically considered ophthalmoplegia may be divided into supranuclear, nuclear and infranuclear varieties.

**Supranuclear ophthalmoplegia.**—Areas of the frontal lobes and possibly also of the parietal and occipital lobes are connected





CRANIAL NERVE NUCLEI.

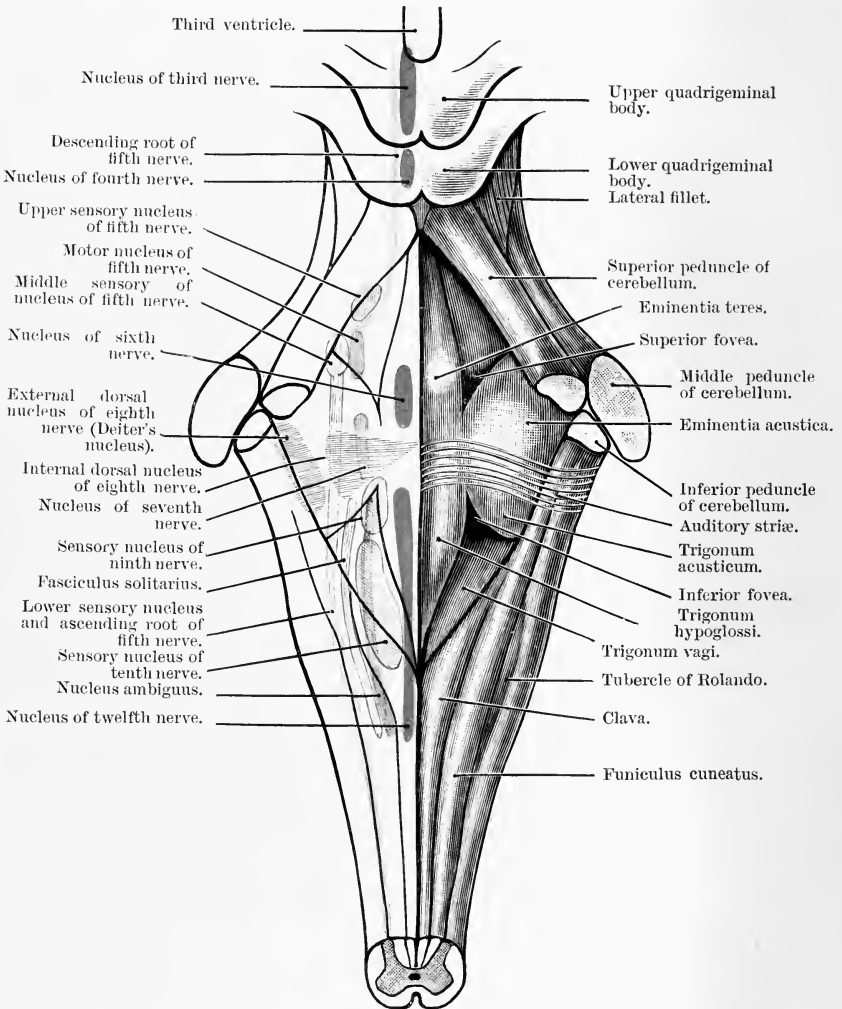


FIG. 17.—Diagram of the floor and lateral boundaries of the fourth ventricle, showing the positions of the nuclei of the cranial nerves on the left side. (From Morris's Anatomy, after Arthur Robinson.)

with the nuclei for the eye muscles. (Fig. 14, p. 183.) The track from the cortex is supposed to pass through the internal capsule near its genu, and thence to the nuclei through the mesial third of the basal portion of the crus cerebri. Supranuclear lesions do not produce isolated ocular paralyses, but manifest themselves by temporary conjugate deviations of the eyes. The crus cerebri, however, is traversed by the rootlets of the third nerve on their way out from the adjacent nuclei, and consequently the paralysis produced by lesions of this part of the brain are of a nuclear or infranuclear type.

**Nuclear Ophthalmoplegia.**—The nucleus of the third nerve is compound, its total length being about a third of an inch. Its anterior part encroaches on the floor of the third ventricle, and its posterior or lower limit corresponds to the groove between the anterior and posterior quadrigeminal bodies. Degenerative nuclear disease, such as, for example, results from syphilis, may pick out portions of the compound nucleus, producing paralysis of isolated muscles or of specially associated ocular movements. Such isolated paralyses as loss of accommodation, loss of the pupil contraction which normally accompanies convergence of the eyes, loss of convergence and accommodation although the internal recti still act in conjugate movements, and paralysis of a single extraocular muscle, are examples of this selective action. The most familiar example of all is the Argyll Robertson pupil, in which miosis and loss of light reflex co-exist. It is true that the usual explanation of this double phenomenon is that the miosis is due to a lesion of the fibres of the cervical sympathetic in some part of their course through the central nervous system before emerging by the cervical roots, and that the failure to react to light is due to a lesion of Meynert's fibres which pass between the superior corpora quadrigemina and the nucleus of the third nerve. It is possible, however, that actual degeneration of a part of the third nucleus can produce both phenomena. The ophthalmoplegia of nuclear disease may be very extensive and is often bilateral. Weakness of the orbicularis palpebrarum is sometimes associated with ophthalmoplegia, leading to the

presumption that the motor fibres to this muscle, which reach it through the facial nerve, are really derived from part of the third nerve nucleus.

The nucleus of the fourth nerve innervates the superior oblique muscle of the eye, and lies in the floor of the iter beneath the posterior quadrigeminal bodies. This nucleus is in serial continuity with the nucleus of the third, and suffers in a similar way.

The nucleus of the sixth nerve is some way behind that of the fourth. It lies beneath the eminentia teres of the fourth ventricle, immediately above the acoustic striæ. The sharp bend which the issuing fibres of the facial nerve make around the sixth nucleus explains how a comparatively small lesion may involve both the sixth and the facial nerves. The sixth nucleus is important in that it is the nuclear centre which produces conjugate deviation of the eyes to its own side, the associated action of the opposite internal rectus being brought about by fibres of the posterior longitudinal bundle which connect the sixth nucleus with the appropriate part of the third nucleus of the opposite side.

In addition to being subject to degenerative lesions the ocular nuclei may be damaged by vascular lesions of the pons or crus, and by gross lesions of the cerebral structures in their immediate neighbourhood. Tumours of the pons; corpora quadrigemina, pineal gland and optic thalamus may all be in a position to do this, and tend to involve the nuclei of both sides. Again, all the nuclei lie near the floor of parts of the ventricular system, being situate in the grey matter of the third and fourth ventricles and connecting iter, consequently they may be irritated by ventricular inflammation and compressed by large ventricular effusions.

**Infranuclear ophthalmoplegia** is produced by lesions of the nerves below the level of the nuclei.

After leaving its nucleus and before appearing at the base of the brain, the third nerve traverses the crus cerebri. The motor tract of the opposite side of the body also traverses the crus, so the typical paralysis produced by a lesion of this part of the brain is a crossed or alternate paralysis, the third nerve palsy being

associated with paralysis of face and limbs on the opposite side. The nerve fibres passing to their point of exit in the oculomotor groove of the crus at first converge from the separate portions of the compound nucleus, and so a lesion in the crus may involve only a few of the converging rootlets, the ophthalmoplegia will then be incomplete and practically indistinguishable from a lesion of part of the compound nucleus itself. Again, of all the motor fibres in the crus those of the face are nearest the third nerve, and so a small lesion may pick out the third nerve and the facial fibres, the rest of the motor tract escaping. The resulting facial paralysis is in all cases of supranuclear type, the lower part of the face being chiefly involved.

It is sometimes difficult to distinguish a lesion in the crus itself from one at the base of the brain which compresses it; in such cases the presence of symptoms due to involvement of the corpora quadrigemina which form the dorsal part of the crus enable the lesion to be located in the crus itself.

The fourth nerves after leaving their nuclei, skirt the iter, decussate and appear on the dorsal aspect of the brain at the valve of Vieussens. They thus come into relation with structures far removed from the other ocular nerves, and consequently the superior oblique muscles may be paralysed by the pressure of a tumour of the anterior end of the cerebellar vermis, or of the posterior quadrigeminal bodies, or of the valve of Vieussens. Both nerves are likely to be involved in such cases.

The sixth nerve on its way from its nucleus to its point of exit at the lower border of the pons, traverses the lower and ventral part of the latter. Its paralysis may be associated with opposite hemiplegia if the lesion be in the pons.

At the base of the brain the third nerves lie in the narrow interpeduncular space, and both may be compressed by a growth here or by an aneurysm of the posterior cerebral artery.

The fourth nerve winds round the outer aspect of the crus.

The sixth nerve has the longest course in the meninges coming into relation with the ventral aspect of the pons,

consequently paralysis of the sixth nerve, *i.e.*, of the external rectus, has no great localising value.

In the cavernous sinus the third, fourth and sixth nerves have come together, and are associated with the first division of fifth. A lesion in this region may involve all the nerves mentioned, necessarily on one side only. Thrombosis of the cavernous sinus, growths of the pituitary body, or aneurysms of the intracranial part of the internal carotid, are lesions possible here.

At the sphenoidal fissure the same nerves are close together and the optic nerve adjacent. A basic meningitis or a periostitis may involve them all.

In the orbit the same nerves may be involved by cellulitis, wounds, or rarely growths. In such cases proptosis may be associated, but this may also result from cavernous sinus thrombosis.

If defects of vision are associated with ocular paralyses the nature of the defect should be ascertained. A lateral homonymous hemianopia points to a lesion of the optic tract behind the chiasma, *i.e.*, in the region of the crus or the pulvinar. Bitemporal hemianopia indicates a lesion in the chiasma region.

#### THE FIFTH NERVE.

The fifth being a mixed nerve, both motor and sensory symptoms may result when it is either irritated or paralysed. The sensory fibres, after entering the pons, end in the substantia gelatinosa Rolandi, and in an expanded terminal portion of this structure which is known as the **sensory nucleus**. The cells of termination of the sensory part of the nerve form a column of considerable vertical extent which reaches from the level of the middle cerebellar peduncle to that of the second cervical nerve. (Fig. 17, p. 191.) The whole of this long column is very unlikely to be affected by disease within the pons, and, as a rule, complete anæsthesia in the territory of the fifth is due to a lesion of the collected sensory fibres as they lie in the issuing nerve root.

The **motor nucleus** which lies in the grey matter at the lateral angle of the fourth ventricle, at the level of origin of the nerve root from the pons, may be damaged by gross lesions at this level, or involved together with other bulbar nuclei, in degenerative disease. The nucleus is connected with the motor cortex of the opposite frontal region by fibres which lie in the genu of the opposite internal capsule. (Fig. 12, p. 179.)

The descending root of the fifth, which arises in the grey matter around the aqueduct of Sylvius, is now held to be motor rather than sensory and probably joins the motor division of the nerve. The rare condition known as facial hemiatrophy is thought to be due to disease of this root.

The roots of the nerve, on their way to the surface, intersect the fibres of the middle cerebellar peduncle and appear on the lateral surface of the pons nearer to its upper than its lower border. The position of the issuing root is made use of as an arbitrary point of division between the pons and the middle peduncle. A tumour in this locality may occasion paralysis of the fifth nerve with deafness, from implication of the eighth nerve, and cerebellar symptoms from interference with the cerebellar peduncle.

In the membranes at the base of the brain lie the nerve roots, the Gasserian ganglion and the three terminal divisions. The ganglion bears the same relation to the sensory fibres as a posterior root ganglion does to a sensory spinal nerve. The small motor root does not enter the Gasserian ganglion but passes beneath it to join the mandibular nerve. The position of the ganglion in a hollow near the apex of the petrous bone, renders it liable to implication by tumours or other diseases of the middle or posterior fossa, or of the petrous bone. Gummatous or tuberculous disease of the membranes may involve the issuing root, the ganglion, or the terminal divisions between the ganglion and their foramina of exit.

The ophthalmic division of the fifth traverses the cavernous sinus and the sphenoidal fissure, and is most likely to suffer in conjunction with the motor nerves of the eyeball. It may

be damaged by growths of the adjacent pituitary body, by aneurysm of the internal carotid artery, by periostitis at the sphenoidal fissure, or by lesions in the orbit. The close relation of this nerve to the cavernous sinus constitutes a serious difficulty in the complete removal of the Gasserian ganglion.

The maxillary division, after its exit from the skull by the foramen rotundum, traverses the spheno-maxillary fossa, applies itself closely to the orbital aspect of the palate bone, and enters the infraorbital canal. It may be damaged by bone disease or fractures. Its relation to the maxillary antrum, in the roof of which it lies, accounts for the paralysis caused by maxillary tumours and the neuralgia which may result from antral disease.

The mandibular nerve, unlike the other two divisions, contains motor as well as sensory fibres. This division is rarely the sole seat of paralysis, but is sometimes damaged by fractures or callus of the mandible and by parotid tumours.

The **motor** filaments of the fifth supply the muscles of mastication. Of these the temporals and masseters are superficial, and their action can be gauged by feeling the hardening they undergo when called into action. Wasting of these muscles causes flattening above and below the zygoma, and if they undergo secondary contracture they may interfere with the free opening of the mouth. Loss of power in the pterygoid muscles causes the lower jaw to deviate towards the paralysed side when the mouth is opened, owing to the unbalanced action of the muscles of the opposite side. The tensor palati which appears to derive its motor nerve from the fifth, is probably innervated from the spinal accessory nucleus, and escapes in ordinary fifth nerve paralysis. Paralysis of the mylo-hyoid and anterior belly of the digastric, both of which are supplied by the mylo-hyoid branch of the inferior dental nerve, is not easily recognised clinically, other muscles being also able to elevate the hyoid bone. Rarely irritation of a sensory filament of the fifth causes trismus or tonic contraction of the muscles of mastication. This is said to be especially associated with irritation of the last molar tooth from caries or abscess. It may also



occur when there is disease of the temporo-mandibular articulation, which is supplied by the auriculo-temporal branch of the fifth nerve.

The **sensory** symptoms referable to the nerve may, like the motor, be either irritative or paralytic. The cause of epileptiform neuralgia, such as is treated successfully by excision of the Gasserian ganglion with its attached sensory root is obscure. Similar intense neuralgia may be produced by the irritation of a tumour of the meninges, for the fifth nerve distributes recurrent filaments to them. If its trunk is implicated by the tumour, anæsthesia as well as neuralgia may be expected. When terminal filaments of the nerve are exposed to irritation the pain is often referred to other parts to which the nerve is distributed. Malignant disease of the tongue is well known as a cause of pain referred to the ear, which pain may be accompanied by salivation, also brought about through the agency of the fifth nerve. During eruption of the temporary molars, children may be observed to pull and rub their ears, and otitis may be suspected. Pain in a decayed tooth is often referred to the corresponding tooth in the jaw of the same side. Decayed teeth may also give rise to frontal, infraorbital, mental, or auricular pain. Frontal headache may accompany disease of the nose or its accessory air sinuses. As already indicated, the grey matter of the sensory nucleus is continuous with that from which the cervical nerves spring, and this may account for the radiation of pain from the territory of the fifth to that of adjacent cervical nerves.

Herpes often occurs in the area of distribution of the fifth nerve. When the nasal branch is involved, as is shown by the appearance of the eruption near the tip of the nose, ulceration of the cornea, iritis or even cyclitis may supervene. This association is explained by the fact that nerves for the front parts of the eye are derived from the nasal nerve during its course through the orbit, reaching the eye through the lenticular ganglion.

The rapid destruction of the eye which sometimes occurs in

disease of the ophthalmic division is probably an irritative and not a paralytic phenomenon, and so is not a result of anæsthesia of the cornea, and consequent failure to remove irritating particles.

The areas of the face supplied by the three divisions of the fifth nerve overlap each other; the area supplied by the posterior division of the second cervical nerve (the first has no posterior division) also overlaps the posterior parts of the areas of the first and second divisions of the fifth, while the skin overlying the parotid gland is in part supplied by the great auricular nerve, derived from the anterior division of the second cervical. Consequently the areas of anæsthesia produced by lesions of the several divisions are much smaller than the anatomical distribution would lead us to expect. The boundary between the fifth and cervical areas may be designated as the vertex-ear-jaw line, since it extends from the vertex in front of the ear and along the margin of the mandible. A line drawn through the supraorbital notch to the interval between the two bicuspids of the lower jaw passes over the points of exit of the three divisions of the fifth on the face. The infraorbital division appears close below the lower margin of the orbit, and the mental nerve half way between the alveolar edge and the lower margin of the jaw.

The distribution of the fifth nerve to the mucous membranes of the nose, mouth, and tongue, anterior parts of the palate and fauces, and to the conjunctiva and cornea should be borne in mind. The fifth nerve is not immediately concerned in the sense of smell, but changes in the mucous membrane of the nose, consequent upon lesions of the fifth nerve, may indirectly affect this special sense.

There is no doubt that **taste** fibres are present in the root, and in the first, second, and third divisions of the fifth. It is held that the taste fibres from the anterior part of the tongue travel in the lingual trunk to the point where the chorda tympani leaves it; thence by the chorda tympani to the facial trunk and geniculate ganglion, and from the latter by the great superficial petrosal nerve to the sphenopalatine ganglion,



DIAGRAM OF COURSE OF TASTE FIBRES.

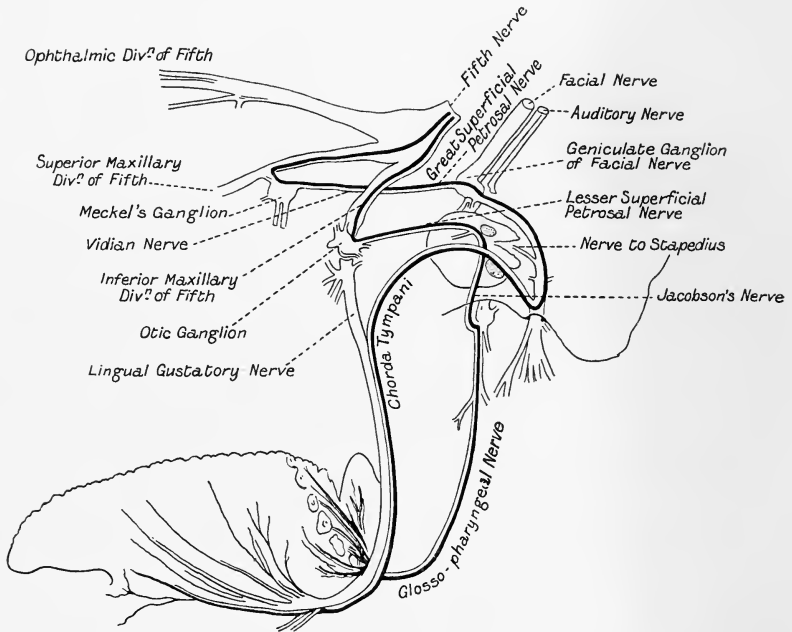


FIG. 18.—The thick black lines indicate the course believed to be taken by the taste fibres. 1. From the front and sides of the tongue through the chorda tympani to the facial trunk, thence through the great superficial petrosal and Vidian nerves to Meckel's ganglion and so to the superior maxillary division of the fifth nerve. 2. From the back of the tongue and neighbouring parts through the glossopharyngeal nerve, its tympanic branch (Jacobson's nerve) and the lesser superficial petrosal nerve, to the otic ganglion and the inferior maxillary division of the fifth nerve.

and second division of the fifth. Thus is explained the fact that a lesion of the petrosal nerve has abolished taste on the anterior part of the tongue.

The taste fibres from the posterior part of the tongue and the circumvallate papillæ travel up in the glossopharyngeal nerve, but leave this by the tympanic branch, and, running in the small superficial petrosal nerve, travel by way of the otic ganglion into the third division of the fifth. (Fig. 18.)

Within the pons the taste fibres are separate from the common sensory fibres of the fifth, and by some unknown path reach the internal capsule of the opposite side, finally terminating in a taste centre at the tip of the temporo-sphenoidal lobe.

Another description of the peripheral course of the taste fibres is given by Edinger. According to this writer the terminal nucleus of taste is a mass of grey matter, which has the same vertical extent as the sensory nucleus of the fifth, and which receives afferent fibres in succession from the glossopharyngeal nerve, through its petrous ganglion; the chorda tympani, through the geniculate ganglion of the facial, and the portio intermedia; and the lingual nerve, through the Gasserian ganglion.

#### FACIAL PARALYSIS AND FACIAL SPASM.

The facial, like other cranial nerves, may be paralysed by supranuclear, nuclear, or infranuclear lesions.

A **supranuclear lesion** may involve the cortex or the conducting fibres from the cortical centre to the facial nucleus. The lower end of each ascending frontal convolution is connected with the opposite facial nucleus by fibres which traverse the genu of the internal capsule and pass along the base of the crus. In the crus the facial fibres are immediately internal to the innermost fibres of the pyramidal system, following the general rule that the fibres which decussate first lie nearest to the mid line of the body.

The cortical centres for articulatory movements of the tongue and movements of the larynx lie in the lowermost part of the

ascending frontal convolution, and in the adjacent part of the third frontal convolution, just below the face centre. The cortical centres for the arm lie in the ascending frontal convolution above the face centre. (Fig. 14, p. 183.) Consequently lesions of the face centre usually involve also the centres for speech and for arm movements. In a similar manner disease of the conducting tracts below the face centre tends to implicate the adjacent fibres for the tongue and limbs. Thus it comes about that the supranuclear facial paralysis usually presents itself as part of a hemiplegia.

The paralysis caused by a supranuclear lesion is incomplete. In this type of paralysis, which is sometimes called "cerebral," the weakness of the lower facial muscles is more obtrusive than that of the orbicularis palpebrarum and frontal portion of the occipito-frontalis muscles. The escape of the upper part of the face is probably explained by the fact that the facial nucleus receives fibres from *both* hemispheres of the brain, and some of these fibres preside over the movements which are retained. There is no reaction of degeneration in the paralysed muscles. Emotional expression in the face and the winking reflex are retained in cortical and subcortical lesions. The optic thalamus appears to be concerned in emotional expression, and the winking reflex depends on the integrity of the afferent fibres of the optic or ophthalmic nerves, the facial nucleus and the efferent infranuclear facial tract. The supranuclear facial tract crosses the mid line about half way down the pons to reach the facial nucleus. (Fig. 13, p. 182.) Lesions involving the tract above the decussation cause incomplete facial paralysis on the side opposite to the lesion.

**Nuclear and infranuclear lesions.**—Lesions below the decussation in the pons affect the face on the same side as that on which they occur, and since they are near the nucleus of the nerve usually cause complete paralysis.

The facial nucleus lies in the ventral part of the tegmentum, near the junction of the pons with the medulla. (Fig. 17, p. 191.) Isolated lesions are rare. There is reason to suspect that the fibres which the facial nerve distributes to the orbicularis oris

are really derived from the hypoglossal, and not from the facial nucleus. It is also possible that the fibres for the orbicularis palpebrarum and the frontalis muscle arise in the posterior part of the third nerve nucleus, and join the facial fibres by way of the posterior longitudinal bundle. If these statements are correct, in an isolated nuclear lesion of the facial nerve, the muscles thus innervated from other nuclei should escape.

The radicular fibres of the facial nerve may be injured in the pons after they have emerged from the nucleus. They form a bundle which does not pass direct to its point of exit, but first sweeps around the nucleus of the sixth nerve. If these fibres are damaged the facial paralysis which results is complete, involving all the muscles of the face. Associated with the paralysis of the face may be conjugate deviation of the eyes away from the lesion, and weakness of the tongue and limbs on the opposite side of the body. The conjugate deviation is due to damage to the sixth nucleus as it lies in the loop formed by the radicular fibres of the facial nerve, and the weakness of tongue and limbs is caused by implication of the adjacent pyramidal tract.

Complete facial paralysis is characteristic of infranuclear lesions. It is sometimes termed radicular or peripheral. It differs from the cerebral type in that the orbicularis palpebrarum and the frontalis muscles are as much paralysed as the rest of the face. Consequently the eye on the affected side cannot be properly closed, although the associated upward movement of the eyeball appears on the attempt to do so. The tears of the affected eye run down the cheek, either because the weakened lids fall away from the globe so that the puncta lachrymalia are not kept in proper position, or because the paralysis of Horner's muscle renders difficult the passage of the tears into the nasal duct. In both types of facial paralysis the failure of the buccinator allows food to collect between the cheek and gums, and, owing to the drawing of the mouth towards the sound side, the tongue may appear to deviate from the mid line when protruded. It still, however, maintains its proper relation to the

front teeth. There is a reaction of degeneration in the paralysed muscles in the complete type of paralysis, since they are severed from the facial nucleus.

At the base of the brain the facial emerges close to the auditory nerve, the latter lying external and the portio intermedia between the two. The nerves appear at the lower border of the pons, immediately above the restiform body, and they pass together into the internal auditory meatus. As they pass towards the meatus the facial lies in a groove on the upper and fore part of the auditory trunk, whilst the pars intermedia and the auditory artery lie between them. The close relation of the nerves explains why gummatous and other new formations involving the dura at the base of the skull are so likely to cause both facial and auditory paralysis. Lesions of the facial trunk in this situation should not involve the fibres which subserve taste, being above the level at which these are generally supposed to pass into the great superficial petrosal nerve on their way to the fifth trunk.

A few cases of facial nerve paralysis are due to inflammation or caries of the tympanum. The aqueduct of Fallopius which transmits the facial trunk lies in the inner wall of the tympanum, behind the foramen ovale, forming the inner boundary of the isthmus of communication between the attic and the antrum. The wall of the aqueduct is occasionally incomplete, which accounts for the readiness with which the nerve suffers in some cases of otitis, moreover the apertures which transmit the chorda tympani, the nerve to the stapedius and the stylomastoid artery may allow invasion of the aqueduct from the middle ear. The nerve is liable to damage in this position from operations on the mastoid, and the external semicircular canal, which lies immediately behind and above the Fallopian aqueduct, may be injured at the same time. Fractures of the base of the skull may cause facial paralysis when they traverse the petrous bone; in fact the facial nerve is more often paralysed in fracture of the base than any other cranial nerve. Impairment of taste on the front part of the tongue indicates a lesion of the facial



nerve in such a position as to involve the trunk between the point where the chorda tympani joins it and the point where the great superficial petrosal nerve leaves the geniculate ganglion, since the taste fibres leaving the facial nerve pass by the petrosal nerve to enter the brain by way of the fifth nerve. (Fig. 18, p. 199.)

In the adult the trunk of the facial nerve emerges from the stylomastoid foramen in a protected position, but in the infant the opening of the canal is practically exposed on the lateral aspect of the skull and is liable to injury by the forceps during delivery. The posterior auricular branch, which supplies the *retrahens aurem* and the *occipitalis* muscles is given off immediately after the exit of the nerve, so these muscles occasionally escape when the nerve is injured near the mandible by wounds or direct violence. The nerve trunk enters the parotid gland just above the level of the tip of the lobule of the ear, hence it may be involved by parotid tumours or inflammations, or by vertical incisions into the gland carried above the level indicated. The nerve had also been compressed by gummatous and lymphatic glandular swellings in this region and injured by operations undertaken for removal of the latter. The exposed position of the filaments of distribution on the face is no doubt a contributing factor in the production of the ordinary or so-called rheumatic type of facial paralysis. It is doubtful whether the usual explanation that the nerve is compressed in the aqueduct of Fallopius on account of its own swelling is a valid explanation of the production of this form of palsy.

Although the facial nerve is usually considered and described as purely motor in function certain **sensory** symptoms may accompany facial paralysis. The defect of taste on the fore part of the tongue has already been mentioned. There may also be some defect of smell on the paralysed side, which may be attributed to weakness of the sniffing movements of the nostril on that side. Defect of hearing when present may be due to inflammation of the tympanum or to simultaneous implication of the facial and auditory nerves at the base of the brain. When the stapedius muscle is paralysed the unbalanced action of the

tensor tympani increases the sensitiveness of the ear to musical tones. Neuralgic pain at the back of the ear and in the teeth and gums of the paralysed side may accompany facial paralysis. The anatomical explanation is obscure but it has been supposed to be due to irritation of sensory fibres of the fifth nerve which have joined the facial nerve in the Fallopian canal.

The pathology of **facial spasm** is obscure, but it is sometimes due to reflex irritation from the territory of the fifth nerve and particularly from its orbital branches. Exceptionally irritation of the facial nerve near its origin from the pons or of the cortical facial centres originates the spasm.

#### THE AUDITORY NERVE.

The auditory nerve consists of two distinct portions; of these one is called **vestibular** and is concerned in equilibration or orientation of the body, and the other is called **cochlear** and concerned in hearing. The ampullary and cochlear portions of the labyrinth in which these nerves respectively commence are continuous, and the combined nerves form a common trunk as far as the surface of the pons; the nuclei of termination in the fourth ventricle are also closely adjacent, but the ultimate central terminations of the two nerves are widely separated. Hence in labyrinthine lesions and lesions of the common auditory trunk both portions of the nerve are equally exposed to injury, and it is usual to find that impairment of hearing is associated with vertiginous sensations. The latter, especially if of sudden onset, may be so obtrusive that for some time the accompanying deafness may be overlooked, the more so since in labyrinthine lesions the defect may only be marked in the case of the higher pitched sounds.

The organ of hearing being enclosed in the petrous bone can receive and appreciate vibrations directly from the bone. This fact is taken advantage of when a tuning fork or watch is applied directly to the mastoid or vertex in testing nerve conduction, the failure to appreciate vibrations applied in this way being an

indication of nerve deafness. The presence of the internal carotid artery in a canal in the petrous bone probably accounts for the pulsatile tinnitus which occurs in some cases.

Inflammatory disease of the labyrinth may originate in it or extend to it from the middle ear through the foramina in the inner wall of the tympanum, or pass from within the skull along the sheath of the auditory nerve, for the subarachnoid space around the auditory nerve communicates with the perilymph spaces of the internal ear. Such an extension is not uncommon in leptomeningitis.

A combination of nerve deafness with facial paralysis and suppuration of the middle ear indicates that the inflammation involves the bony labyrinth. Paralysis of the same two nerves with a healthy tympanum would lead to the inference that the lesion was in the internal meatus or at the base of the brain. New growths in the petrous bone or posterior fossa of the skull, gummatous and other varieties of meningitis in this locality, and occasionally an aneurysm may involve the facial and auditory nerves at the base. Sometimes the sixth and other adjacent nerves are also paralysed. It has been observed that the facial nerve (*portio dura*) is more resistant to the effects of surrounding inflammation than the eighth nerve (*portio mollis*).

The fibres of the vestibular nerve are derived from the semi-circular canals, the utricle and the saccule. After traversing the local ganglia in the petrous bone they are continued through the internal meatus towards the restiform body. Up to this point, as already mentioned, both parts of the auditory nerve are likely to suffer together. On reaching the surface of the brain the vestibular fibres diverge from the cochlear fibres to enter the pons on the ventral aspect of the restiform body, which thus becomes interposed between the two portions of the auditory nerve. The vestibular fibres terminate in certain nuclei in the outer angle of the fourth ventricle, including Deiter's nucleus. (Fig. 17, p. 191.) The fibres are thus brought into connection with other parts of the mechanism of equilibration, including the cerebellum, the ocular nuclei and the antero-lateral columns of the spinal cord. These

complex connections explain the extreme vertigo and inco-ordination induced by sudden lesions of the vestibular nerve.

The fibres of the cochlear or auditory portion of the nerve commence in Corti's organ in the cochlea and join the spiral ganglion of the latter. Thence fibres traverse the meatus in company with the vestibular portion of the nerve. Diverging finally from the vestibular nerve and passing dorsal to the restiform body, the fibres terminate in a couple of ganglia closely applied to the latter and known as the acoustic tubercle and the ventral cochlear nucleus.

In localising the position of a lesion in the pons or higher parts of the brain which causes deafness, a knowledge of the course taken by the auditory impulses from the nuclei in the pons to the auditory perceptive centres in the temporal lobes is essential, since it is only by the associated symptoms that the localisation can be effected.

Most of the fibres from the nuclei, but not all, cross to the opposite side of the pons, some passing dorsally through the striæ acousticæ which are visible on the floor of the fourth ventricle and other passing more ventrally in the trapezoid body. Uniting in the lateral fillet this double set of crossed fibres passes through the internal geniculate body, traverses the sensory limb of the internal capsule and passes thence by the corona radiata to the superior temporo-sphenoidal convolution. The afferent tracts are also connected with the superior olivary bodies and the posterior corpora quadrigemina. The whole tract consists of an elaborate system of nerve cell relays. It will be gathered that each ear is connected not only with the auditory centre of the opposite side but also with that of the same side.

The association of word deafness with a certain amount of defect of hearing in the right ear points to the left temporal lobe as the seat of disease. Deafness from a lesion of the internal capsule is associated with hemianopia and hemianæsthesia since the auditory fibres are in immediate relation here with the visual tract and the fibres of common sensation.

Deafness from a lesion in the corpora quadrigemina or from

pressure on the adjacent fillet, such as might occur from quadrigeminal tumours or enlargement of the pineal body, will be associated with defects in the movements of the eyes and other symptoms referable to the corpora quadrigemina.

Deafness from involvement of the auditory tract in the pons is rare, probably because lesions of the pons usually occur in the tegmental or dorsal region, whilst the auditory tract lies lateral in the lateral fillet and ventral in the corpus trapezoides. The occurrence of paralysis of other cranial nerves which arise from the pons and of opposite hemiplegia will localise the lesion sufficiently.

#### THE GLOSSOPHARYNGEAL, VAGUS, BULBAR AND SPINAL ACCESSORY NERVES.

From both the anatomical and the clinical points of view the glossopharyngeal, vagus and bulbar portion of the spinal accessory nerves are best considered together as forming parts of one large nerve. The fibres are mixed, some being afferent or sensory, and others efferent or motor in function.

Unilateral lesions of the motor cortex or motor tracts above the motor nucleus of the nerve do not produce any appreciable paralysis of the larynx, pharynx or palate to which the nerve is distributed, but if *both* cortical centres or supranuclear tracts be damaged a condition known as pseudo-bulbar paralysis is induced. Hence the inference that the bulbar nucleus is connected with each hemisphere of the brain.

The motor fibres arise from the **nucleus ambiguus** which extends from the level of the highest issuing roots of the glossopharyngeal down to the point of decussation of the fillets which is a short distance above the decussation of the pyramids. (Fig. 17, p. 191.) From this nucleus the motor fibres for the larynx, pharynx and soft palate arise, consequently it has been termed the nucleus of phonation and deglutition. The unity of the nucleus explains the combination of paralyse met with in bulbar palsy, *i.e.*, of larynx, soft palate and pharynx, to which may be added paralysis of the tongue and lips if the hypoglossal nucleus is also affected.

Chronic nuclear degeneration producing this type of paralysis may exist alone or be a part of tabes, general paralysis, syringomyelia or amyotrophic lateral sclerosis. Acute affections of the nucleus may occur in hæmorrhage or softening of the medulla. Since the reflex arc is interrupted, loss of the palatine, pharyngeal and laryngeal reflexes may be expected in nuclear lesions, whereas they are retained in lesions above the level of the nucleus.

In the membranes at the base of the brain and also at the base of the skull, the glossopharyngeal, vagus and bulbar accessory nerves are close together and are in addition joined by the spinal portion of the accessory nerve. The last nerve is distributed to the sternomastoid and trapezius, consequently the involvement of these two muscles as well as the larynx, palate and pharynx points to a lesion of the membranes or bones of the base. Basal meningitis, basal tumours and aneurysms of the vertebral artery are lesions which may cause this combination of paralyses.

The action of the **laryngeal motor mechanism** is easily investigated with the laryngoscope and may afford valuable indications not only in disease of the bulbar nuclei but also in certain intrathoracic diseases such as tumour and aneurysm which also cause laryngeal paralysis.

The superior laryngeal nerve, which conveys motor impulses to the crico-thyroid muscle, is also the sensory nerve of the upper part of the larynx. The nerve trunk is comparatively short and running deeply is well protected, consequently isolated paralysis of the crico-thyroid muscle is exceptional. Suicidal wounds of the neck and tumours or enlarged glands in this position may involve the nerve. The crico-thyroid may also be paralysed by lesions of the vagus at or above the level of the ganglion of the trunk from which the superior laryngeal nerve is given off, but then the other laryngeal muscles will also be affected. The function of the crico-thyroid is to tilt backwards the cricoid cartilage, and so render tense the vocal cords.

The motor fibres for the other laryngeal muscles run in the recurrent laryngeal nerves. The long course of these nerves exposes them to damage in widely separated localities. Bilateral

paralysis may be produced by cancer of the upper parts of the œsophagus or by tumours of the thyroid gland or operations at the root of the neck on either of these structures, for both recurrent nerves are in close contact with the sides of the gullet and the thyroid. The left recurrent nerve, winding as it does round the aortic arch, is frequently compressed by aortic aneurysms, whilst aneurysms of the innominate or right subclavian arteries may paralyse the right nerve, but this is more commonly implicated in glandular enlargements within the thorax and may be damaged by phthisis near the pleural dome.

In progressive lesions of the recurrent laryngeal nerve the muscles usually fail in a definite order according to Semon's law, the abductors first and then the adductors. The nerve bundles in the recurrent laryngeal trunks for the abductor and the adductor groups are distinct, but this anatomical arrangement does not altogether explain the greater vulnerability of the abductors. They have been compared in this respect to the extensor muscles of the limbs.

In abductor paralysis the paralysed cord during inspiration appears both shorter and higher than its fellow. This appearance is due to the sloping surface of the cricoid facet on which the arytenoid cartilage rests, so that when the abductors come into play the latter cartilage is dragged downwards as well as backwards and outwards. The free edges of the true cords are directed upwards as well as inwards, and when the abductors of both sides are paralysed the inspiratory rush of air tends to drive the cords together like a valve, producing urgent dyspnoea.

The arytenoideus muscle escapes in unilateral laryngeal paralysis, possibly because it is supplied by both recurrent nerves.

Paralysis of the **soft palate** also points to an affection of the motor fibres of the vago-glossopharyngeal group. The lowermost fibres are those concerned, and these are usually spoken of as the bulbar accessory fibres. They arise from the nucleus ambiguus and supply the levator palati, probably reaching the palate through the pharyngeal plexus. Hence the palate is paralysed

in disease of the bulbar nuclei or of the nerve roots in the subdural space. Like the pharynx it may also be paralysed by peripheral neuritis such as occurs in diphtheria. Unlike the other palatine muscles, the tensor palati is held to be supplied by the motor division of the fifth nerve through the otic ganglion. Care must be taken not to mistake asymmetry of the soft palate for paralysis; defect of movement is the only safe diagnostic guide.

The afferent fibres of the glossopharyngeal nerve end in the posterior or dorsal vago-glossopharyngeal nucleus subjacent to the trigonum vagi, and in the fasciculus solitarius or ascending root of the glossopharyngeal which, in many respects, resembles the large ascending root of the fifth nerve.

Irritative symptoms referable to the vago-glossopharyngeal group are more common than paralyses. They include cough, vomiting, laryngeal spasm, vertigo and derangements of cardiac action.

It appears that cough may be reflexly induced from any territory supplied by branches of the vagus and glossopharyngeal nerves, and sometimes from other regions as well. The glossopharyngeal filaments from the tonsil, pharynx and middle ear, and the vagal filaments from the auditory meatus, pharynx, œsophagus, stomach, larynx, trachea, bronchi, lungs, and possibly also the filaments from the pericardium and heart may convey impulses which originate cough.

As with cough, so with vomiting; it is well known that irritation of the fauces, pharynx or auditory canal may induce this as also may lesions of the lungs, mediastina, pleuræ, heart, pericardium, stomach, liver, kidneys, pancreas and adrenals, to all of which the vagus distributes filaments.

Laryngeal spasm as the result of sensory irritation is seen in connexion with foreign bodies and inflammations of the larynx, pressure on the trachea and bronchi, and as a symptom of tabes.

Cardiac irregularities have been found in some instances to be due to tumours implicating the cardiac plexuses. Fatal cardiac arrest sometimes follows exploratory puncture of the chest owing to reflex inhibition of the heart.



## THE HYPOGLOSSAL NERVE.

As with other motor nerves, paralysis of the hypoglossal may be of supranuclear, nuclear, or infranuclear origin.

**Supranuclear Paralysis.**—The cortical centre for the tongue lies at the posterior extremity of the third left frontal convolution, and is connected by the pyramidal fibres with the hypoglossal nucleus of the opposite side. Lesions of the centre or of the conducting tracts above the nucleus rarely affect the tongue alone but usually give rise to a hemiplegia which is more or less complete. The reasons for this have already been given when dealing with the other motor cranial nerves.

**Nuclear Paralysis.**—The hypoglossal nucleus lies ventral to the central canal of the cord, and when the canal opens up into the fourth ventricle the nucleus lies subjacent to the trigonum hypoglossi. (Fig. 17, p. 191.) The nucleus is implicated by the same types of lesion as those already indicated when dealing with the vago-glossopharyngeal nucleus, indeed the two nuclei being closely adjacent usually suffer together. The results of intracranial disease have led to the suggestion that the hypoglossal nucleus gives rise to the motor fibres of the lips as well as to those of the tongue.

**Infranuclear paralysis** of the hypoglossal may be the result of a lesion of the medulla where this is traversed by the issuing nerve roots. In such a case there is every likelihood that the adjacent pyramidal tract will be damaged so that a crossed paralysis of tongue on side of lesion and limbs on the opposite side of body will be produced.

Lesions in the subdural space will implicate the nerve somewhere between its points of exit in the groove between the pyramid and the olive and its passage through the anterior condyloid foramen. This foramen is only separated from the jugular foramen by the bony eminence known as the eminentia innominata. Given a lesion in this neighbourhood the adjacent vago-glossopharyngeal fibres are almost certain to suffer at the same time as the hypoglossal, so that a combined unilateral paralysis of the

tongue, soft palate and vocal cord results. A lesion outside the base of the skull may involve the same nerves after their exit and produce paralysis of similar distribution. Tumours and inflammatory affections at the base of the skull, disease of the occipito-atloid joint and caries of the highest cervical vertebræ may all implicate the hypoglossal and adjacent nerves in this way.

Unilateral paralysis of the tongue results in its deviation towards the paralysed side when protruded owing to the unbalanced action of the sound genio-glossus muscle. When at rest in the mouth the root of the tongue appears higher on the paralysed side from loss of tone in the hyoglossus of that side. Unless the paralysis is bilateral there is but little interference with the power of chewing, deglutition or articulation. If the lesion be nuclear or infranuclear the tongue wastes, and as a result the mucous membrane of the affected half appears too large for it and is thrown into folds. There is also a reaction of degeneration in the affected muscles.

## THE SPINAL CORD.

### MYELITIS.

In most cases myelitis appears to be due to an infective agent carried to the spinal cord by the blood stream. The rarity with which inflammation of the spinal cord follows injury is no doubt due to the efficient way in which it is protected by the vertebral canal, and the intervention of the membranes with their fluid contents between the cord and its bony sheath. Notwithstanding this, inflammation of the cord has been known to follow falls from a height on to the feet, blows, twists or wrenches of the spine, quite apart from fractures and dislocations.

Inflammation may spread directly to the cord from the vertebral canal, or from the encasing membranes, and, in the latter case, may show an annular peripheral distribution which is no doubt brought about by the way in which the pial septa penetrate the periphery of the cord carrying with them small vessels.

Inflammation may also travel to the cord along the connective tissue sheaths of the nerves, and the so-called "reflex urinary paraplegia" is probably an example of this method of invasion.

The distinction of a mere softening of the cord, the result of vascular occlusion, from the softening which is the result of inflammation is not at all easy. The myelitis which is met with within a couple of years of syphilitic infection is, with considerable reason, regarded by many as a primary vascular softening. In such cases the vascular obstruction must be the result of endarteritis of minute terminal vessels in the cord, for there is a free vertical anastomosis not only of the spinal arteries on the surface of the cord but also, to a less degree, of the larger arteries in the cord itself.

As the result of myelitis the nervous structures at the site of the disease are more or less destroyed, but, in addition to this, certain secondary degenerations of the cord are found. These secondary degenerations occupy definite tracts in the white matter, and some ascend from the site of lesion whilst others descend. The degeneration of these tracts is dependent on their severance from their trophic cells. The descending degenerations involve the crossed and direct pyramidal tracts, whilst tracts which undergo ascending degeneration are the direct cerebellar tract, the antero-lateral ascending tract of Gowers, and the tracts which form the postero-internal and postero-external columns of the cord, or the column of Goll and the column of Burdach. (Fig. 19, p. 222.) The lumbo-sacral fibres of the postero-external column do not pass up to the bulb in this column, but after a short upward course enter the postero-internal column and complete their ascent in this. Consequently the postero-external column is only degenerated for a short distance above the lesion, whilst the degeneration in the postero-internal column passes as high as the grey matter of the nucleus gracilis. The higher the lesion the more complete the degeneration of the postero-internal column, since the nerve fibres which enter it from the upper lumbo-sacral sensory roots are affected as well as the lower.

But inflammation may also extend from the local lesion for a

TABLE OF REPRESENTATION OF MUSCLES IN SPINAL SEGMENTS.

	C.1	2	3	4	5	6	7	8	D.1	—	—	—
Small flexors of head .. .. .	x	x	..	..	..	..	..	..	..	..	..	..
Depressors of hyoid bone .. .. .	x	x	..	..	..	..	..	..	..	..	..	..
Small rotators of head .. .. .	x	x	x	..	..	..	..	..	..	..	..	..
Complexus .. .. .	x	x	x	..	..	..	..	..	..	..	..	..
Splenius .. .. .	x	x	x	..	..	..	..	..	..	..	..	..
Sterno-mastoid .. .. .	..	x	x	..	..	..	..	..	..	..	..	..
Levator anguli scapulae .. .. .	..	..	x	x	x	x	..	..	..	..	..	..
Scaleni .. .. .	..	..	x	x	x	x	..	..	..	..	..	..
Trapezius .. .. .	..	?	x	x	x	?	..	..	..	..	..	..
Diaphragm .. .. .	..	..	x	..	x	..	..	..	..	..	..	..
Rhomboids .. .. .	..	..	..	?	?	..	..	..	..	..	..	..
Supraspinatus .. .. .	..	..	..	?	?	x	..	..	..	..	..	..
Infraspinatus .. .. .	..	..	..	..	..	x	..	..	..	..	..	..
Teres minor .. .. .	..	..	..	..	x	..	..	..	..	..	..	..
Coraco-brachialis .. .. .	..	..	..	..	x	..	..	..	..	..	..	..
Biceps .. .. .	..	..	..	?	x	..	..	..	..	..	..	..
Brachialis anticus .. .. .	..	..	..	..	x	?	..	..	..	..	..	..
Deltoid .. .. .	..	..	..	?	x	?	..	..	..	..	..	..
Supinator longus .. .. .	..	..	..	?	x	?	..	..	..	..	..	..
Supinator brevis .. .. .	..	..	..	..	x	?	..	..	..	..	..	..
Pectoralis major .. .. .	..	..	..	..	x	x	x	..	..	..	..	..
Pectoralis minor .. .. .	..	..	..	..	x	x	x	..	..	..	..	..
Serratus magnus .. .. .	..	..	..	..	x	?	?	..	..	..	..	..
Subscapularis .. .. .	..	..	..	..	?	x	..	..	..	..	..	..
Teres major .. .. .	..	..	..	..	..	x	?	..	..	..	..	..
Pronator radii teres .. .. .	..	..	..	..	..	x	?	?	..	..	..	..
Pronator quadratus .. .. .	..	..	..	..	..	x	?	?	..	..	..	..
Latissimus dorsi .. .. .	..	..	..	..	..	x	x	..	..	..	..	..
Triceps .. .. .	..	..	..	..	..	x	x	?	..	..	..	..
Extensors of wrist .. .. .	..	..	..	..	..	?	x	x	..	..	..	..
Extensors of fingers .. .. .	..	..	..	..	..	?	x	x	..	..	..	..
Flexors of wrist .. .. .	..	..	..	..	..	..	x	x	..	..	..	..
Flexors of fingers .. .. .	..	..	..	..	..	..	x	x	..	..	..	..
Interossei .. .. .	..	..	..	..	..	..	x	x	x	..	..	..
Thenar muscles .. .. .	..	..	..	..	..	..	..	x	x	..	..	..
—	D.1	2	3	4	5	6	7	8	9	10	11	12
Intercostals .. .. .	x	x	x	x	x	x	x	x	x	x	x	x
Pupil dilator .. .. .	?	x	x	..	..	..	..	..	..	..	..	..
Rectus abdominus .. .. .	..	..	..	..	x	x	x	x	x	x	x	x
Obliquus externus .. .. .	..	..	..	..	..	..	x	x	x	x	x	x
Obliquus internus .. .. .	..	..	..	..	..	..	x	x	x	x	x	x
Transversalis .. .. .	..	..	..	..	..	..	x	x	x	x	x	x
—	L.1	2	3	4	5	S.1	2	3	4	5	—	—
Quadratus lumborum .. .. .	x	?	..	..	..	..	..	..	..	..	..	..
Cremaster .. .. .	..	x	?	..	..	..	..	..	..	..	..	..
Ilio-psoas .. .. .	..	x	x	..	..	..	..	..	..	..	..	..
Pectineus .. .. .	..	x	x	..	..	..	..	..	..	..	..	..
Sartorius .. .. .	..	x	x	..	..	..	..	..	..	..	..	..
Adductors of thigh .. .. .	..	x	x	..	..	..	..	..	..	..	..	..
Quadriceps extensor .. .. .	..	..	x	x	..	..	..	..	..	..	..	..
Gracilis .. .. .	..	..	..	x	..	..	..	..	..	..	..	..
Small external rotators of thigh .. .. .	..	..	..	..	x	..	..	..	..	..	..	..
Gluteus medius and minimus .. .. .	..	..	..	x	x	x	..	..	..	..	..	..
Gluteus maximus .. .. .	..	..	..	..	x	x	x	..	..	..	..	..
Hamstrings .. .. .	..	..	..	..	x	x	x	..	..	..	..	..
Gastrocnemius .. .. .	..	..	..	..	?	?	x	x	..	..	..	..
Soleus .. .. .	..	..	..	..	..	?	x	..	..	..	..	..
Extensors of foot and toes .. .. .	..	..	..	..	x	x	..	..	..	..	..	..
Flexors of foot and toes .. .. .	..	..	..	..	?	x	?	..	..	..	..	..
Peroneus longus .. .. .	..	..	..	x	..	..	..	..	..	..	..	..
Peronei .. .. .	..	..	..	..	..	x	?	..	..	..	..	..
Intrinsic muscles of foot .. .. .	..	..	..	..	..	x	x	..	..	..	..	..
Muscles of perineum .. .. .	..	..	..	..	..	..	..	x	x	..	..	..
Sphincter ani .. .. .	..	..	..	..	..	..	..	x	x	..	..	..



short distance upwards along tracts which should degenerate downwards and *vice versâ*. For instance the pyramidal tracts may be found degenerated upwards for a short distance. This is probably due to a direct spread of inflammation in the neuroglial planes, and is quite comparable to the way in which inflammation spreads by continuity in other parts of the body, and of the same nature as the inflammation which is found in the nerve roots, which arise from the cord at the site of the myelitis.

Two distinct groups of symptoms occur in myelitis. The first group comprises motor and sensory phenomena which are due to the interference with the transmission of impulses up and down the cord, the second group consists of symptoms due to the destruction of local centres at the site of the myelitis. Anæsthesia and loss of power below the site of the lesion belong to the first group, whilst local atrophy of muscles with loss of electrical reactions in them and abolition of reflexes are indications of local destruction. A zone of hyperæsthesia just above the level of the lesion is due to irritation of the sensory roots at the upper level of the myelitis. As a rule the reflexes of the parts below those actually supplied by the diseased segments of the cord are exaggerated and the nutrition of the muscles fairly preserved, but there is reason to believe that when the lesion is completely transverse, which is equivalent to a transection of the cord, the reflexes below the lesion are abolished.

The upper limit of a myelitis is best determined by the situation of the upper limit of anæsthesia, whilst the vertical extent of the lesion can be gauged by the loss of reflexes and distribution of muscular wasting.

The dorsal region of the cord supplies nerves to the intercostals, the long muscles of the back and the flat muscles of the abdomen. Wasting of individual intercostal muscles is not easily observed, and the long muscles of the back and flat muscles of the abdominal wall do not appreciably waste since they receive nerves from a great number of dorsal segments.

The cervical and lumbar enlargements supply muscles of the limbs, and detection of wasting and of the reaction of degeneration

in these muscles is a simpler matter. Cervical myelitis in the region of the fourth segment causes paralysis of the diaphragm, and since the impulses to the intercostal muscles are also blocked by the same lesion, death results from respiratory failure. A cervical myelitis is also liable to spread to the adjacent medulla involving the vital centres in this region.

A myelitis so situated as to involve the region of the cervical cord which supplies the arm may give rise to alterations in the size of the pupil, as will be explained in the section on compression of the cord.

It is not easy to give a simple description of the way in which cord inflammations affect the mechanism of the urinary bladder. If that part of the cord corresponding to the origin of the third and fourth sacral nerves be involved there is no power to retain the urine, which dribbles away almost as soon as it enters the bladder, because the sphincter is paralysed. When the lesion is at the junction of the dorsal and lumbar regions there is retention with overflow. When the lesion is above the ninth dorsal segment the act of micturition may occur automatically as soon as a fair quantity of urine has accumulated in the bladder, the patient being quite unconscious of the act. In this case all the local bladder centres are intact, but their higher connections are severed.

#### ACUTE ANTERIOR POLIOMYELITIS.

This disease, commonly known as **infantile paralysis**, is due to an acute lesion of the motor cell groups of the anterior cornua of the spinal cord. The grey matter of the anterior cornua, and also that forming the base of the posterior cornu, is supplied with blood by branches derived from the anterior spinal artery, so the lesion occurs in the territory supplied by that vessel. (Fig. 19, p. 222.) The febrile onset of the disease suggests an infective inflammation, but in a few of the cases there is no fever, and this has led to a suggestion that these may be of thrombotic origin.

The motor cells of the anterior cornua are arranged in definite

groups. These groups are most numerous in the cervical and lumbar enlargements and extend in a vertical direction through a varying number of cord segments, some extending through as many as three or four whilst others are limited to one. It is assumed that each cell group represents a particular muscle. If the cell group is entirely destroyed the muscle is completely paralysed, whilst partial destruction, which is more likely to occur when the cell group extends through several segments, leads to paralysis of part of the muscle only.

Several types of infantile paralysis may be recognised, depending upon the position occupied by the spinal lesion.

In the *leg type* the peroneal and the anterior tibial groups of muscles are involved, occasionally the posterior tibial group, including the calf muscles, as well. Reference to the table of muscular representation in the cord, indicates that the lesion in such cases is in the lower lumbar and sacral regions. The functions of the bladder and rectum may be interfered with owing to the proximity of the centres for these organs. The resulting deformities affect the ankle and foot. Owing to the continued unopposed activity of the calf muscles talipes equinus may be produced. When paralysis preponderates in the peronei, the foot becomes intumed, and talipes varus occurs, because of the unopposed action of the anterior tibial group.

In the *thigh type* of paralysis the iliacus, psoas, quadriceps extensor and glutei suffer, the adductors often escaping. The lumbar region of the cord is the site of the lesion in such cases. Owing to the unopposed action and ultimate contracture of the hamstrings, the knee may become fixed in a position of flexion. The head of the femur may fall from its proper position in the acetabulum, and even become further displaced. Paralysis limited to the muscles of the thigh produces serious disability owing to the position of the paralysed segment at the junction of the trunk and lower limb. Under these circumstances the part of limb below the knee is practically useless although its muscles are intact.

The arm may be paralysed in either its upper or lower



segments or both. In paralysis of the *lower arm type* the flexors and extensors of the wrists and fingers and the small muscles of the hand are involved. Sometimes the intrinsic muscles of the hand suffer alone. The supinator longus usually escapes. The lesion in the cord occupies the seventh and eighth cervical and the first dorsal segments. The deformity known as claw hand may result.

In paralysis of the *upper arm type* the scapular muscles, deltoid, triceps and supinator longus are involved. The lesion corresponds to the fifth and sixth cervical segments. The distribution of the paralysis is very similar to that met with as the result of lesions of the fifth and sixth cervical roots in the posterior triangle of the neck. The paralysis of the deltoid allows the humerus to fall away from the glenoid cavity simulating dislocation. This deformity is more marked if the supraspinatus as well as the deltoid is paralysed.

When the spinal muscles are paralysed, certain curvatures of the spine occur, owing to loss of muscular support and the unopposed action of unparalysed muscles. Spinal curvature may also result from tilting of the pelvis when muscles attached to this are weak.

The paralysed limbs often show vaso-motor disturbance, stunted growth and thinning of the bones. These changes are due to the involvement of the centres in the cord which supply vaso-motor and trophic fibres and also in part to disuse of the limbs. Pain is sometimes complained of at the onset of the disease, actual anæsthesia is found in rare cases, and persistent hyperalgesia is not uncommon. Diffusion of the lesion in the grey matter of the cord, to the adjacent tracts for painful impulses and for common sensation, probably explains these occurrences.

#### PROGRESSIVE MUSCULAR ATROPHY.

This affection is sometimes called **chronic anterior poliomyelitis**.

An anatomical difference lies at the root of the two types of

this disease. The type which is known as the progressive spinal muscular atrophy, in which spastic symptoms are absent, corresponds to a degeneration of the lower motor segment or neuron. This segment comprises an anterior cornual cell, and its axis cylinder process, which is prolonged through the corresponding anterior root to a muscle. In the other type of disease, spastic symptoms coexist with muscular wasting. This type is known as amyotrophic lateral sclerosis, and is characterised by degeneration of the upper motor segment or neuron, as well as the lower. The upper neuron comprises a pyramidal cell of the motor cortex with its axis cylinder process, which is prolonged through the internal capsule and pyramidal tracts to become functionally connected, through the medium of the grey matter of the anterior cornu, with the lower neuron.

In both types the muscles which waste appear to be successively attacked according to the order in which their cell groups are arranged in the cord. The cervical enlargement which corresponds to the brachial plexus is the most common site of the disease, and it commences with equal frequency at either extremity of this enlargement. Occasionally the upper part of the lumbar enlargement suffers, and rarely other parts. In its later stages the disease may spread upwards to the bulbar nuclei and the signs of glosso-labio-laryngeal palsy be super-added.

The upper part of the cervical enlargement contains the nerve cell groups of the shoulder muscles, and its lower part those corresponding to the small muscles of the hand. Accordingly the deltoid muscle or the muscles of the thenar eminence are usually the first muscles in which the disease is recognised.

When the muscles of the thenar eminence are involved the power of abducting the thumb from the plane of the palm and also that of opposing the thumb to the fingers, are first to become defective, since these movements are almost entirely performed by the short thumb muscles. At the same time the thenar eminence becomes wasted. Ultimately the thumb becomes rotated outwards so that its palmar surface looks

in the same direction as the corresponding surfaces of the fingers.

Atrophy of the first dorsal interosseus muscle or abductor indicis usually follows that of the thenar muscles. This muscle forms the prominence which is seen on the dorsum of the hand between the metacarpals of the thumb and index finger, and so wasting is easily recognised, at the same time power of abducting the index finger from the mid line of the hand is lost. The appearance of hollows between the other metacarpal bones indicates wasting of the rest of the interossei, whilst wasting of the lumbricales which lie between the tendons of the long flexors in the palm, causes those tendons to become very prominent.

Besides abducting and adducting the extended fingers the interossei have the task of flexing the first phalanges and extending the other two. Consequently, should they be paralysed some time before the long flexors and extensors of the wrist have followed suit, the claw hand is produced; this position is the outcome of the loss of action of the interossei, the first phalanges being over-extended, and the other two flexed.

Still following the order of arrangement of motor cells, the disease involves the flexors of the fingers and wrists before the extensors of the same parts.

Of the upper arm muscles the deltoid usually suffers first, often with the supra- and infra-spinati, and the clavicular portion of the pectoralis major.

Muscles which usually escape are the upper part of the trapezius, the sterno-mastoid, and the triceps. The latissimus dorsi and lower half of the pectoralis major are late in showing wasting. The last two muscles are innervated from the sixth and seventh cervical segments; the trapezius by the spinal accessory, which is derived from the second to the sixth cervical segments. The levator anguli scapulæ is innervated from the third and fourth cervical segments.

When the legs waste, the glutei, extensors of the knee, and muscle groups of the front and outer sides of the leg usually suffer. These groups are represented in the lumbar enlargement.

When atrophy involves the extensor muscles at the back of the neck the head falls forwards until the chin rests on the chest.

#### TABES DORSALIS.

The afferent or sensory system of the spinal cord and those portions of the posterior roots which intervene between the posterior root ganglia and the point of entry of the roots into the cord, are the parts which are principally involved in tabes dorsalis. The lumbar region and the cauda equina are usually the sites of the most advanced disease. The peripheral nerves of the body may also suffer in varying degrees, and certain of the cranial nerves, both motor and sensory, may also be invaded. Consideration of the course taken by the fibres of a posterior spinal root on entering the cord shows that the tabetic lesions in the posterior columns correspond to the distribution of the posterior root fibres. (Fig. 19.)

From a posterior root three sets of intraspinal fibres may be derived. The first set consists of fine fibres, which pass directly to form Lissauer's tract at the tip of the posterior cornu of the grey matter, and end in the substantia gelatinosa of that cornu. The second set of fibres form the cornu-radicular zone. Some of these pass directly forwards to become connected with the motor cells of the anterior cornu, the rest do not terminate in the segment at which they entered the cord, but pass upwards for a short distance, being gradually displaced forwards by the corresponding fibres of the roots which enter above them. They are thus pushed into the anterior and middle parts of the postero-external column, or column of Burdach, and ultimately come into connexion with the cells of Clarke's column, which in turn give origin to the fibres of the direct cerebellar tract. The third set consists of long fibres, which soon pass from the postero-external column into the postero-internal column of Goll, in which they are continued up to the nucleus of the funiculus gracilis in the medulla. The long fibres which ascend in Goll's column are principally derived from the posterior roots of the

THE SPINAL CORD.

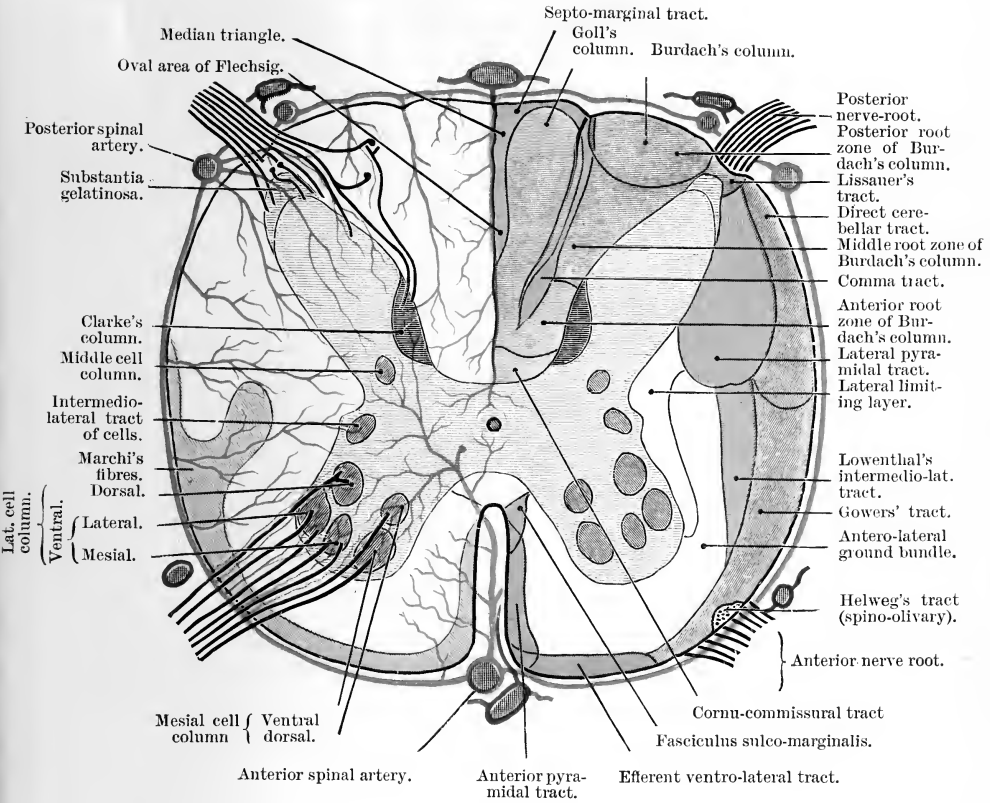


FIG. 19.—Diagram showing the tracts, cell columns, and vascular supply of the spinal cord. (From Morris's Anatomy after Arthur Robinson.)



lumbo-sacral region. It is doubtful if any fibres are contributed to this column by the posterior roots in the thoracic region from the third to the twelfth inclusive. The corresponding ascending fibres from the posterior roots of the cervical enlargement are said to continue their ascent in Burdach's column, reaching the nucleus of that column in the medulla.

All these sets of fibres may degenerate in tabes, and in addition the process in some cases extends to the cells of Clarke's column, and to the direct cerebellar tracts derived from these.

Attempts have been made to correlate the different sensory symptoms of the disease with the different fibre groups. Thus the loss of muscle tone and of the tendon reflexes is referred to the interruption of those fibres which pass directly to the anterior cornual motor cells, and so complete the local reflex arc. It is also conjectured that the long ascending fibres of the postero-internal columns subserve the muscle sense, that the fine fibres which enter the grey matter through Lissauer's tract are fibres of common sensation, and that the fibres which establish a connexion with the cells of Clarke's column convey impulses to the cerebellum, and so form part of the mechanism of equilibration.

There are certain tracts of fibres in the posterior columns of the cord, which originate from the cells of the grey matter of the posterior cornua, and are hence termed endogenous. These tracts are commissural in function, and do not consist of fibres derived from the posterior roots. They either escape entirely in tabes or are affected very late in the disease. The tracts in question are:—(1), the comma tract which lies in the middle third of the postero-external column in the dorsal region; (2), the cornu-commissural zone, which is immediately adjacent to the posterior commissure in the lumbar and sacral regions; (3), the oval area of Flechsig, which abuts on the posterior fissure in the lumbar region, and is continuous in the sacral region with (4) the sacral triangle of Gombault and Phillippe; (5), the septo-marginal tract, which is continuous with the oval area of Flechsig, and can be traced upwards as high as the eleventh dorsal segment.

In forming an estimate of the amount of wasting of the posterior roots outside the cord, it is well to bear in mind that they are normally twice or three times the size of the anterior. The degeneration can be traced distally as far as the posterior root ganglion, and there ceases. The changes in the ganglion are often insignificant, whilst the changes in the sensory nerves beyond the ganglion are variable, and tend to be most marked at the periphery. In the process of development the cells of the posterior ganglia send out processes which bifurcate, one branch growing into the cord as a posterior root fibre, whilst the other becomes connected with skin, muscle, joint, or tendon as part of the peripheral sensory nerve. A lesion of the sensory nerve trunks, or the posterior roots of the spinal cord, may cut off all impulses from the structures mentioned. Impulses from the joints, tendons, and muscles are mainly concerned in producing the sense of position, and deprivation of these impulses may induce ataxy with muscular atony and loss of sense of position, without the necessary existence of any skin anæsthesia at all.

The ataxy which in some instances accompanies the peripheral neuritis of alcoholism, influenza, and diphtheria, is probably due to lesions of the peripheral sensory nerves.

When cutaneous anæsthesia is present in tabes it is of the segmental type. Often there is an anæsthetic zone corresponding to the fourth and fifth dorsal segments. Extending higher it may involve the dorsal segmental areas corresponding to the inner side of the arm, but is rarely found to extend on the thorax above the third rib, for the skin above this is supplied by descending cervical nerves which are rarely implicated. Segmental anæsthesia, usually restricted to analgesia, is often found in the areas of the lumbo-sacral roots, particularly the first sacral which supplies the sole of the foot, and the fifth lumbar which supplies the peroneal region of the leg. The dissociation of sensation which occurs in various forms and various degrees, is attributed to unequal affection of the various fibres which go to make up the compound nerves, and intra-medullary



projections of the nerve roots. Skin reflexes vary in their activity inversely with the degree of local cutaneous anæsthesia.

A convenient means of testing the loss of tone in the muscles is found by taking advantage of the fact that the hamstrings, when possessing their normal tone are not of sufficient length to allow the leg to be fully extended on the thigh at the same time that the thigh is fully flexed on the abdomen. This position can often, however, be assumed in tabes, and certain cases of peripheral neuritis in which muscle tone is lost.

The perforating ulcer of the foot usually originates at the site of a corn, and is obviously due to pressure and suppuration in anæsthetic tissues.

The trophic joint and bone lesions of tabes are usually attributed to the lesion of special trophic fibres, which are supposed to follow the paths taken by the fibres for pain and temperature rather than those of the other afferent tracts.

The Argyll Robertson pupil has been discussed in the section on ophthalmoplegia.

The intraocular portion of the optic nerve is accessible to direct examination, and the occurrence of primary optic atrophy may afford valuable evidence in doubtful cases of tabes. Of the other cranial nerves we can only judge by function. The ascending root of the fifth nerve, which corresponds to the posterior roots of the cord, is sometimes involved, and so bilateral facial neuralgia exceptionally forms a symptom of tabes, corresponding to the lightning pains and to the sciatica of the lumbo-sacral form of the disease.

#### SYRINGOMYELIA.

Syringomyelia is characterised by the presence of a dilated central canal or of other cavities within the spinal cord, together with more or less proliferation of the embryonic tissue in their neighbourhood. The lesions are practically confined to the posterior half of the cord, and usually limited to the posterior columns, which are formed last during the process of development. The disease is a congenital defect, the primitive canal

for some reason remaining unusually large, and embryonic tissue persisting around it. In some cases a central canal may be recognised, distinct from the cavities in the nerve tissue. To explain the separation of the two it is assumed that the posterior portion of the primitive canal becomes shut off from the anterior part, and persists in the form of a cavity in the region of the posterior septum of the cord. In other instances cavities lie in the posterior horns of the grey matter, and may be due to the disintegration of the embryonic tissue which forms the basis of these.

Syringomyelia usually occurs in the cervico-dorsal region, and the disease accordingly manifests itself chiefly in the arms. The symptoms are referable to disease of the grey matter, and consist of local muscular atrophy with dissociation of sensation. The sense of touch is usually retained whilst painful and thermal sensations may be absent. Trophic lesions are also met with.

TABLE OF LEVELS OF ORIGIN AND EXIT OF SPINAL NERVES.

Roots.	Level of Origin from Cord.	Point of Exit from Canal.
C. 1	... Just above arch of atlas ... ..	Between atlas and occiput.
2	... Ranges from just above to just below posterior tubercle of atlas ... ..	Above axis.
3	... At or just above spine of axis ... ..	Above 3rd cervical vert.
4	... Ranges from spine of axis to spine of 3rd cervical vertebra ... ..	Above 4th cervical vert.
5	... Ranges from lower border of spine of axis to lower border of spine of 4th cervical v.	Above 5th cervical vert.
6	... Ranges between lower borders of spines of 3rd and 5th cervical vertebra ... ..	Above 6th cervical vert.
7	... Ranges between top of 4th and bottom of 6th cervical spines... ..	Above 7th cervical vert.
8	... Ranges between upper borders of 5th to 7th cervical spines ... ..	Above 1st dorsal vert.
D. 1	... Ranges between space above 6th and space below 7th spines ... ..	Above 2nd dorsal vert.
2	... Ranges between lower borders of 6th cervical and 1st dorsal spines ... ..	Above 3rd dorsal vert.
3	... Ranges between upper edge of 7th cervical to lower border of 2nd dorsal spine ...	Above 4th dorsal vert.
4	... Ranges from top of 1st to top of 3rd dorsal spine ... ..	Above 5th dorsal vert.
5	... Ranges from top of 2nd to top of 4th dorsal spine ... ..	Above 6th dorsal vert.
6	... Ranges from lower edge of 2nd dorsal spine to upper of 5th ... ..	Above 7th dorsal vert.
7	... Ranges from top of 4th to bottom of 5th dorsal spine... ..	Above 8th dorsal vert.



SEGMENTAL CUTANEOUS AREAS.

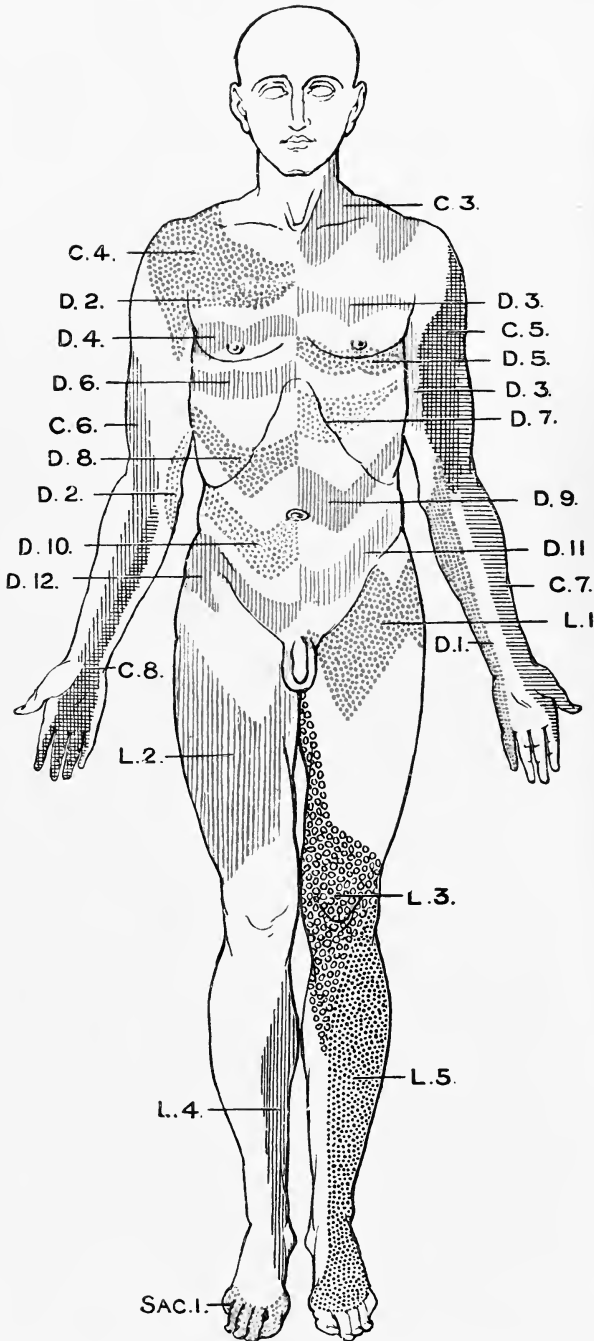


FIG. 20.—This figure shows segmental cutaneous areas. The several dorsal, lumbar, and sacral areas are indicated each by the initial letter followed by a number. (After Head.)

SEGMENTAL CUTANEOUS AREAS.

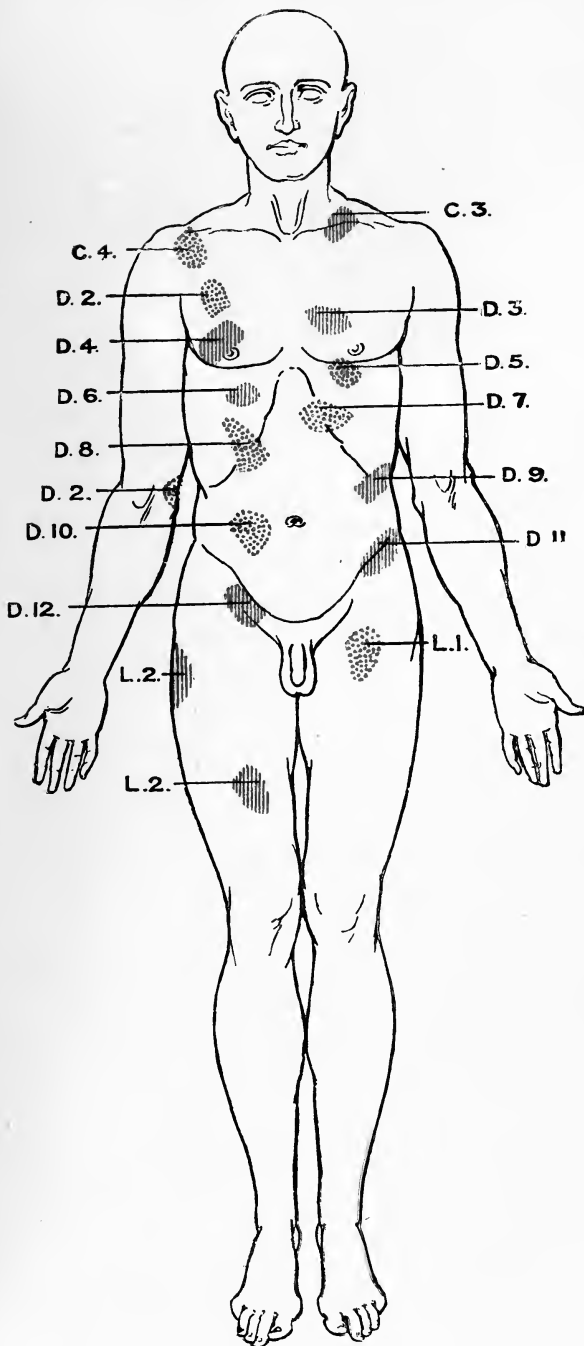


FIG. 21.—This figure shows the “maximum spots” (seats of most marked tenderness and pain) of the different areas. (After Head.)

[To face figure 20.





SEGMENTAL CUTANEOUS AREAS.

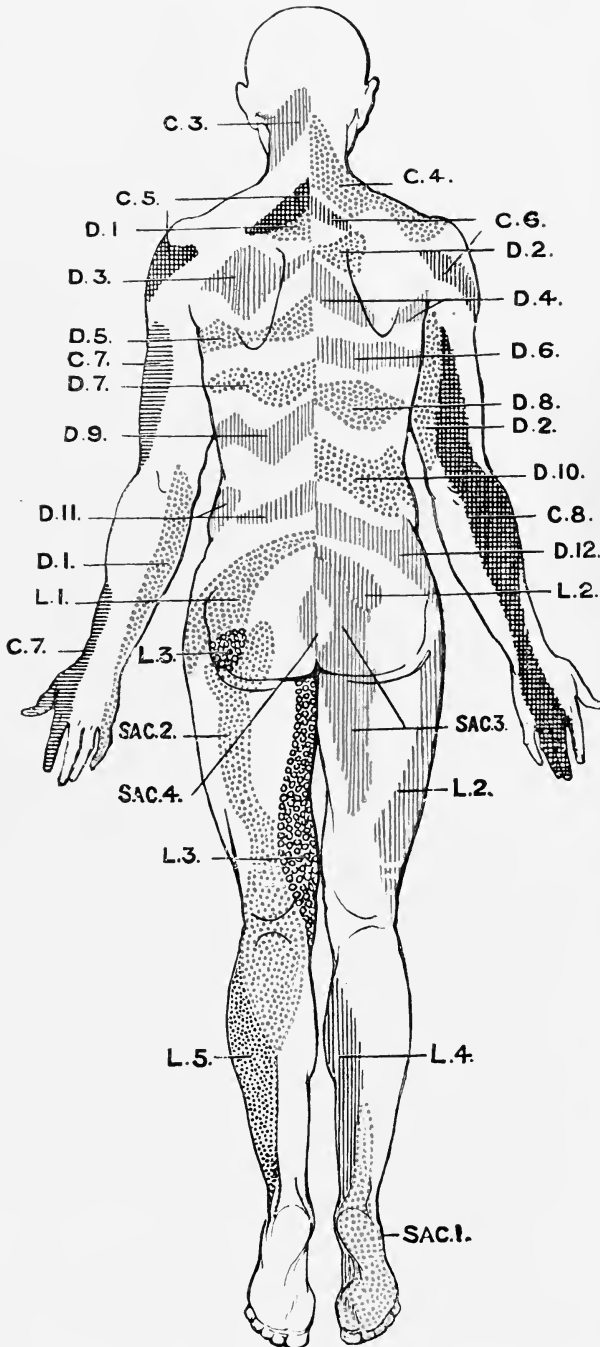


FIG. 22.—This figure shows segmental cutaneous areas. The several dorsal, lumbar, and sacral areas are indicated each by the initial letter followed by a number. (After Head.)

[To follow figure 21.]



SEGMENTAL CUTANEOUS AREAS.

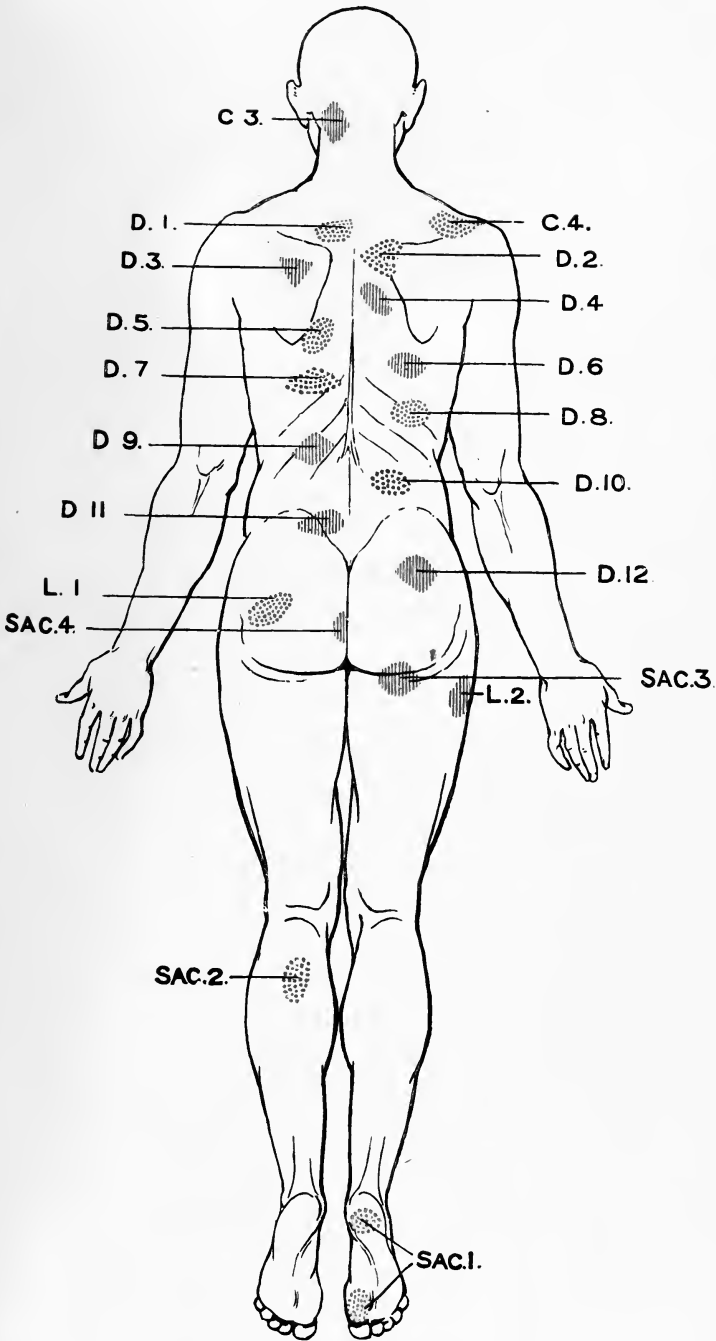


FIG. 23.—This figure shows the “maximum spots” (seats of most marked tenderness and pain) of the different areas. (After Head.)

[To face figure 22.



TABLE OF LEVELS OF ORIGIN AND EXIT OF SPINAL NERVES—*continued.*

Roots.	Level of Origin from Cord.	Point of Exit from Canal.
D. 8 ...	Ranges from top of 5th to top of 6th dorsal spine ... ..	Above 9th dorsal vert.
9 ...	Ranges from midway between 5th and 6th to top of 7th dorsal spine... ..	Above 10th dorsal vert.
10 ...	Ranges from midway between 6th and 7th to middle of 8th dorsal spine ... ..	Above 11th dorsal vert.
11 ...	Ranges from top of 7th to top of 9th dorsal spine ... ..	Above 12th dorsal vert.
12 ...	Ranges between top of 8th and bottom of 9th dorsal spine ... ..	Above 1st lumbar vert.
L. 1 ...	Ranges from top of 9th to bottom of 10th dorsal spine... ..	Above 2nd lumbar vert.
2 ...	Ranges between 9th and 11th dorsal spines ... ..	Above 3rd lumbar vert.
3 ...	Ranges between top of 10th and bottom of 11th dorsal spine ... ..	Above 4th lumbar vert.
4 ...	Ranges between bottom of 10th and top of 12th spine ... ..	Above 5th lumbar vert.
5 ...	Ranges between top of 11th and middle of 12th dorsal spine ... ..	Above 1st sacral vert.
S. 1 ...	Ranges between lower border of 11th dorsal spine and top of 1st lumbar spine ... ..	Above 2nd sacral vert.
2 ...	Usually between 12th dorsal and 1st lumbar spines ... ..	Above 3rd sacral vert.
3 ...	Usually between 12th dorsal and 1st lumbar spines ... ..	Above 4th sacral vert.
4 ...	Usually between 12th dorsal and 1st lumbar spines ... ..	Above 5th sacral vert.
5 ...	At lower border of 1st lumbar spine ... ..	Above coccyx.
Coccygeal...	Between lower border of 1st and upper border of 2nd lumbar spines ... ..	

*Summary.*

- The eight cervical nerves arise from the cord between the occiput and the sixth cervical spine.
- The upper six dorsal nerves between the sixth cervical and the fourth dorsal spines.
- The lower six dorsal nerves between the fourth and ninth dorsal spines.
- The five lumbar nerves arise between the ninth and twelfth dorsal spines.
- The five sacral nerves opposite the twelfth dorsal and first lumbar spines.

COMPRESSION OF THE SPINAL CORD.

The spinal cord, being suspended in the spinal canal, is liable to compression, bruising, or laceration in fracture-dislocations of the vertebral column. Moreover, the bony unyielding character of the neural canal renders compression almost inevitable when inflammatory or malignant disease extends into the cavity from the adjacent vertebræ, or arises in the spinal membranes. The dural sheath of the cord is separated from the surrounding bones

by a layer of fatty tissue and a considerable venous plexus. The former tissue may give rise to lipomatous growths or be the seat of hydatid disease, or diffuse suppuration, and the veins may be torn in injuries to the spinal column leading to considerable extravasation of blood. The thickening of the dura mater which is known as pachymeningitis may produce pressure on the cord; hydatids and new growths may be found inside the dura mater and a sudden effusion of fluid into the membranes, the result of hæmorrhage or of rupture of an abscess will also produce pressure symptoms.

The intervertebral foramina afford channels by which new growths, abscesses or even hydatids may invade the spinal canal from without. The close relation of the aorta to the bodies of the vertebræ explains the ease with which aneurysms of the descending thoracic and abdominal portions of the vessel erode the walls of the canal and ultimately exert pressure on the structures contained in it.

The retroperitoneal glands are favourably placed for invasion of the spinal column and spinal canal when the site of malignant disease, whilst the thoracic part of the spine is often the seat of new growth secondary to carcinoma of the breast.

Tumours within the dura, being confined by that membrane, more easily produce compression than do tumours outside it.

Since the spinal nerve roots cross the subdural space and traverse the dura to escape through the intervertebral foramina it is not surprising that pain should be referred to the sensory roots at the level of compression (Figs. 20—23, p. 226). This is most marked in connexion with new growths, especially those which arise in the spinal membranes, and the pain may persist after the skin supplied by the particular nerve roots has become anæsthetic. Root pain is not such a marked feature in compression by aneurysm or caries. Since the anterior spinal roots are motor in function, pressure on these may produce muscular spasm of the muscles which they supply, but more commonly the muscles simply waste.

The wasting is easily recognised when it occurs in the muscles

of the extremities, but recognition is not so easy when the intercostals or muscles of the abdomen are involved. It is possible, however, to tell if there is any weakness of the upper or lower parts of the recti by observing the movement of the umbilicus. This normally does not vary its position on the patient sitting up, but if the part of the muscle below the umbilicus is paralysed the unopposed action of the upper part will draw up the umbilicus. If the part of the rectus above the umbilicus is affected, the umbilicus will be drawn downwards instead of upwards. The rectus is supplied by the fifth to the twelfth dorsal nerves. As the umbilicus is in the field of the ninth dorsal root, a lesion paralysing that part of the rectus which lies below it belongs to the tenth, eleventh or twelfth dorsal segments.

In addition to the radiating pains produced by compression of the spinal roots, the local pressure on the dura mater is responsible for a deep-seated local pain. This is probably explained by the fact that the dura is supplied with nerve filaments, and is known to be extremely sensitive.

Besides root symptoms, compression produces symptoms referable to the cord itself. The points at which there is the least spare space in the theca are said to lie in the mid-cervical region and at the two extremities of the dorsal region, and these are the situations in which most instances of compression by tumour occur. The pressure exerted on the cord is at first more or less localised, and so unilateral symptoms may be produced, becoming bilateral as the pressure increases. The compression may produce local softening, either from occlusion of vessels or the onset of an inflammatory condition.

The *compression symptoms* usually, but not invariably, occur in the following order:—Pain, motor paralysis, sensory paralysis. The pain, which is distinct from the root pains and localised pain already mentioned, is often referred to points at some distance below the lesion in the spinal cord, not infrequently to joints. Also when anæsthesia develops the sensation is first affected in the lower parts of the body. There are two anatomical facts which may have some bearing on this. First, the fibres

which ascend from the lowest parts of the cord are longest and travel further from their trophic centres in the posterior root ganglion, hence they may be more susceptible to pressure. Secondly, as the fibres of the successive sensory roots enter the cord, those of them which ascend in the posterior columns tend to displace inwards the fibres which enter immediately below them, so that the fibres which enter lowest finally come to lie near the surface of the postero-median column close to the median septum, whilst the higher fibres approach nearer and nearer the central part of the cord. Consequently fibres from the lowest roots in the posterior columns at all events, are most exposed to concentric compression. But it is stated that the march of anæsthesia may be the same in eccentric pressure, such as occurs for instance with hæmorrhage into the grey matter.

The motor paralysis below the level of the lesion is of the spastic type, and accompanied by interference with the action of the bladder and rectum. Pressure on the pyramidal tracts accounts for the motor symptoms. Curiously enough the direction of invasion of paralysis is the reverse of the anæsthesia, the highest muscles suffering first. No arrangement of fibres in the pyramidal tracts is at present known which would account for this method of invasion. The conversion of the spastic into a flaccid condition in the terminal stages of the disease is held to indicate the establishment of a total transverse lesion of the cord, possibly induced by softening or myelitis.

There are certain motor symptoms in compression paraplegia which are special to definite regions of the cord. These comprise alterations in respiration, alterations in the pupils, alterations in the heart's action, vaso-motor and secretory phenomena.

Since the main motor supply of the diaphragm is derived from the fourth cervical nerve, fibres of which together with some from the third and fifth nerves make up the phrenic trunk, a lesion in this region is peculiarly dangerous. It not only cuts off the impulses descending to the intercostals from the respiratory centre, but also gives rise to paralysis of the diaphragm so that respiration ceases.

Pupil dilator fibres, arising in the neighbourhood of the third cranial nerve nucleus, are known to pass downwards in the lateral columns of the cord to emerge by the first and second dorsal roots. Thence they pass to the inferior cervical ganglion, and so by the cervical sympathetic chain to the eye. Unilateral pressure on the cervical cord may affect the fibres descending in it and give rise to unilateral myosis.

The cardio-accelerator fibres in the cord pursue a similar course to the pupil dilator fibres but emerge lower in the fourth to the ninth dorsal roots. Compression of the cord may irritate or paralyse these fibres, and acceleration or slowing of the heart rate will result.

Vaso-dilatation may occur in acute compression. The vaso-dilator fibres for the whole body leave the spinal cord in the middle of its extent, probably reaching from about the second dorsal to the second lumbar nerves.

The vaso-constrictor nerves have a much more extensive outflow.

Since secretory fibres for the sweat glands also leave the cord these are liable to be paralysed where nerve roots are compressed, and so the injection of pilocarpine has been used to determine the level of compression, the sweat ceasing at this line.

The upper level at which sensation is in any way altered is usually taken to indicate the level of the compressing lesion. The level thus determined often proves too low. Owing to the oblique course of the nerve roots in the spinal canal before they emerge, the seat of compression is in most cases higher than the upper limit of anæsthesia, the difference corresponding to the vertical length of the nerves in the canal. Even when this is allowed for by the use of suitable tables, the level of the anæsthesia may prove to be considerably below the level of the lesion, and this is due to the fact that sensory root symptoms may be absent at the level of the lesion and that all the sensory fibres ascending in the cord from the parts below have not suffered equally, but that the longest tracts have first ceased to conduct.

## THE SPINAL NERVES.

## INJURIES AND DISEASES OF NERVE PLEXUSES.

The **brachial plexus** is responsible for the whole nerve supply of the arm. If a lesion involves all parts of the plexus the arm will be completely paralysed. The damage is usually incomplete and three types of paralysis may be recognised, corresponding to the upper, middle and lower parts of the plexus respectively.

The *upper arm type* is variously known as Duchenne's obstetrical paralysis of the infant, Erb's paralysis, anæsthetic paralysis and climber's paralysis. The muscles paralysed are the deltoid, supraspinatus, infraspinatus, teres minor, biceps, brachialis anticus and supinator longus. The position assumed by the arm is characteristic. It hangs powerless by the side; the humerus is rotated inwards by its unopposed internal rotators; the flexors of the elbow being powerless the forearm is extended; the supinating action of the biceps is absent, so the hand is fully pronated, and the palm directed backwards and outwards. The muscles which are paralysed derive their supply from the fifth and sixth cervical roots. The same muscular group may be put in action in health by faradising Erb's motor point which lies just behind the posterior border of the sterno-mastoid in front of the transverse process of the sixth cervical vertebra. The lesion is due to the shearing action of the clavicle, which under certain circumstances compresses the plexus against the first rib. For this to occur the arm must be abducted and raised vertically above the shoulder, a movement which causes the clavicle to travel directly backwards. The muscular mechanism of the shoulder is not enough to produce this, but force must be applied to the abducted and raised arm. Hence the lesion may occur at birth when the arm is above the head, as the result of forcible artificial respiration, when the weight of the body hangs on the arm in hand over hand climbing, or when a child is lifted by one arm. By some authors it is held that a similar lesion may be produced, owing to traction on the upper cords of



the plexus, when the shoulder is forcibly depressed, and the neck laterally flexed towards the opposite shoulder.

The *intermediate type* of paralysis corresponds to a lesion of the middle part of the plexus. It is usually associated with the upper arm type, but may be observed as a residual palsy after the upper part of the plexus has recovered. The muscles paralysed are the triceps, and the extensors of the wrist and fingers. The attitude is characteristic, the elbow being flexed, the wrist dropped, the fingers somewhat bent, and the hand lying midway between pronation and supination. The seventh cervical root is the one chiefly involved.

The *distal type* of paralysis is known as Klumpke's. The lesion involves the lower fibres of the plexus, corresponding to the eighth cervical and first dorsal nerves. The small muscles of the hand and the flexors of the fingers are paralysed. This type of paralysis is accompanied by myosis, narrowing of the palpebral fissure and retraction of the eyeball, phenomena referred to implication of the oculo-pupillary fibres which pass into the cervical sympathetic from the upper dorsal nerves.

The intermediate and distal types of paralysis are uncommon, the corresponding nerves being protected by the fact that the inner part of the clavicle has a forward convexity, and so does not so readily compress the lower part of the plexus. Or, if the lesion be due to stretching and laceration, the fact that the lower fibres of the plexus are not rendered taut so easily as the upper will account for their comparative immunity.

The brachial plexus may be involved by other lesions such as tumours, disease of the cervical spine, cervical meningitis, local pressure or wounds in the lower part of the posterior triangle of the neck, and dislocations of the shoulder. It is sometimes the seat of neuritis.

Anæsthesia is not so readily produced as motor paralysis, and when present, if due to a lesion of the nerve roots, will have the corresponding segmental distribution. (Figs. 20 and 22, p. 226.)

The **lumbar** and **sacral plexuses** may be involved by local lesions such as malignant growths, psoas abscesses or spinal

disease. A true neuritis may also occur. Sometimes neuritis is a manifestation of a general toxæmia of pregnancy, sometimes it is due to the local extension of pelvic inflammation, for the plexus lies in the planes of pelvic connective tissue.

The most interesting lesion from an anatomical point of view is a sudden paralysis which may come on during childbirth. The paralysis is attributed to pressure by the foetal head on its passage through the pelvis. The muscles supplied by the peroneal nerve are usually affected. The peroneal nerve is derived from the dorsal divisions of the lumbo-sacral cord, and the first and second sacral nerves. The dorsal constituents of the lumbo-sacral cord lie next to the bone as the cord passes over the true pelvic brim and so are exposed to pressure, whereas the lower roots of the sacral plexus lie on the pyriformis muscle, which affords some protection. The gluteal nerves are sometimes involved at the same time as the peroneal; these nerves are also derived from the dorsal divisions of the lumbo-sacral cord and first and second sacral nerves.

#### INJURIES TO THE PERIPHERAL NERVES OF THE LIMBS.

Division of the peripheral nerves of the extremities is a common lesion, especially in the upper limb. Hence a thorough knowledge of the distribution of these nervous trunks is of great practical importance. Injury may be the result of accident or operation.

**The Upper Extremity.**—The chief nerves that may be damaged are the ulnar, the median and the musculo-spiral.

The **ulnar nerve** arising from the inner cord of the brachial plexus runs along the inner side of the arm, and enters the forearm in the space between the internal epicondyle of the humerus and the olecranon process of the ulna, in which position it is liable to injury in fracture of the condyle, in excision of the elbow joint and in punctured wounds. Passing between the two heads of the flexor carpi ulnaris, it pursues a course down the ulnar side of the forearm under cover of this muscle, and at the wrist, lying on the radial side of the pisiform bone, it enters the palm

of the hand superficial to the anterior annular ligament, quickly to divide into its superficial and deep divisions:

In its course it is distributed as follows:—

*Muscular. In forearm:—*

To flexor carpi ulnaris.

To flexor profundus digitorum, ulnar half.

As a consequence of the paralysis of these muscles, adduction of the hand towards the ulnar side and full flexion of the last phalanx of the ring and little fingers is lost.

*In hand:—*

By main trunk to palmaris brevis.

By deep branch to abductor minimi digiti, flexor brevis minimi digiti, flexor ossis metacarpi minimi digiti, all seven interossei, two ulnar lumbricales, adductor transversus pollicis, adductor obliquus pollicis, flexor brevis pollicis, deep part.

The deep branch runs between the flexor brevis and abductor minimi digiti muscles, and often grooves the ulnar side of the base of the process of the unciform bone, and at this spot it may be divided by a punctured wound. It is easily found between the pisiform bone and the unciform process.

By paralysis of these muscles the following movements cannot any longer be carried out:—abduction of all the digits except the thumb, adduction of the thumb, index, ring and little fingers, flexion of the first phalanx of all the fingers, and perfect extension of the second and third phalanges. It will thus be seen that a very characteristic form of paralysis is induced, which can readily be demonstrated by asking the sufferer to attempt to move the index and little fingers away from the next to which they are placed.

In its earlier stages the typical claw-hand (*main en griffe*) of ulnar nerve paralysis is seen in the over-extension (dorsi-flexion) of the first phalanx of the ring and little fingers, with partial flexion of the second and third. This position of these two digits is probably due to the unopposed action of the extensors and of the flexor sublimis tendons owing to the paralysis of the two ulnar lumbricales. If it were dependent upon

the loss of action of the interossei alone, then the index and middle fingers should also be affected, but in the early stages at least this is not the case. Later all four fingers may assume the typical position, but the ring and little fingers always exhibit it in a more pronounced manner.

When degeneration of the muscles, cut off from their trophic supply, occurs, great diminution of the hypothenar eminence and some diminution in that of the thenar eminence follows. Intermetacarpal hollows also appear on the dorsum of the hand, particularly noticeable in the first space by the disappearance of the abductor indicis or first dorsal interosseus.

*Cutaneous. In forearm:—*

The palmar cutaneous branch, given off in the lower third of forearm, pierces the deep fascia, passes into the palm superficial to the anterior annular ligament, and is distributed to the skin of the hypothenar eminence and ulnar half of palm.

The dorsal cutaneous branch often given off as high as in the middle third of the forearm, is directed obliquely backwards and downwards deep to the tendon of the flexor carpi ulnaris, and becomes cutaneous at the back of the ulnar side of the forearm in its lower fourth. On the dorsum of the hand this branch supplies filaments to the skin of the ulnar side, and terminates by sending branches to the dorsal aspect of the ulnar side of the little finger and the contiguous sides of the ring and little fingers, which supply the skin as low down as the middle of the second phalanx.

*In hand:—*

The superficial branch is purely cutaneous, and like the dorsal branch supplies the skin of the palmar aspect of the ulnar side of the little finger and the contiguous sides of the ring and little fingers to their distal extremity, afterwards turning round on to the dorsal aspect for the supply of the cutaneous structures over the lower half of the second and the whole of the terminal phalanges.

It will thus be seen that when the ulnar nerve is entirely paralysed there is loss of sensation over the ulnar half of palm, the hypothenar eminence, the ulnar half of the back of the hand, and the whole of the palmar and dorsal aspects of the little finger and the ulnar half of the ring finger.

Later in the course of ulnar nerve paralysis trophic changes occur in the same area.

It is important to recollect that if the ulnar nerve is cut just above the wrist—perhaps the most usual spot—the dorsal cutaneous branch may escape any injury, and the patient will therefore retain sensation on the back of the ring and little fingers as low as the middle of the second phalanx, but below this the skin will be anæsthetic.

*Articular.*

*In arm*—To elbow joint.

*In foreman*—To wrist and carpal joints.

*In hand*—To ulnar two carpo-metacarpal joints.

To metacarpo-phalangeal and interphalangeal joints of ring and little fingers.

In paralysis of the nerve, trophic changes may occur in the small distal joints leading to a condition akin to ordinary osteo-arthritis.

The **median nerve**, arising by two heads, one from the outer and the other from the inner cord of the brachial plexus, runs a median course down the arm, crossing the brachial artery at its middle, as a rule superficially but sometimes deeply, from without inwards.

It gives off no branches in the arm.

At the bend of the elbow it lies under cover of the bicipital fascia and internal to the brachial artery. It is not often wounded in the arm or at the bend of the elbow. The nerve enters the forearm between the two heads of the pronator radii teres, and it is possible for it to be compressed by this muscle during violent exercise such as tennis playing, but being deeply placed it is little exposed to actual division until it reaches the lower fourth of the forearm. It is here much more superficial,

and placed still in a median position exactly posterior to the tendon of the palmaris longus muscle, if this is present.

Just above the anterior annular ligament is perhaps the most usual spot for the nerve to be accidentally divided. An incised wound caused by passing the hand through a pane of glass is the common form of injury to cause the division. At the same time the surrounding flexor tendons may be severed. It is very necessary to be able to distinguish between the divided ends of nerve and tendon, for accurate suturing in these cases is highly essential. The following differences may be considered :—

	<i>Nerve.</i>	<i>Tendon.</i>
<i>Colour ...</i>	... Greyish pink. Dull. ...	... Yellowish white. Shining.
<i>Cut surface ...</i>	... Round. Tends to be frayed. ...	... Transversely oval. Clean cut.
<i>Retraction ...</i>	... Proximal end not greatly retracted.	... Proximal end markedly retracted.
<i>Action ...</i>	... Manipulation of distal end may excite action of short muscles of thumb.	... Pull on distal end will cause flexion of a digit.

The median nerve gives off the anterior interosseous branch soon after it enters the forearm.

The main trunk supplies the pronator radii teres (the branch to this muscle may arise just above the flexure of the elbow) the flexor carpi radialis, the palmaris longus and the flexor sublimis digitorum in the forearm.

The anterior interosseous branch supplies the flexor longus pollicis, the radial half of the flexor profundus digitorum, and the pronator quadratus: all of these muscles will be paralysed if the nerve is cut just above the bend of the elbow, and the loss of power will be easily recognised by the inability to flex the terminal phalanx of the thumb, and the second and third phalanges of the index and middle fingers, and to pronate the hand.

In the lower third of the forearm the palmar cutaneous branch is given off.

The median nerve enters the palm of the hand by passing deep to the anterior annular ligament, and immediately gives off a motor branch to the chief muscles of the thenar eminence, viz.,

the abductor pollicis, the flexor ossis metacarpi pollicis and the flexor brevis pollicis (superficial head).

The rest of the nerve is supplied to the two radial lumbricales, and to the skin on the palmar aspect of the thumb, index, middle and the radial half of the ring finger. The terminal filaments of these cutaneous branches also pass round on to the dorsal aspect of the thumb and supply the bed of the nail, and to the dorsal aspect of the index, middle and the radial half of the ring finger, to supply sensation as high as the middle of the second phalanx, about the level beyond which cutaneous hairs cease to be found.

It will thus be seen that in division of the median nerve close above the wrist there will be loss of power of abducting the thumb and of directly flexing its metacarpal bone and proximal phalanx; also loss of sensation on the whole of the palmar aspect of the thumb, index, middle, and radial half of the ring finger, and over the dorsal aspect of the same, in the case of the thumb extending upwards as far as the middle of the terminal phalanx, and in the case of the other digits as high as the middle of the second phalanges.

The **musculo-spiral nerve** is derived from the posterior cord of the brachial plexus and lies at first behind the third portion of the axillary and the commencement of the brachial artery. It then winds round the inner side of the humerus to reach the musculo-spiral groove on the posterior aspect of the bone, between the external and internal heads of the triceps muscle, to appear on the outer side of the arm above the elbow between the brachio-radialis and the brachialis anticus.

During this course it supplies the following muscles:—The three heads of the triceps, the anconeus, brachio-radialis, extensor carpi radialis longior, and a small portion of the brachialis anticus, in addition to giving one internal and two external cutaneous branches. Finally, it divides into its two terminal branches, the radial and the posterior interosseous.

The *radial* nerve is purely cutaneous and is distributed to the dorsal aspect of the thumb, as low down as the bed of the nail,

the dorsal aspect of the index, middle and radial side of ring fingers as low down as the middle of the second phalanx. One of these digital branches can be easily palpated as it crosses the tendon of the extensor longus pollicis where that structure forms the ulnar boundary of the *tabatière*.

The *posterior interosseous* branch of the musculo-spiral reaches the back of the forearm between the two planes of the supinator brevis muscle, supplying this muscle in its course, and winding round the radial side of the neck of the radius. It is then distributed to all the extensor muscles, namely, superficially to the extensor carpi radialis brevior, extensor communis digitorum, extensor minimi digiti, extensor carpi ulnaris; and deeply to the extensor ossis metacarpi pollicis, extensor brevis pollicis, extensor longus pollicis, and the extensor indicis.

From its deep position the musculo-spiral nerve is not greatly exposed to external injury, but is liable whilst lying in the musculo-spiral groove to be pressed upon by callus formed around a fracture of the middle of the shaft of the humerus. Also before it reaches the posterior aspect of the limb it may be paralysed by the pressure of the handle of a crutch, or by the pressure induced by the arm hanging over some sharp edge, such as the back of a chair, or even the edge of an operating table. The nerve is sometimes paralysed by a sudden and violent contraction of the triceps, being compressed as it lies in the musculo-spiral groove.

The result of division will be a permanent paralysis unless the continuity of the nerve is restored, whilst the result of pressure may be only a temporary paralysis. The paralysis is evidenced by wrist-drop—that is, loss of power to extend the carpus, hand, and digits—associated in the majority of cases by anæsthesia, partial, or complete, over the distribution of the radial nerve.

In the case of musculo-spiral paralysis from lead poisoning, the brachio-radialis (supinator longus) muscle usually escapes.

The **musculo-cutaneous nerve** is derived from the outer cord of the brachial plexus, and is perhaps the least frequently damaged of any of the nerves of the upper extremity. It



passes through the coraco-brachialis, supplying that muscle, and then comes to lie beneath the biceps and upon the brachialis anticus, both of which it innervates. Above the elbow-joint it is not so very far internal to the musculo-spiral, and when exposed may be mistaken for the latter nerve.

The **circumflex nerve** is derived from the posterior cord of the brachial plexus, and winds round the back of the surgical neck of the humerus, supplying the deltoid and teres minor muscles, together with the shoulder-joint and some of the skin covering it.

The nerve is liable to injury in fracture of the surgical neck, separation of the upper epiphysis, and dislocation of the head of the humerus.

**The Lower Extremity.**—Injury to the nerves of the lower extremity is comparatively rare, but disease affecting the function of these nerves is not infrequently seen.

The **great sciatic nerve** emerges from the pelvis through the great sacro-sciatic foramen below the pyriformis muscle, at a point which may be roughly indicated upon a line from the posterior superior spine of the ilium to the most prominent part of the tuberosity of the ischium, at the junction of the lower third with its upper two-thirds. Here it is deeply placed under the gluteus maximus muscle, and can be considerably stretched in complete flexion at the hip-joint especially when the knee is kept extended. A little lower down it lies half-way between the great trochanter and the tuber ischii, and deep pressure here will give rise to severe pain when the nerve is inflamed. Proceeding down the back of the thigh, it next lies between the hamstrings and the vastus externus, dividing about the middle into the peroneal or external popliteal, and the internal popliteal nerves.

The sciatic nerve is accompanied by an artery which may be of great value in the formation of a collateral circulation after ligature for femoral or external iliac aneurysms.

The main trunk supplies the biceps, semimembranosus, semitendinosus, and a portion of the adductor magnus.

In sciatica, tender spots may be found at the sciatic notch and in the middle of the posterior aspect of the thigh.

The *peroneal* or *external popliteal nerve* passes down the outer side of the popliteal space, and reaches the front of the leg by winding round the outer side of neck of the fibula in the substance of the peroneus longus muscle, where it may be readily rolled under the finger. It passes down deeply placed upon the interosseous membrane to the front of the ankle, on the outer side of the anterior tibial artery. On the dorsum of the foot it runs forward in the first interosseous space to give sensation to the contiguous sides of the great and second toes.

In its course it supplies the following structures:—two branches to the knee-joint, the superior and inferior external articular, a branch—the *communicans fibularis*—to join a corresponding one from the internal popliteal, the *communicans tibialis*, to form the external or short saphenous nerve, which is distributed to the dorsal aspect of the outer side of the foot, and part of the little toe; by its musculo-cutaneous branch, both the peronei muscles, the longus and the brevis, and the skin of the dorsum of the foot on the inner side of the great toe, and the contiguous sides of the second and third, third and fourth, fourth and fifth, with a twig to reinforce the external saphenous on the outer side of the dorsum of the little toe; and, by the main trunk of the anterior tibial nerve, the following muscles, the *tibialis anticus*, *extensor longus digitorum*, *extensor proprius hallucis*, *peroneus tertius*, and the *extensor brevis digitorum*.

The external popliteal nerve may be severed in punctured wounds of the upper part of the popliteal space or at the neck of the fibula. It is also liable to injury when the biceps tendon is divided subcutaneously, seeing that it lies adjacent to the inner border of this structure. Paralysis of the muscles supplied by this nerve is also frequently seen in cases of infantile paralysis.

The *internal popliteal* nerve runs down the middle of the popliteal space, and gives off branches to the following structures:—three to the knee-joint, the superior internal articular, azygos articular and inferior internal articular; a cutaneous

branch, the *communicans tibialis*, to join the *communicans fibularis* from the peroneal, and to form the external or short saphenous; and muscular branches to the two heads of the *gastrocnemius*, the *plantaris*, the *popliteus*, and part of the *soleus*. The branch to the *popliteus* is of interest, because it arises just as the nerve passes behind the upper border of the muscle, and passing posterior to the muscle winds round its lower border to supply the same on its deep or anterior aspect.

The *posterior tibial*, the continuation of the internal *popliteal*, runs down the back of the leg deeply placed, supplying in its course the rest of the *soleus*, the *tibialis posticus*, the *flexor longus digitorum* and the *flexor longus hallucis*, and divides a little above the ankle joint, about half-way between the internal malleolus and the heel, into the internal and external *plantar* branches, of which the internal is the larger.

The *external plantar* nerve supplies the following muscles, the *abductor minimi digiti*, *flexor brevis minimi digiti*, *accessorius*, the outer two *lumbricals*, all the *interossei*, the *adductor obliquus* and the *adductor transversus hallucis*, with a cutaneous branch to the plantar aspect of the contiguous sides of the fourth and fifth digits and the outer side of the little toe.

The *internal plantar* nerve gives branches to the *abductor hallucis*, the *flexor brevis digitorum*, *flexor brevis hallucis* and the two inner *lumbricals*, and a cutaneous supply to the plantar aspect of the inner side of the great toe and the contiguous sides of the first and second, second and third, third and fourth toes. As these digital branches proceed forwards to supply the skin covering the toes they pass between the heads of the metatarsal bones, and as the outcome of the crowding together of the bones by pressure of narrow boots, the nerves going to supply the contiguous sides of the fourth and little toes may be caught between the base of the first phalanx and the head of the metatarsal bone of the little toe on the one hand, and the head and neck of the metatarsal bone of the fourth toe on the other. Pain may not only be experienced at the actual site of pressure, but may radiate up nearly as high as the knee.

## CHAPTER XII.

### DISEASES OF THE VASCULAR SYSTEM.

#### HEART AND PERICARDIUM.

##### PERICARDITIS.

INFECTION carried by the blood stream or by lymphatics, is probably the commonest cause of pericarditis. This is the explanation of rheumatic, tuberculous, septic and some pneumonic varieties. The vessels and lymphatics of the serous pericardium are intimately connected with those of the myocardium so both the heart and its investing sac may be involved simultaneously.

Extension from neighbouring structures occasionally gives rise to pericardial inflammation. The close way in which the pericardium is enveloped by the lungs and pleural sacs explains the occasional spread of inflammation to it from pneumonic, tuberculous or gangrenous lung. Empyemata may in similar manner give rise to purulent pericarditis. The connective tissue basis of the pericardium is continuous with that of the lung, pleura and mediastinum, and inflammation may simultaneously involve all these structures or extend from one to the other. Adjacent lymphatic glands may invade or infect the pericardium. Four groups of these lie in immediate contact with the sac. The glands which lie along the internal mammary arteries are in front, the cardiac group of glands above, the glands around the tracheal bifurcation, and those about the lower end of the œsophagus, behind. Most of these glands are in direct lymphatic connexion with the sac, and may themselves become enlarged as the result of pericarditis.

Occasionally aortic aneurysms burst into the pericardial cavity. The first part of the aorta is actually within the sac, and the descending thoracic aorta passes down close behind it. Aneurysms

in either of these positions may, by rupture, give rise to intrapericardial hæmorrhage. Since the œsophagus is in intimate relation with the posterior part of the sinus obliquus, the pericardium may be perforated by foreign bodies in, or opened by malignant disease of, this tube. Disease of the ribs, sternum or vertebræ may invade the sac by continuity, and abscesses in the heart wall may rupture through the visceral layer. Very rarely gastric

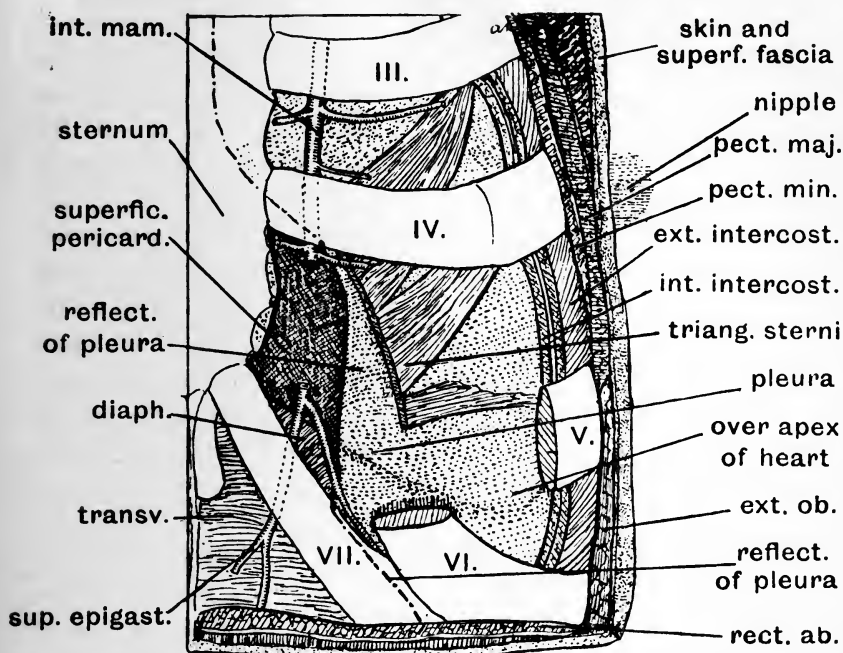


FIG. 24.— THE RELATIONSHIPS OF THE PERICARDIUM AND PLEURA TO THE THORACIC WALL IN THE APICAL REGION. (After Frohse, Hughes and Keith.)

ulcers have been known to open into the pericardium, this being rendered possible by the projection of the lower part of the sac beyond the anterior margin of the left lobe of the liver, so that only the diaphragm intervenes between it and the stomach. In some instances the pericardium bears a similar relation to the transverse colon near the splenic flexure. Subphrenic abscesses may open into the lower part of the sac.

The triangular area of superficial cardiac dulness corresponds to that part of the pericardium which is uncovered by lung. Its limits are the fourth costal cartilage above; the impulse of the heart externally; and internally, for all practical purposes, the left edge of the sternum. The extension of this dull area in pericarditis is due to distension of the sac, and gradual retraction outwards of the lung margins. It is stated that the costo-mediastinal pleural reflections do not always move outwards with retreating lung margins, in such cases the parietal pleura still covers the pericardium in front. The normal upper limit of the pericardial sac corresponds to the mid-point of the manubrium sterni, this being the summit of a tubular prolongation of the pericardium common to the ascending portion of the aorta and the pulmonary artery up to its point of bifurcation. (Fig. 26, p. 249.) In moderate pericardial effusions the area of percussion dulness is pyriform in outline, the upper narrower extension corresponds to this tubular prolongation, the lateral limits correspond with, or may pass beyond, the lateral limits of the heart as projected on the chest wall, and the lower limit is lower than the inferior border of the heart, for just as occurs in the case of the pleural sac, there is a reserve space in the pericardial sac and this space lies in front and below, so that the line along which the pericardium leaves the upper surface of the diaphragm in front crosses a little below the base of the ensiform cartilage.

The pericardial sac when distended, exercises pressure on the heart, and also on the structures in relation with it externally. The low pressure in the auricles and pulmonary veins and the comparative thinness of their walls, renders them more susceptible to pressure than are the other parts within the sac. Of the parts outside the sac the lungs suffer most easily. Pressure on the lower part of the left lung often gives rise to signs near the inferior angle of the left scapula, simulating pneumonia. Occasionally similar signs appear in the fourth and fifth interspaces of the right side near the sternum, and correspond in situation with the anterior part of the middle lobe of the right lung. Distension of the sinus obliquus which lies between the points of entry

of the pulmonary veins gives rise to some difficulty in swallowing, for the œsophagus lies immediately behind this part of the sac. The vagi are in close contact with the œsophagus and some authors refer the vomiting which occasionally occurs in pericarditis to pressure on these nerves. The weight of fluid in the sac tends to depress the diaphragm, but the heart does not travel downwards since it is fixed to the upper and posterior part of the sac by the veins and arteries.

The tendency of pericardial friction to persist along the line of the sternum is due to the fact that there is no spare room for antero-posterior distension, the heart being interposed between the projecting spinal column and the sternum. Distension tends rather to occur laterally and downwards below the cardiac level. In tapping the pericardium the pleura may be avoided by making the puncture through the fifth or sixth intercostal space of the left side close to the sternal edge, *i.e.* internal to the internal mammary vessels; but since it is often a matter of great difficulty to distinguish a dilated heart from a distended pericardium, it is safer to proceed surgically by resection of cartilage and exposure of the sac before puncturing it, or the under aspect of the sac may be reached through the abdominal wall in the subcostal angle without opening the peritoneum.

#### VALVULAR DISEASE OF THE HEART.

An accurate conception of the effects of the various valvular lesions on the heart can only be formed by a knowledge of its surface anatomy. The normal position of the cardiac impulse, or apex beat, the outlines of the cardiac chambers as projected on the anterior chest wall, and the positions of the various valves, must be thoroughly understood. (Fig. 26, p. 249.)

The **cardiac impulse** varies in position with age and with the posture of the body. In the infant the heart lies higher and is more horizontal than in the adult, possibly on account of the higher position of the diaphragm. The impulse is consequently found in the fourth intercostal space, but descends into the fifth

space by the seventh year. Until the fourth year it lies outside the line of the nipple, but should not pass more than three-quarters of an inch beyond this. From the fourth to the ninth year it beats in the nipple line. After the thirteenth year the impulse may be expected to occupy the same position as in the adult.

In the erect posture in the adult, the impulse should appear in the fifth left intercostal space, close above the sixth rib. It is three and a half inches from the mid-line in the male and three inches from the same line in the female. The area of impulse should not be quite so large as that of a circle an inch in diameter; in other words, it is completely covered by the bell of an ordinary stethoscope. The impulse may be made more apparent by leaning forwards. It may appear in the fifth interspace in recumbency, and yet in the erect position be found displaced behind the sixth rib, a fact which should lead to examination of patients in both postures if possible. Even though the heart is enlarged the actual apex may be so concealed by the left lung as not to be felt until the patient is turned well over on to the left side; this often causes the impulse to appear far back in the axilla in such cases, and gives some idea of the increase in size of the heart.

In old age the impulse is often displaced downwards into the sixth intercostal space, the heart being depressed by elongation of the ascending aorta in consequence of senile changes. (Fig. 25.)

The position of the normal impulse is often loosely defined as being in the fifth intercostal space a little below and internal to the nipple. The variable position of the nipple is a defect in this method of localising the position. The nipple should lie on or just below the fourth rib, four inches from the mid line of the body. When the breasts are enlarged or pendulous, the relations of the nipple are subject to considerable alteration.

The cardiac impulse is normally caused by the left ventricle, and, being superficial, affords important indications as to the condition of the wall of that cavity, becoming heaving in hypertrophy and feeble and diffuse in dilatation. In diseased conditions other parts of the heart may give rise to appreciable



DILATATION OF AORTA.

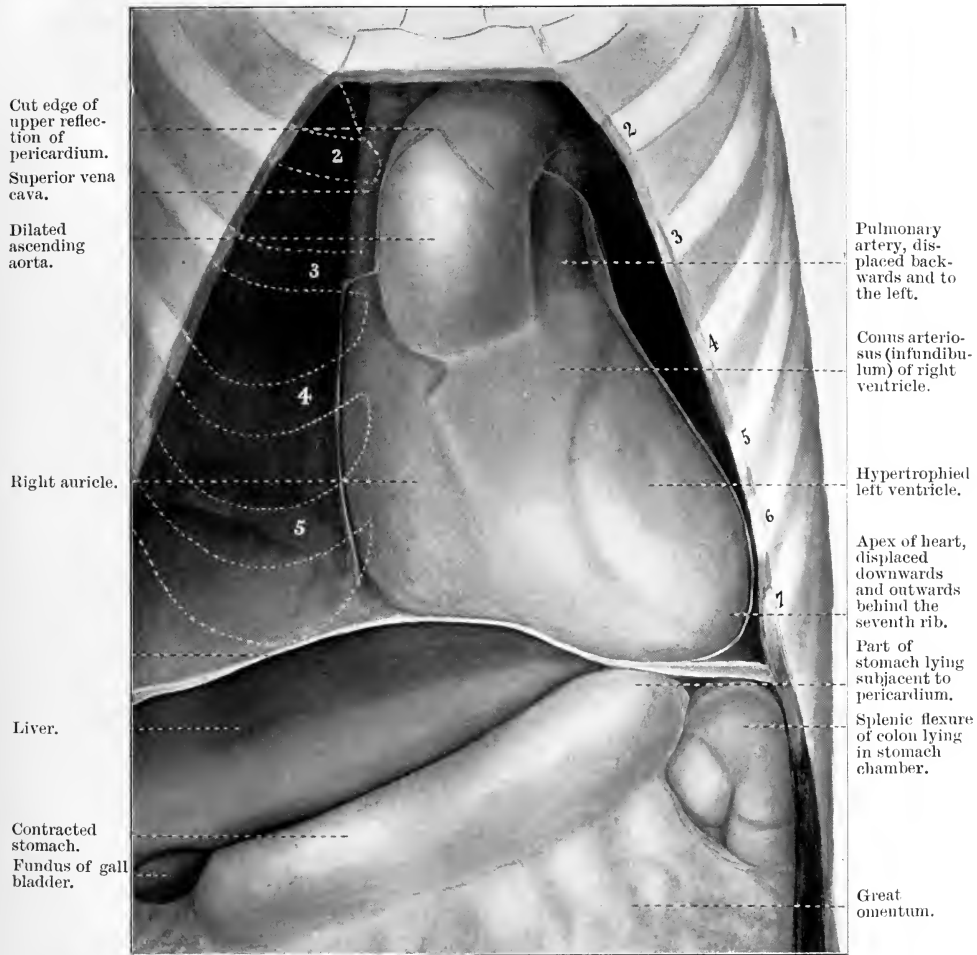


FIG. 25.—Dissection of an old subject in whom the aorta was diseased and dilated, and in consequence the heart displaced downwards and hypertrophied. The drawing also shows backward displacement of the pulmonary trunk. Below the diaphragm the relation of the splenic flexure of the colon to the stomach chamber, when the latter viscus is contracted, is well seen; also the relation of the stomach to the pericardial sac.



impulses, and the normal impulse may be concealed. For this reason it is more correct in all cases to speak of the visible or palpable thrust as the cardiac impulse than as the apex beat.

The **outline of the heart** projected on the chest wall is as

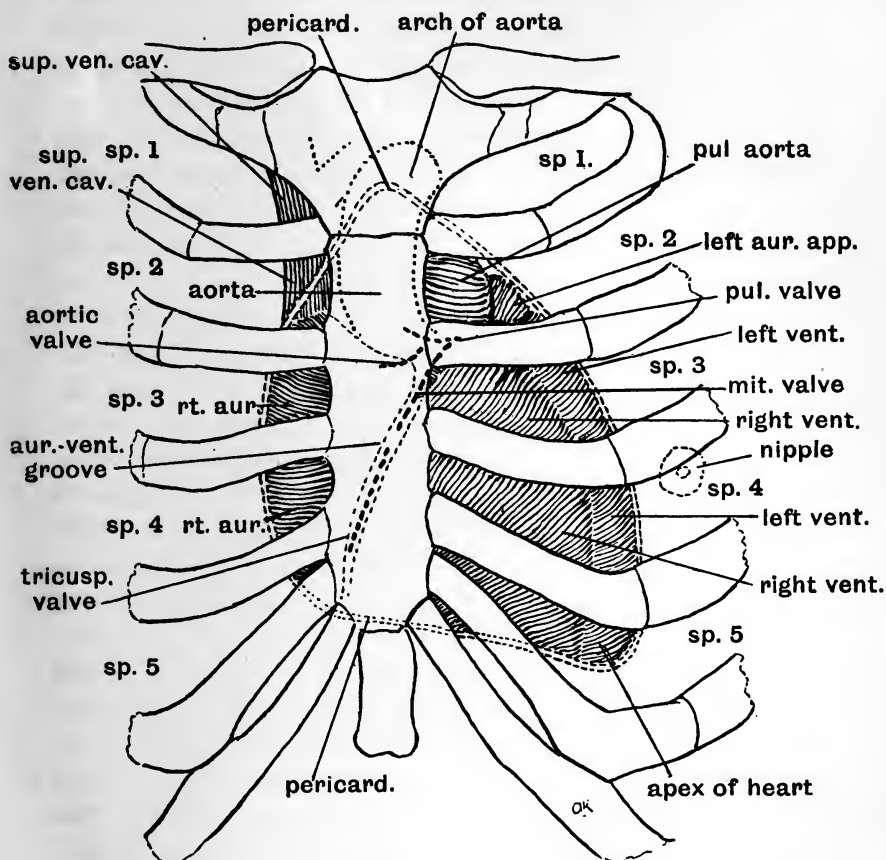


FIG. 26.—THE RELATION OF THE HEART TO THE WALL OF THE THORAX.  
(Hughes and Keith.)

follows:—The upper border is a line drawn across the sternum at the upper edges of the third costal cartilages; it extends half an inch beyond the sternum to the right and an inch to the left. The left border, which represents the margo obtusus of the left ventricle, corresponds to a curved line drawn from the left

extremity of the upper border to the impulse. This line is convex outwards, and at the level of the fourth left costal cartilage lies three inches to the left of the mid-line of the sternum. The right border, corresponding to the right auricle, is represented by a line, convex to the right, drawn from the right extremity of the upper border to the sixth right costal cartilage near the sternum. This line extends an inch and a half to the right of the mid-sternal line at the level of the fourth costal cartilage. The lower border, or *margo acutus* of the right ventricle, extends from the sixth costal cartilage of the right side to the impulse. This line passes to the left with slight downward obliquity, crossing the sternum at the base of the ensiform cartilage.

A line drawn obliquely across the cardiac area from the lower border of the left third to the right sixth costal cartilage, commencing at the mid-line of the sternum and terminating at the right edge, indicates the furrow between the right auricle, which lies above and to the right, and the ventricles which lie below and to the left. The leftmost strip of the cardiac area, nearly an inch broad, commencing at the lower border of the third left costal cartilage and terminating at the impulse, represents the anterior part of the left ventricle. Immediately above the left ventricular strip lies the left auricular appendix.

The heart is in contact laterally with resonant lung, and wedge-shaped portions of pulmonary tissue also pass forwards between its anterior surface and the chest wall. These wedge-shaped prolongations gradually thin off and leave uncovered the lower part of the anterior surface of the right ventricle. The part uncovered corresponds to the area of superficial cardiac dulness. The area is limited above by the fourth left costal cartilage, internally by the left edge of the sternum, and externally by an oblique line starting from the fourth left costal cartilage in the parasternal line and passing thence to the position of the cardiac impulse. This area is dull to light percussion, and can be easily determined. The deep cardiac dulness which should correspond to the actual outline of the heart

requires heavy percussion, and cannot be determined with anything like the same accuracy owing to the intervention of resonant pulmonary tissue and the vibrant qualities of the sternum.

The **valvular orifices** of the cavities of the heart lie so closely together that it is unsafe to assign a murmur to a definite orifice because it happens to be loudly heard at a surface area corresponding to the orifice in question. The valves which guard the orifices of the right side of the heart lie more or less in front of those belonging to the left cavities. This is especially the case with the valves of the pulmonary artery and the aorta. In diseased conditions the relations may become altered. The aorta may displace the pulmonary artery considerably to the left, and the tricuspid orifice may come more and more in front of the mitral. Nevertheless, as a general rule, pulmonary, aortic and tricuspid murmurs are best heard at spots corresponding to the positions of the respective valves as projected on the surface of the chest. The mitral valve is an exception, the murmurs belonging to this valve are best heard where the left ventricle impinges on the surface of the chest—*i.e.*, at or near the cardiac impulse.

The fibrous cusps of the valves are devoid of vessels. The vegetations of early endocarditis are not actually on the free edges of the valves, but lie in positions corresponding to the points of maximum contact of the cusps involved. As the result of inflammation the thinnest portions of the valves become contracted, and then the vegetations may appear to lie on the free edges. They appear on the side of the valve which is remote from the blood pressure—*i.e.*, on the ventricular aspects of the aortic and pulmonary valves, and on the auricular aspects of the mitral and tricuspid segments.

**Aortic Disease.**—The aortic valve lies under cover of the left edge of the sternum close to the lower border of the third *left* costal cartilage. It is in this position the aortic diastolic murmurs are earliest detected and best heard. The “aortic cartilage” is the second *right* costal cartilage, and corresponds

to the level at which the ascending aorta, in consequence of its forward inclination, comes nearest to the back of the sternum. An aortic systolic murmur is often loudest in this situation, but a faint diastolic murmur may be easily overlooked if only sought for at this spot. Diastolic aortic murmurs are conducted downwards over the left ventricle towards the impulse of the heart; systolic murmurs are conducted along the ascending aorta to the great arteries at the root of the neck. The forcible distension of the sinuses of Valsalva with blood during the elastic recoil of the aorta and other arteries in diastole occasionally drives air out of the overlying lung with a slight puff and simulates a diastolic murmur. Systolic murmurs may be produced in a similar manner. These exocardial murmurs may generally be recognised on account of the marked way in which they vary with the respiration.

The aortic valve segments are supported by a complete and strong fibrous ring, which offers great resistance to dilatation of the orifice. It is far more common for the mitral valve, which lies next behind the aortic in the circulatory circuit to give way under the stress of high back pressure. When dilatation does occur at the aortic orifice it is usually the result of degenerative lesions in later life. Paradoxically a dilated aortic orifice may give rise to a systolic murmur, indicating obstruction, as well as to the diastolic murmur of regurgitation. The obstructive murmur may be due to the tight stretching of the valve cusps across the lumen of the enlarged vessel.

The base of the anterior segment of the mitral valve is structurally continuous with the bases of the aortic cusps, so inflammation can and does often extend by continuity from the one valve to the other. The orifices of the coronary arteries lie behind the aortic valve segments, and although this position may shelter them from emboli, it renders them liable to implication in aortic disease, and adds to the gravity of the situation.

When the aortic orifice is obstructed or the valve incompetent, changes take place in the surface relations of the left ventricle. In stenosis, these may be the result of pure hypertrophy, but in

incompetence, the ventricle is usually as much dilated as hypertrophied. In both cases the outer boundary of the ventricle is carried to the left, and the cardiac impulse displaced downwards and outwards. The outward displacement of the left margin of the ventricle will increase the area of superficial cardiac dulness towards the left owing to the retraction of the lung from the surface of the enlarging heart. When the left ventricle is much hypertrophied and the right chambers of the heart are as yet but slightly involved, the impulse of the left ventricle can sometimes be felt in the epigastrium, but this is exceptional; an epigastric impulse as a rule is caused by the right ventricle.

**Mitral Incompetence.**— The mitral valve lies behind the sternum, opposite the attachments of the fourth costal cartilages. The right ventricle intervenes between it and the back of the bone. The systolic murmur of mitral incompetence is best heard at the cardiac impulse, this being the spot where the left ventricle comes nearest to the surface of the chest. The murmur may be traced towards the axilla, gradually fading away as the ventricle recedes, and a thicker wedge of lung becomes interposed between the heart and the parieties of the thorax. The murmur may in some instances be heard at the back between the scapula and the spine; it is probably conducted to this spot by an independent route, for that part of the base of the left ventricle which lies immediately below the left auricle rests directly on the structures in front of the spinal column. The murmur is therefore said to be conducted by the vertebræ from the base or “shoulder” of the left ventricle. Another explanation attributes the conduction of the murmur to the back to the presence here of the left auricle which receives the regurgitant stream of blood.

A systolic murmur may exist at the mitral orifice even though the valve cusps are not diseased. The ring of fibrous tissue which supports the valve segments takes its origin from the central fibro-cartilage of the heart. On the side of the orifice which lies remote from the fibro-cartilage, the fibrous ring is exceedingly thin and cannot offer much resistance to stretching. Consequently the mitral valve depends for its competency on the

transverse contractile power of the cardiac muscle rather than on the strength of its own fibrous foundation. Temporary weakness of the cardiac muscle therefore may occasion temporary mitral incompetence which will disappear when the myocardium recovers its normal tone. In conditions of high arterial tension such as obtain in chronic renal disease and in arteriosclerosis a murmur develops at the mitral orifice as the result of increased back pressure, although the stronger aortic valve, which is more directly exposed to the pressure, remains competent.

Mitral incompetence early gives rise to dilatation and hypertrophy of the left ventricle and the left auricle. Since the left auricle is interposed between the base of the left ventricle and the structures immediately in front of the spinal column, it is not accessible to clinical examination, but the rise of pressure in the cavity, transmitted backwards through the pulmonary veins, gives rise to an accentuation of the second sound at the pulmonary orifice. The pulmonary valves are superficial and lie at the upper border of the third left costal cartilage, at its junction with the sternum. The "pulmonary cartilage" is the second *left* costal cartilage, and marks the spot where the pulmonary artery approaches nearest to the surface of the body before dividing into its two large branches. The accentuated sound may be heard either over the valves or over the pulmonary cartilage; in the former position the valve closure may also be felt. Hypertrophy and dilatation of the right ventricle and auricle follow in turn. The results of increase in size of the left ventricle have already been discussed in the section on aortic disease, but it must be pointed out that the alteration of the position of the cardiac impulse in mitral incompetence is the resultant of two forces. The hypertrophy of the left ventricle displaces the impulse downwards and somewhat outwards, whilst the hypertrophy of the right ventricle tends to displace it outwards, and using the diaphragm as a fulcrum, even to lift it up.

**Mitral Stenosis.**—In mitral stenosis all the cavities of the heart, with possibly the exception of the left ventricle, ultimately become enlarged, and, if the occurrence of a pulmonary diastolic



murmur is accepted, every valve orifice, except the aortic, may be the site of a murmur.

The presystolic murmur of mitral stenosis is audible over a curiously limited area internal to the cardiac impulse. Both thrill and murmur are communicated from the solid structures of the heart to the chest wall, and by firm pressure with the stethoscope the murmur can be modified or even suppressed.

A systolic tricuspid murmur is common in the later stages of the disease. This murmur is heard over an area corresponding to the tricuspid valve. The tricuspid orifice is comparatively superficial, and lies obliquely across the vertical mid-line of the sternum opposite the fourth and fifth costal cartilages and the fourth intercostal spaces. The murmur is diffused over a considerable area of the lower part of the sternum, and often extends as far outwards as the cardiac impulse, which it is well to bear in mind is often caused by the right instead of by the left ventricle in these cases. The structure of the tricuspid fibrous ring is similar to that of the mitral, and the valve yields even more readily to pressure. Indeed the tricuspid valve may be said to be normally incompetent under increased pressure, this being a device to obviate over distension and even rupture of the right ventricle under circumstances of sudden muscular effort.

The increased pressure in the pulmonary circuit may result in atheromatous changes in the pulmonary artery and temporary incompetence of the pulmonary valves. In such cases a diastolic murmur appears a short distance to the left of the sternum just outside the position of the aortic valves. The murmur is transitory, disappearing when the circulation improves, and there appears good reason for the belief that it is really produced at the pulmonary orifice which is displaced in the same direction as the conus arteriosus, to the position of which reference will later be made.

The disappearance of the second sound at the impulse of the heart, which is a common occurrence in mitral stenosis, is probably due to interposition of the right ventricle in front of

the left, which thus prevents the normal conduction of the aortic second sound to the apex by the left ventricle. It is said that the pulmonary second sound is always inaudible at the impulse. Moreover, in mitral stenosis the aorta is insufficiently filled, its elastic recoil is consequently less and its second sound is fainter.

The left auricle, lying as it does immediately behind the site of obstruction, may attain a great size and is usually enlarged in all its diameters. Being interposed between the ventricle and the spinal column, it may actually, when distended, indirectly play a part in production of the cardiac impulse, by thrusting the ventricles forwards. In such exceptional cases the commencement of the impulse is really presystolic in time.

Upward enlargement of the left auricle may raise the roots of the lungs and actually exercise some pressure on the bronchi, since the latter are in contact with the upper part of the posterior surface of the auricular chamber. Lateral enlargement of the left auricle has been supposed to give rise to an outward extension of cardiac dulness in the third and fourth left interspaces, but this dull area is more correctly assigned to an upward enlargement of the conus arteriosus of the right ventricle. In some instances of great enlargement transversely, the left auricle has been found pulsating to the right of the sternal edge, having passed across the back of the right auricle, to which it is normally posterior, and then gained the surface on the right side. This great transverse or horizontal enlargement is usually rather associated with carditis and pericarditis than with mitral stenosis.

The pulmonary veins, which return the oxygenated blood from the lungs to the left auricle, dilate on account of the increased pressure in the latter cavity and may show patches of atheromatous change.

The obstruction to the pulmonary circulation causes the right ventricle to hypertrophy and dilate in its turn, and the muscular mass of this cavity is a very important factor in maintaining the circulation through the constricted mitral orifice. The right ventricle forms the greatest part of the anterior surface of the



MITRAL STENOSIS.

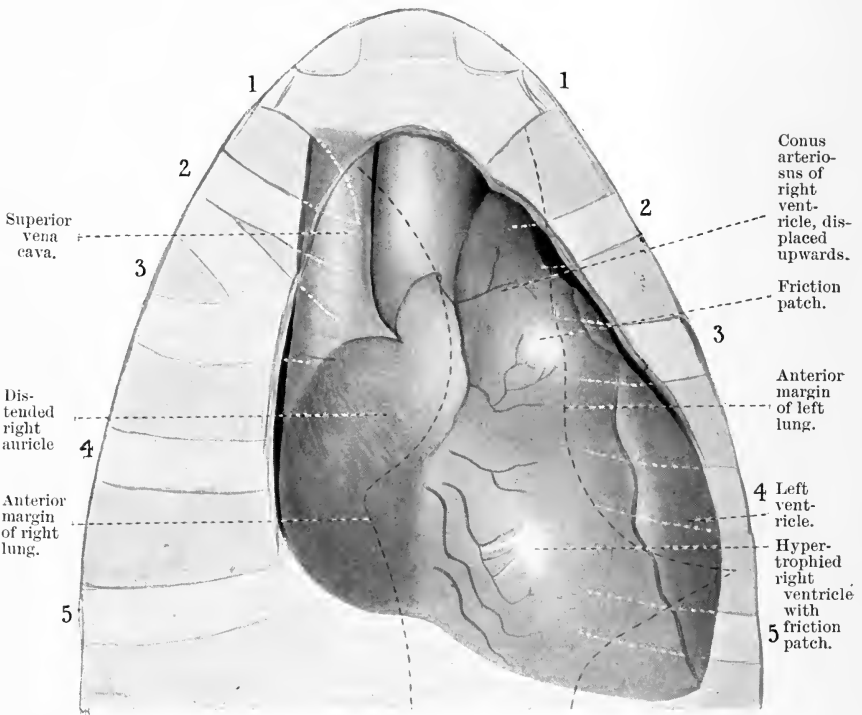


FIG. 27.—To show alterations in size and position of the right cavities of the heart, caused by a moderate degree of mitral stenosis.

heart. Its outline, as projected on the surface, has already been defined. Its inferior wall rests on the diaphragm. The results of hypertrophy of this chamber are the appearance of pulsation in the epigastrium and an increase of cardiac dulness in two directions, namely transversely to the left and upwards on the left side of the sternum. The upward extension of dulness is due to an increase in size of the conus arteriosus, which is that part of the right ventricle from which the pulmonary artery springs. The appendix of the left auricle normally lies in the position occupied by the upward extension of dulness, but as a rule the enlarging conus completely displaces the auricle. The enlarging right ventricle, pushing its way to the left, ultimately may completely separate the left ventricle from the anterior wall of the chest, and also takes the place of the latter cavity in producing the cardiac impulse. As a result the impulse is displaced mainly to the left and not so much downwards as in hypertrophy of the left ventricle.

The increasing pressure in the pulmonary circuit ultimately affects the right auricle. The outer limit of this auricle normally extends an inch and a half to the right of the sternum. It is covered by the anterior part of the right lung, and consequently takes no part in the superficial cardiac dulness. The presence of a wedge of lung tissue in front of the auricle, and the close approximation of the costal cartilages in this situation are obstacles to the satisfactory examination of this cavity of the heart. Increase in the size of the cavity causes the superficial cardiac dulness to extend up to or beyond the right margin of the sternum. Only when the auricle is much distended and the lung retracted is actual dulness obtained to the right beyond the sternal edge. Rarely the auricle may be seen to pulsate in this situation.

From what has been stated it will be gathered that the area of cardiac dulness is increased in three directions by mitral stenosis. The main increase is transverse to right and left, but there is also an extension upwards, consequently the percussion outline of pericardial effusion is in some instances very closely simulated.

## CONGENITAL DISEASE OF THE HEART.

**Lesions near the origin of the pulmonary artery from the right ventricle** are found in more than three-quarters of the cases of congenital disease. The pulmonary trunk, the pulmonary valves, or the infundibular part of the right ventricle may be involved. It is necessary to point out that the infundibulum, or that part of the right ventricle from which the pulmonary trunk arises, is also known as the *conus pulmonalis* or the *conus arteriosus*. The term *bulbus arteriosus* is applied to a segment of the embryonic heart, which, becoming subdivided, not only gives rise to the infundibulum of the right ventricle, but also to the corresponding part of the left and to the stems of the aorta and pulmonary artery. The bulbus, before this subdivision, is looked upon by embryologists as consisting of two parts, one part near the heart is the *bulbus proper*, and a more distal portion is called by them the *conus arteriosus*. Since the latter term is also applied to the infundibulum some confusion is apt to arise.

The right ventricle of the human heart is formed by the fusion of two chambers, the infundibulum, which is a part derived from the *bulbus arteriosus*, and the sinus, or body of the ventricle, which is the right half of the foetal common ventricle. The union of these constituent parts is indicated on the wall of the ventricle by a muscular lip, which projects downwards into the cavity and intervenes between the tricuspid and the pulmonary orifices. This projecting lip is known as the supra-ventricular crest. At the level of the crest a fibrous ring of thickened endocardium is sometimes found, and this, when much exaggerated, forms a diaphragm with a central perforation of varying size. When this is the case, a heart with three ventricles results, the infundibulum being sharply separated from the sinus of the ventricle. (Fig. 29.) In other instances the development of the infundibulum is arrested at an early stage, and the chamber is represented by a small cavity or even

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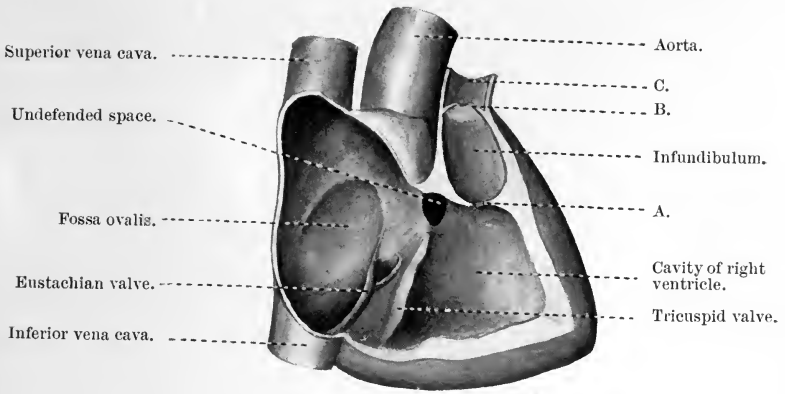


FIG. 28.—Diagram of right cavities of the heart to show possible sites of obstruction at each end of the infundibulum of the right ventricle. Obstruction at A gives rise to a heart with three ventricles. Obstruction at B is in the position of the pulmonary valve. Obstruction at C involves the stem of the pulmonary artery.

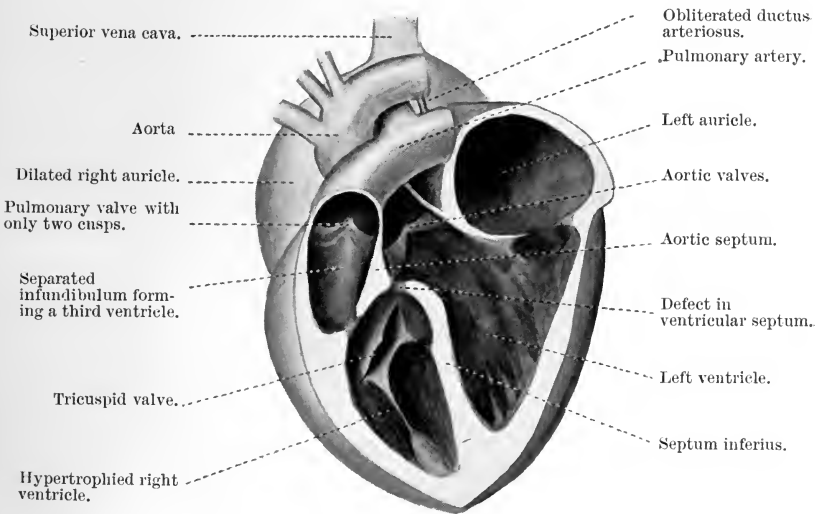


FIG. 29.—Heart with three ventricles, from a man aged 19 years. Semi-diagrammatic. (St. Thomas's Hospital Museum.)





a narrow strait of communication between the pulmonary trunk and the sinus or body of the ventricle.

The pulmonary cusps are developed at the distal end of the bulbus arteriosus from endocardial projections known as cushions, which give rise to the segments of both the aortic and the pulmonary valves. The pulmonary orifice may become narrowed or even completely occluded by developmental defects in this position.

The common arterial trunk beyond the valves is the conus arteriosus of embryologists. This becomes subdivided into the pulmonary artery and the aorta by the growth and fusion of dorsal and ventral ridges which, commencing above at the origin of the fifth branchial arch, grow downwards with a spiral twist to unite with the endocardial cushions from which the semilunar valves are derived. Narrowing or obliteration of the pulmonary trunk may result from a faulty position of the spiral septum. Defect in coalescence of the ridges which form the septum may be the cause of certain rare communications between the aorta and pulmonary artery, whilst complete failure of the septum will result in the presence of a common trunk which conveys blood to the lungs and the systemic vessels as well.

Thus it comes about that congenital defects may occur on the right side at the junction of the infundibulum with the sinus of the right ventricle, a position normally indicated by the supra-ventricular crest and lying just above the undefended space; or in the infundibulum itself, or at the site of the pulmonary valve, or in the pulmonary trunk between the valve and the ductus arteriosus. Of these defects the commonest is one at the position of the pulmonary cusps. (Fig. 28.)

Congenital defects of the infundibulum or of the pulmonary artery are often associated with defects in the ventricular or auricular septa. Malpositions of the aorta and pulmonary artery often coexist as well. The spiral septum of the conus arteriosus not only separates the pulmonary artery from the aorta, but also, after fusion with the endocardial cushions of the bulbus from which the semilunar valves are derived, projects downwards

into the ventricle as the "aortic septum" to complete the septum between the ventricles. Consequently it is quite possible that the septal defects and malpositions of the great vessels may be due to the same unknown influences which cause the pulmonary obstruction. However this may be, the septal defects and arterial malpositions are usually looked upon as secondary results of the pulmonary lesion and explained as follows:—

When stenosis of the pulmonary artery occurs before the eighth week of foetal life, the ventricular septum is as yet incomplete, and the excess of pressure in the right ventricle is relieved by the passage of some blood across the top of the septum into the left ventricle. As a result the septum fails to complete its union with the elements which enter into the formation of its upper part and remains unclosed. The congestion in the right auricle is similarly relieved by the passage of blood across the incomplete auricular partition. The high pressure in the right ventricle dislocates the growing ventricular septum to the left, so that both the aorta and the pulmonary artery retain their connection with the right ventricle, this being the portion of the common ventricular cavity with which they communicate at their first formation. When there is a considerable aperture in the ventricular septum, this may by itself suffice to relieve the overpressure in the right cavities, and the auricular septum may become complete. Should the ventricular septum be entire, having completed its growth before the pulmonary obstruction occurs, the auricular septum, which is completed later than the ventricular, must of necessity remain pervious, otherwise there is no sufficient exit for the blood from the right cavities of the heart.

Complete obliteration of the infundibular channel or of the pulmonary artery is usually spoken of as atresia. The secondary cardiac changes which result are similar to those which occur with stenosis. The right ventricle carries on the systemic circulation, the left becoming atrophied, since it fails to establish its proper connection with the aorta. If, however, the ventricular septum is complete and the aorta has established its normal

CONGENITAL HEART DISEASE.

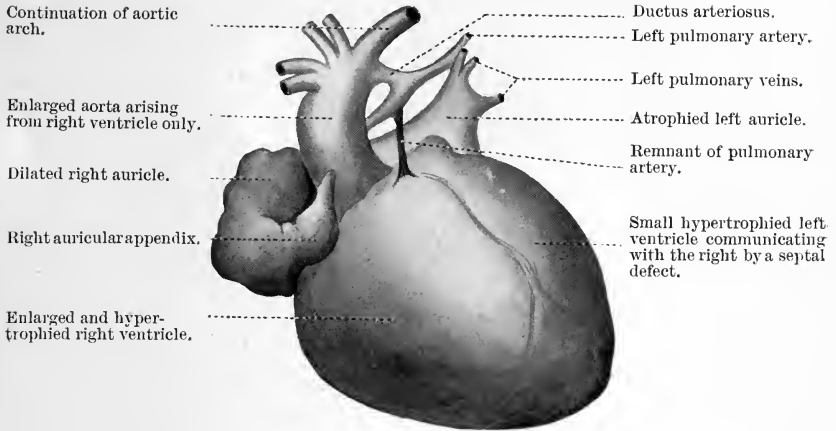


FIG. 30.—Atresia of trunk of pulmonary artery. Aorta arising from the right ventricle. Death at four years of age.

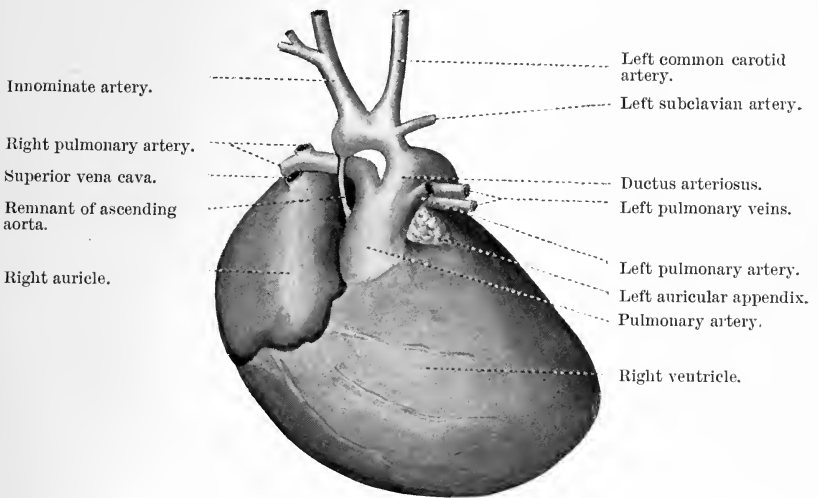


FIG. 31.—Atresia of aorta. The foramen ovale was patent, but the ventricular septum complete. The left chambers of the heart were atrophied.





CONGENITAL HEART DISEASE.

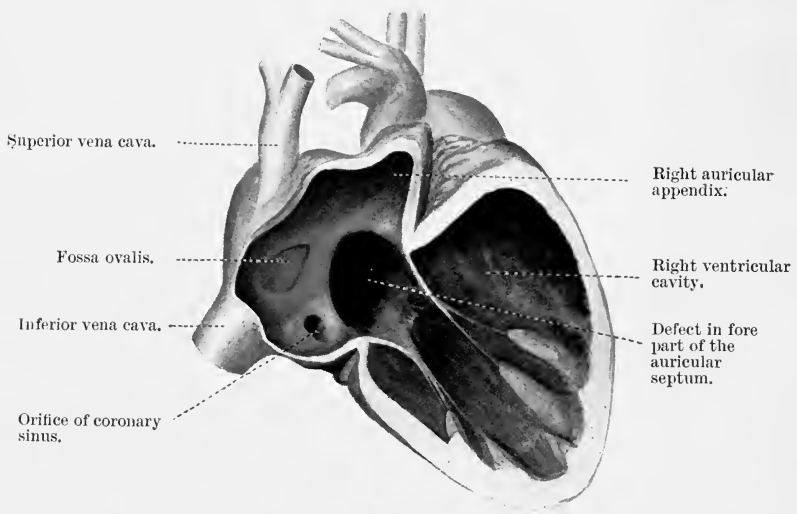


FIG. 32.—Heart of adult with large defect in the auricular septum. Fossa ovalis intact. (St. Thomas's Hospital Museum.)  
Semi-diagrammatic.

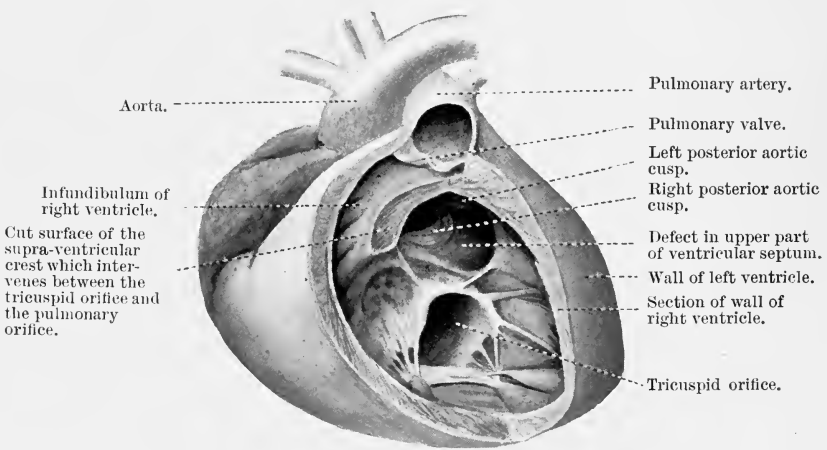


FIG. 33.—The heart of an adult in which the highest part of the interventricular septum is wanting. The aortic valves are seen through the septal opening, and the aorta communicates freely with both ventricles. (St. Thomas's Hospital Museum.)  
Semi-diagrammatic.

connexion with the left ventricle, it is the right ventricle which atrophies since it has no function to perform. As a general rule in congenital disease of the heart, those parts which in consequence of the defect are thrown out of the circulatory circuit will atrophy, whilst those which have increased work thrown upon them will become hypertrophied. (Fig. 30.)

When the pulmonary channel is obliterated the blood usually reaches the lungs through the ductus arteriosus, and this may also carry part of the blood when the pulmonary artery is stenosed. Sometimes, however, the pulmonary blood supply is carried by enlarged bronchial arteries, which, as is well known, show considerable variations in their points of origin.

**Defects in the cardiac septa** are very common in congenital heart disease, and are often, as already explained, associated with obstruction to the exit of blood from the right ventricle. They may, however, occur without such obstruction. A knowledge of the development of the cardiac septa throws light upon the positions in which septal defects are likely to be found.

Defects in the **auricular septum** are occasionally very large, the septum being represented merely by a crescentic fold at the upper and back part of the auricular cavity. The fold in such cases occupies the position at which the septum primum or primitive septum of the auricle is known to make its first appearance. If the septum primum has developed to a greater degree, but still failed to fuse with that part of the ventricular septum which lies between the auriculo-ventricular orifices, a defect will exist just above the top of the latter. (Fig. 32.) This opening, between the base of the tricuspid cusp and the position of the foramen ovale appears to correspond in position with the ostium primum of embryologists, for this ostium is between the lower part of the downgrowing septum of the auricles and the two endocardial projections which divide the common auricular canal into the right and left auriculo-ventricular orifices. Normally the ostium is closed by fusion of these structures. The substance of the septum primum becomes fenestrated and finally widely perforated in its upper part, giving rise to the ostium secundum, a part of

which persists as the foramen ovale. The ostium secundum is closed by the downgrowth of a secondary septum from the roof and posterior part of the auricle. This new septum, which lies to the right of the septum primum, is known as the septum secundum. Its curved lower edge, applied to the right side of the septum primum, forms the annulus ovalis, and thus comes to bound the foramen ovale above, whilst the septum primum bounds it below. Since the septum secundum restores the integrity of the upper part of the auricular partition, defects in this position are attributed to failures in the development of the septum in question.

Patency of the foramen ovale is due to a failure of development of that part of its membranous floor which is derived from the septum primum. An oblique valvular slit is frequently found at the margin of the fossa ovalis, and is caused by failure of the membranous floor to fuse with the annulus. Such an opening is kept closed during life by the excess of the blood pressure in the left auricle over that in the right.

Defects in the **ventricular septum** were at one time supposed to be always situated at the site of the pars membrancea septi or undefended space, but this is not strictly true. The ventricular septum is of compound origin, being formed above by the fusion of the endocardial cushions of the auricular canal with those of the bulbus arteriosus and below by the septum inferius which separates the primitive ventricle into two, and, growing upwards, fuses with the endocardial cushions. The septum inferius becomes attached to the anterior cushion of the bulbus in front and with the posterior cushion of the auricular canal behind. Between these two attachments lies the interventricular foramen. Quite early in foetal life this foramen becomes closed by fusion of its margins, and its site is indicated by the undefended space of the fully formed heart.

The upper part of the completed ventricular septum, in front of the undefended space, forms the septal wall of the infundibulum of the right ventricle, and is often known as the aortic septum. Indications of the compound nature of this portion of





CONGENITAL HEART DISEASE.

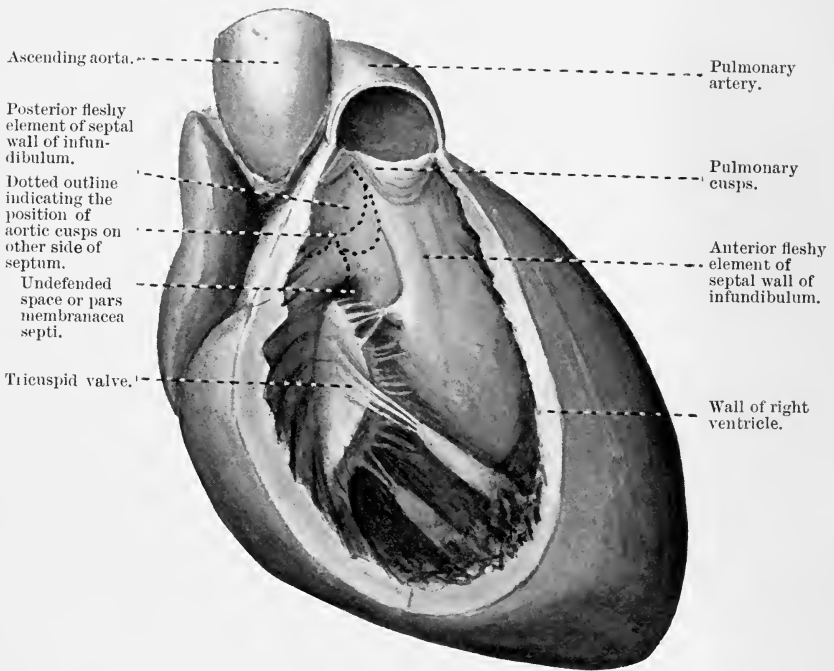


FIG. 34.—To show the elements which constitute the aortic septum or septal wall of the infundibular portion of the right ventricle, and the position of the pars membranacea septi or undefended space.

the completed ventricular septum may often be recognised. The angle formed by the meeting of two of the cusps of the pulmonary valve corresponds exactly in position with the angle between two of the aortic cusps on the other side of the septum. The cusps which are thus adjacent were continuous with each other until the spiral septum divided the common passage above them into the aorta and pulmonary artery. These cusps may be conveniently termed septal, and the anterior corresponding pair were developed from the ventral endocardial cushion of the bulbus, whilst the posterior corresponding pair arose from the dorsal cushion. On the septal wall of the infundibulum a line or furrow may sometimes be traced from the angle between the septal cusps of the pulmonary valve downwards and backwards to the undefended space. The portion of the septum in front of this line appears as a thick muscular band passing downwards and forwards; this represents the false septum of the reptilian heart. The portion behind the line passes downwards and backwards, and becomes in part continuous with the supraventricular crest already described. The line or furrow represents in its upper part the fusion of the endocardial cushions which divided the bulbus into the aortic and pulmonary orifices, and in its lower part, which inclines towards the undefended space, represents the line of fusion of the aortic septum with the septum inferius. (Fig. 34.)

The commonest site for a perforation of the interventricular septum to occupy is immediately in front of the undefended space. This corresponds to the lower part of the furrow of the aortic septum. A perforation posterior to the undefended spot is less common, and is usually associated with a defect in the adjacent part of the auricular septum, since it lies at the point of fusion of the septum inferius with the posterior endocardial cushion of the auricular canal. The undefended space itself may be defective in conjunction with septal defects in front of it or behind it. (Fig. 33.) The rarest defects of all are in the extreme fore part of the anterior region of the septum.

Apertures in the region of the undefended space and

immediately in front of or behind it will establish a communication between the left ventricle and the sinus portion of the right. A defect in the fore part of the septum will establish a communication between the left ventricle and the infundibulum of the right.

**Lesions at the origin of the aorta or in the aorta itself** are much less common than the corresponding lesions of the pulmonary artery. Like the latter lesions they may involve the conus arteriosus of the left ventricle, the orifice of the aorta or the aortic trunk. Although rare, complete impermeability of the aorta appears to occur more frequently than the corresponding condition of the pulmonary stem. (Fig. 31.)

Stenosis or atresia of the aorta may also occur at or below the point where the ductus arteriosus joins it. Normally the aorta shows a slight narrowing at the situation of the ductus, to which the term aortic isthmus has been applied. Stenosis or atresia at this spot probably arises in connexion with the formation of the ductus. As a result of this the descending aorta may be entirely supplied by the ductus arteriosus. If the ductus becomes impervious or retains its connexion with the proximal portion of the artery, a collateral circulation is carried on by means of anastomoses between branches of the subclavian arteries which arise above, and the intercostal and phrenic arteries which arise below the constriction. The presence of the enlarged anastomosing arteries may enable the diagnosis to be made during life.

The aortic isthmus is succeeded by a fusiform dilatation known as the aortic spindle. The lower limit of the spindle is supposed to mark the site at which the atrophied right aortic arch joined the descending aorta. A second constriction which sometimes occurs lower than the constriction at the isthmus probably arises in connexion with the obliteration of this right aortic arch.

Congenital atresia or stenosis of the auriculo-ventricular orifices may be attributed to defects in the endocardial cushions which subdivide the auricular canal. (Figs. 35 and 36.)

CONGENITAL HEART DISEASE.

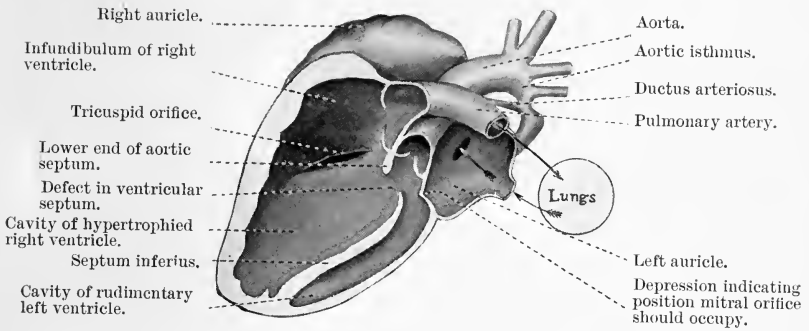


FIG. 35.—Mitral atresia, rudimentary left ventricle. Defect in upper part of the ventricular septum, and patent foramen ovale. Patent ductus arteriosus. Descending thoracic aorta supplied partly by aortic isthmus and partly by ductus arteriosus. Semi-diagrammatic.

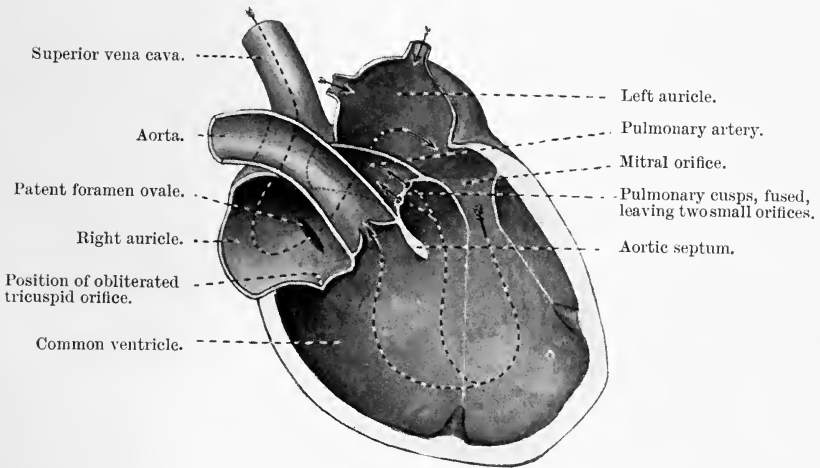


FIG. 36.—The heart of a "young person" with complete atresia of the tricuspid orifice. The aorta and pulmonary artery are transposed both laterally and antero-posteriorly. The pulmonary artery is stenosed at the site of its valves and the septum inferius of the ventricles is wanting. The course of the circulation is indicated by the dotted arrows. Semi-diagrammatic. (St. Thomas's Hospital Museum.)



Excess or deficiency in the number of semilunar cusps at the aortic and pulmonary orifices may similarly be due to malformations of the endocardial cushions of the aortic bulb.

## BLOOD VESSELS.

### HÆMORRHAGE.

Loss of blood may occur from any part of the vascular system. Hence hæmorrhage is often divided into the arterial, the capillary and the venous varieties.

**Arterial hæmorrhage** from a normal vessel is almost always due to a wound of the artery from without rather than a rupture of its walls by the blood pressure from within. The wound which implicates the vessel may be one which passes down to it through the skin or one which is made into it by the sharp edge of a fractured bone. Hence it follows that the more superficial the artery, the more likely it is to be injured from the surface, and the closer its proximity to a bone, the greater is its danger of laceration after fracture.

Except in the case of gun-shot injuries, arteries of the extremities are more commonly wounded than those of the trunk, and the vessels of the upper extremity more frequently than those of the lower. Probably because of their position the radial and ulnar arteries are the most common vessels to be accidentally divided.

**Arteries of Head and Neck.**—Wounds of the face are apt to be followed by severe hæmorrhage from the *facial* artery or from one or other of its many branches. Crossing the lower border of the mandible immediately in front of the masseter—that is, at a point about one-third the distance between the angle and the symphysis menti—the vessel passes tortuously towards the angle of the mouth, and then ascends more vertically, to terminate near the inner canthus of the eye. In the opening of a dental abscess in connexion with the lower molars, the artery may be exposed to injury unless the knife is kept close to the

bone when the incision is made from the inside, or the opening made well in front of the masseter if it passes through the skin. The coronary branches of the facial artery supply the upper and lower lips, and troublesome hæmorrhage may occur from the superior during an operation upon a hare-lip, or from either after a laceration of the frænum of the upper or the lower lip. The removal of an epithelioma from the lower lip will necessitate division and ligature of the inferior coronary vessels.

The *lingual* artery or its branches may be damaged in wounds of the tongue. As a rule the bleeding vessel can be secured by ligature, or the hæmorrhage, if only slight, controlled by the insertion of a suture. There is little, if any anastomosis between the arteries of the two sides except about the tip of the tongue, so that incisions made in the middle line bleed but little.

The *occipital* artery is deeply placed as it passes backwards before turning upwards on to the scalp, and in this situation a punctured wound may give rise to troublesome hæmorrhage. To expose the bleeding point it may be necessary to divide the fibres of the sterno-mastoid, the splenius capitis, the trachelo-mastoid and even the posterior belly of the diaphragm. Its descending branch, the *arteria princeps cervicis*, may also be injured in the posterior triangle of the neck.

Injuries about the tonsil and the lateral wall of the pharynx, particularly if of the nature of punctured wounds, may be associated with severe hæmorrhage. It is, however, seldom that such a large vessel as the *internal carotid* is opened up, seeing that its position in the adult is at least one inch from the mucous surface of this region. More usually the facial or one of its tonsillar branches, or the *ascending pharyngeal*, is the arterial vessel which is wounded.

In hæmorrhage from any of the branches of the external carotid artery distal to the superior thyroid, if the bleeding point cannot be secured directly, it will become necessary to ligature the main trunk of the external carotid. Because of the very free



communication between the superior and inferior thyroid arteries, the ligature upon the external carotid should be placed distally to the origin of the superior thyroid, so that no blood may be brought from the subclavian through the thyroid axis and its inferior thyroid branch to the superior thyroid, and so to the site of hæmorrhage. This anastomosis, and the danger of interfering with the blood supply of the brain, are the reasons why under these circumstances it is better to ligature the external rather than the common carotid artery.

In cut-throat injuries the vessels frequently escape any damage. This is chiefly because their position is at a posterior plane in consequence of the projection forwards of the thyroid cartilage, by which structure they are protected when the cut is made as high up as it generally is.

**Arteries of the Upper Extremity.**—It is uncommon to get injuries of any of the arterial trunks except those in the lower part of the forearm. The radial and the ulnar close above the wrist are frequently divided in association with the other structures, especially in such injuries as occur from the hand having been thrust through a pane of glass.

The *radial* artery lies on the radial side of the flexor carpi radialis tendon, and, being quite superficial, can be easily picked up and ligatured, if necessary, after some enlargement of the wound.

The *ulnar artery* is somewhat more deeply placed, lying to the radial side of the flexor carpi ulnaris tendon, and it is well to recollect the close proximity of the ulnar nerve, which is placed between the artery and the tendon, for the nerve may be damaged at the same time that the artery is severed.

Punctured wounds of the palm of the hand may involve one or other of the palmar arches. The *superficial palmar arch*, formed as a rule by the superficial branch of the ulnar being joined by the superficial volar of the radial, has its lowest point of convexity at the level of the fully outstretched thumb. A wound of this arch can be readily dealt with by ligature after an incision through the palmar fascia.

The deep palmar arch, on the other hand, is formed by the deep branch of the ulnar anastomosing with the termination of the radial in the palm of the hand, and the lowest point of its convexity is placed at a level which is one finger's breadth nearer the wrist than that of the superficial arch. It lies here deep to all the structures in the palm of the hand, being placed upon the metacarpal bones. Hence it follows that a wound of this arterial arch is very difficult to deal with. Properly applied local pressure is probably the best treatment to adopt in the first instance to arrest the hæmorrhage, unless an aseptic exposure of the bleeding point can be carried out. If, however, pressure should fail, or it is not thought advisable to cut down locally, it will become necessary to ligature the brachial artery. This vessel must be tied, because if the radial and ulnar were secured, blood might still be brought to the bleeding point through the crucial anastomosis in front of the wrist, consisting of the anterior interosseous on the proximal side, the anterior carpals laterally, and the recurrent branches of the deep palmar arch distally.

**Arteries of the Lower Extremity.**—Again, injuries of these vessels, like those of the upper extremity, are by no means common; they may result, however, from punctured wounds.

Undoubtedly the best treatment is to expose the injured vessel and to ligature it on the proximal and on the distal side of the opening into it. This, however, may be a matter of very great difficulty, on account of the effusion of blood into the muscular and cellular planes, the natural anatomical relations of parts being thereby greatly altered and the liability to sepsis very considerably increased.

Gangrene occurs more commonly after obliteration of the femoral artery than after closure of the brachial, because the distance from the heart—that is, from the pump—is greater, and the periphery of the limb will consequently be insufficiently supplied with blood. If the vein happens to be damaged at the same time that the artery is wounded, the probability of gangrene is still greater, the moist variety occurring on account of the obstruction to the return of the flow.

## ANEURYSM.

Certain arteries are more prone to be the seat of aneurysm than others, and many of the reasons for this fact are dependent upon anatomical considerations. Aneurysm of the thoracic aorta is more frequent than that of any other vessel. Following upon this artery the popliteal, the superficial femoral, the common carotid, the subclavian, the axillary, the innominate, and the abdominal aorta are not infrequent sites.

From an anatomical point of view it is convenient to subdivide **thoracic aneurysms** into three groups, namely, those arising from the ascending aorta, those which spring from the oblique segment of the arch, and those which occur in connexion with the descending portion. These limits are purely artificial, and are often overstepped by the progress of the disease. The only aneurysms which can attain a large size without producing serious internal pressure effects are those which project forwards or upwards from the anterior and upper aspects of the aortic arch.

The **ascending thoracic aorta**, or first part of the aortic arch, commences at the lower border of the third left costal cartilage, just beneath the sternal edge, this being the situation of the aortic valves. The vessel inclines upwards, forwards and to the right, and terminates under cover of the right half of the sternum at the level of the "aortic cartilage," which is the second costal cartilage of the right side. This part of the aorta lies in a pericardial sheath which is common to it and the pulmonary artery, so that both vessels are closely invested in the serous layer save where they come into contact with each other. The presence of this pericardial investing sheath accounts for the tendency of aneurysms near the root of the aorta to rupture into the pericardial cavity. The front of the vessel is at first concealed by the root of the pulmonary artery and the right auricular appendix, but higher up it is only separated from the sternum by the right pleura and the mediastinal tissues. Saccular aneurysms arising here tend to present in the second and third right intercostal spaces near the sternal edge, but they may

present in the second interspace of the left side, especially if they arise very near the origin of the aorta. They have also been known to separate the manubrium from the gladiolus, a process which is facilitated by the normal presence of a cartilaginous connection between these two parts of the sternum and the occasional presence of a synovial cavity.

The superior vena cava and the right auricular appendix are closely applied to the right side of the ascending aorta. The cava is sometimes perforated by an aneurysm, giving rise to highly characteristic symptoms and signs. These consist of sudden pain, followed by cyanosis and œdema of the head, neck and arms, the development of a continuous murmur running through systole into diastole, and the presence of a local thrill. When the superior cava is compressed, the resulting lividity and œdema are restricted to the head, neck and arms, but should the pressure be proximal to the point of entrance of the great azygos vein, the chest will also share in the congestion.

An incomplete spiral around the ascending aorta is formed by the pulmonary artery and its right main subdivision. The commencement of the pulmonary artery lies in front of the aorta, the main pulmonary trunk is closely applied to the left side of the vessel, and the right branch passes behind it. The relations of the vessels being so intimate, the pulmonary artery is liable to compression by aneurysms of the ascending aorta. These aneurysms sometimes rupture into the pulmonary artery. Sudden pain, with the development of a continuous murmur, signalise this accident, but œdema, such as occurs when the superior vena cava is similarly perforated, rarely occurs. The right bronchus is more or less separated from the aorta by the right pulmonary artery, but may, like the latter, be subjected to pressure with resulting secondary changes in the right lung. The right lung itself is only separated from the aorta by the superior vena cava and so may be subjected to direct pressure by the aneurysm. The right phrenic nerve lies on the right side of the superior vena cava, and may also be implicated.

Aneurysms at the junction of the ascending and transverse

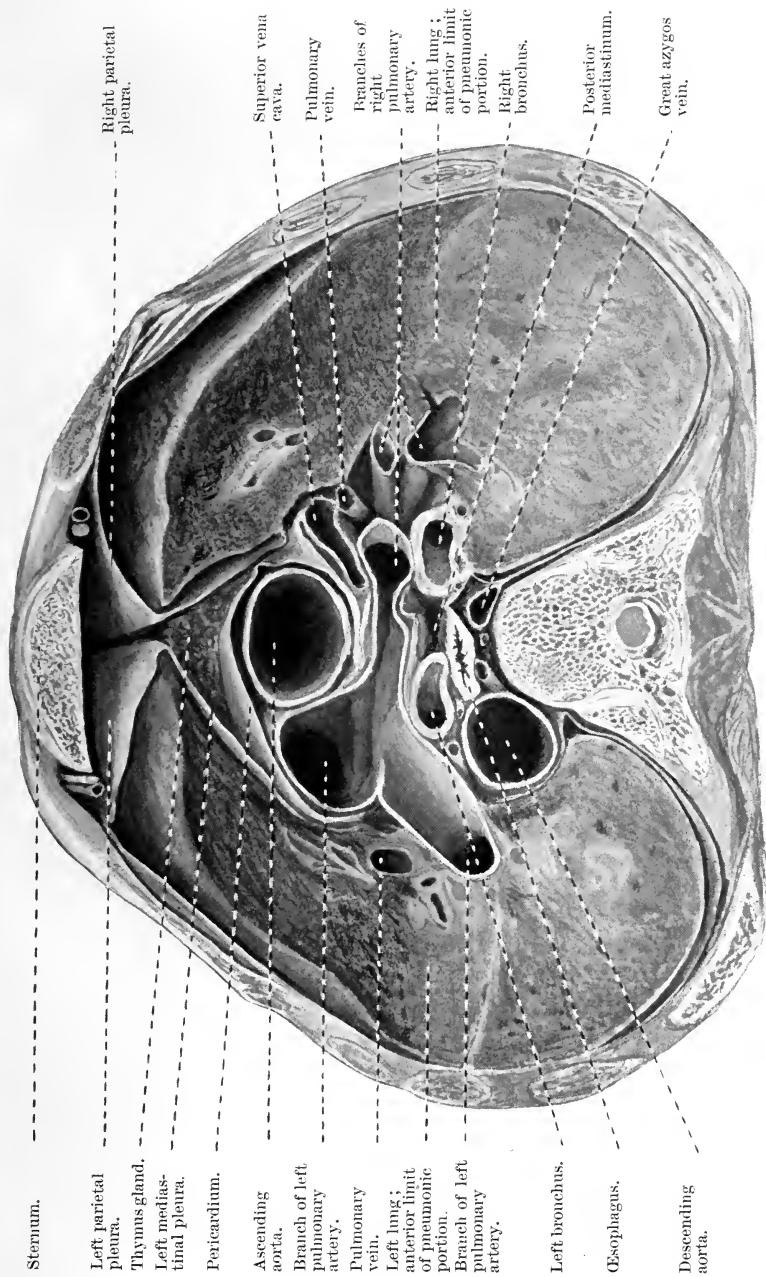


Fig. 37.—Partially dissected transverse section of thorax, passing through the roots of the lungs. From a patient with lobar pneumonia. (Museum, Royal College of Surgeons of England.)



portions of the aorta sometimes rupture into the right pleural sac, which lies close by.

The outline of a healthy ascending aorta normally presents several bulgings. The great sinus of the aorta, which is a longitudinal projection of its right border, must not be mistaken for an aneurysmal dilatation; it is occasionally present in the fœtus, but is best marked in old age. The three sinuses of Valsalva project near the root of the vessel. They correspond to the positions of the aortic valves. Aneurysm of the right sinus has opened directly into the right ventricle, with which the sinus is in relation; aneurysm of the anterior sinus has been known to open into the left ventricle. But such aneurysms are more likely to open into the pericardium with fatal results. These sinus aneurysms may compress and actually obliterate the adjacent branches of the coronary arteries. The orifices of the coronary arteries lie in the anterior and left posterior sinuses, and may be narrowed by the endarteritis which is associated with aneurysm, causing anginal symptoms. Aneurysmal dilatation of the ascending aorta has a tendency to displace the heart downwards, and disease of this part of the vessel is often associated with incompetence of the aortic valves which lie at its orifice.

The **arch of the aorta** is the oblique segment, often loosely termed transverse, which lies in the superior mediastinum. It passes from behind the right side of the sternum at the level of the second costal cartilage, at first upwards, backwards, and to the left, and then directly backwards to the left side of the fourth dorsal vertebra, at the lower border of which it becomes the descending aorta.

An aneurysm in this situation may impair the percussion note of the sternum or give rise to pulsation at the root of the neck. The upper border of the arch as it crosses the mid-line is usually an inch below the interclavicular notch of the sternum, so its pulsation cannot be readily felt, moreover, even when pulsation is recognised in this situation it may be due to the innominate or left common carotid arteries near their origins, or

even to a thyroidea ima artery, or a right subclavian with an abnormal origin to the left of the mid-line. In childhood the aorta may reach to the upper border of the manubrium, but at this period of life aneurysm is usually out of question. The ductus arteriosus, which connects the origin of left branch of the pulmonary artery with the aorta joins the latter just beyond the point of origin of the left subclavian artery. The cicatrix can easily be recognised when the aorta is laid open, and is often the site of atheroma. At this part of the aorta is a normal constriction known as the aortic isthmus, whilst beyond it the vessel often shows a fusiform dilatation which is not aneurysmal, and is called the aortic spindle. The arch of the aorta is moulded on the left aspect of the lower end of the trachea. It leaves its impress on this tube, and forces it decidedly to the right of the mid-line. An aneurysm of the arch may increase the tracheal displacement, so that even in the neck the air tube may be found to be shifted towards the right side. On the left aspect of the arch also lie the œsophagus, the left recurrent laryngeal nerve, and the thoracic duct. All of these structures may be subjected to pressure. An aneurysm in this region may open into the œsophagus. When the trachea is compressed stridor may be heard over both lungs, and is associated with a brassy cough and paroxysmal dyspnœa. The cough and dyspnœa are probably caused by irritation of the filaments which the vagi supply to the trachea.

The right lung and pleural sac are fairly well protected by the intervention of the structures already mentioned as being in immediate relation with the right side of the aortic arch, but aneurysms near the commencement of the arch may invade the right pleural sac.

The left side of the arch is in relation from before backwards with the left phrenic nerve, the superficial cardiac branches of the left vagus and left sympathetic, the left vagus nerve and the left superior intercostal vein. These are the only structures which intervene between the aortic arch and the left lung and its pleural envelope. A deep impression is made by the artery on



the mediastinal aspect of the upper lobe of the left lung. The lung may be directly compressed by aneurysms, and both the left vagus and the left phrenic nerves may be found adherent to the sac. Compression of the phrenic nerve causes paralysis of the corresponding half of the diaphragm. Apart from recurrent laryngeal paralysis it is difficult to correlate pressure on the vagus with definite symptoms. Rupture of the aneurysm into the left pleural cavity is not uncommon.

The lower concave surface of the aortic arch overhangs the root of the left lung, being placed astride the left bronchus and bifurcation of the pulmonary artery. The tracheal tug is best developed when an aneurysm springs from this aspect of the arch, the impulse being communicated directly downwards to the left bronchus, which drags on the trachea and the larynx. The left bronchus may be obstructed or opened, and the left pulmonary artery more or less occluded. Compression of the left recurrent laryngeal nerve, which turns round the aortic arch beyond the remnant of the ductus arteriosus, will produce at first abductor and finally complete laryngeal paralysis on one side. The upper surface of the aortic arch is in relation with the left innominate vein and three great arteries spring from it, the innominate and left common carotid arising close together and the left subclavian further back. Pressure on the innominate veins will give rise to enlargement of the veins of the head, arms, neck, and upper part of the chest, whilst implication of the origin of the arteries mentioned, or pressure on them by the aneurysmal sac, or involvement of their lumina by the accompanying endarteritis will modify their pulses in various ways.

Rupture into the pericardium is an unlikely accident to occur in connection with aneurysms of the aortic arch, but aneurysms at the junction of the arch and the descending aorta have been known to rupture in this way after working forwards above the left bronchus. Rupture into the mediastinal tissues gives rise to extravasation of blood around the œsophagus and pharynx, extending into the neck beneath the deep cervical fascia.

The **descending thoracic aorta** is closely applied to the back

of the root of the left lung and the back of the sinus obliquus of the pericardium. Paroxysmal cough, spasmodic dyspnoea, bronchitis, and deficient air entry in the left lung are very characteristic of aneurysm in the region of the lung root which presses on the back of the left bronchus, into which it may and often does open. More rarely the aneurysm opens into the trachea near its bifurcation. When the aneurysm lies lower down it is behind the heart and pericardium. It is rather exceptional for the pericardium to be perforated in this position, but both heart and pericardium may be forcibly pressed forwards against the anterior chest wall, giving rise to an extensive area of visible cardiac pulsation.

The œsophagus is at first to the right of the descending thoracic aorta, but later comes to lie in front of the vessel, and may even pass a little to its left side, just above the œsophageal opening in the diaphragm. In consequence of its intimate relation to the artery, dysphagia may be produced, and the aneurysm may burst into the food passage. Before passing a bougie in the investigation of dysphagia the possibility of the presence of an aneurysm of this part of the aorta should be carefully considered.

By backward expansion the aneurysm may erode the vertebral bodies. The yielding intervertebral discs persist in a characteristic way, while the resistant vertebral bodies are extensively destroyed. Deep-seated gnawing pain may be the result of the bone erosion, and the corresponding intercostal nerves may be involved, with resulting intercostal neuralgia and segmental hyperæsthesia or anæsthesia. Pressure may be exerted on the spinal cord, either gradually by the advancing aneurysm or suddenly as the result of fracture of the weakened spinal column. In the upper part of the posterior mediastinum the descending aorta is placed on the left sides of the bodies of the vertebræ, and when aneurysm is present an area of dulness, and even of pulsation with projection, may be recognised between the spinal column and the scapula on the left side. The gangliated chain of the sympathetic lies over the costo-central articulations

in this region and may be compressed, but this is rather too low for the pupil fibres to be caught. Lower down in the thorax the aorta lies in a more median position in front of the spine, and behind the vertical part of the diaphragm.

The left pleural sac is closely applied to the left aspect of the vessel, and an aneurysm of this part of the aorta is more likely to rupture into the left pleura than elsewhere. But the right pleural sac is also in relation, being only separated by the thoracic duct and the œsophagus, and even projects behind the œsophagus in the lower part of the thorax in the form of a cul-de-sac, which comes very close to the aorta. So it is not surprising that the percentage of cases of rupture into the right pleura is quite half of that of rupture into the left.

An aortic aneurysm rarely produces symptoms of pressure on the thoracic duct, although the latter is in close proximity; the pressure increases gradually, and probably a collateral lymphatic circulation has time to establish itself.

Aneurysms of the upper part of the descending thoracic aorta have been known to rupture into the mediastinal or sub-pleural tissue, surrounding the œsophagus in tubular fashion, invading the connective tissue basis of the pericardium, burying the vagus nerves and extending even under the serous coat of the stomach. Anomalous symptoms may thus be produced, of which anæmia and vomiting are the chief.

An **innominate aneurysm** is generally associated with some dilatation of the aortic arch itself, and often of the subclavian and common carotid. From the position of the innominate artery the tumour usually appears above the sternum and slightly to its right, and tends to raise the right sterno-mastoid muscle near its origin. The pressure it exerts upon the superior vena cava may lead to considerable dilatation of the venous tributaries of that vessel; hence congestion of both upper extremities and of the head and neck is liable to be in evidence. The right radial pulse may be considerably altered in character, both in the way of size and time.

An aneurysmal sac upon the common **carotid artery** is

usually found near its bifurcation. Pressure symptoms will be in evidence, such as dyspnoea, dysphagia, alteration in voice, and cough, owing to the sac pressing upon the trachea, œsophagus and larynx. Interference with the proper circulation of blood through the brain on the affected side will cause giddiness and some tendency to syncope. Embolism of one or other of the cerebral vessels not infrequently occurs, and hemiplegia may result.

A **subclavian aneurysm** is most usually met with in the third part of the vessel, and more frequently on the right than on the left side. The tumour develops above the clavicle external to the sterno-mastoid, forming one of the swellings met with in the lower part of the posterior triangle of the neck. In connection with its diagnosis it is well to remember the possible occurrence of a cervical rib, which may raise the artery in such a way as to give a very close resemblance to an aneurysmal sac. The most marked evidences of pressure are found in the œdema of the upper limb, from interference with the return of blood through the subclavian vein, which lies anterior to the artery; and in the radiating pain over the area of distribution of the cords of the brachial plexus, which lie posterior and external to the sac.

The **axillary artery** may be the site of an aneurysm, which is most usually of traumatic origin. It frequently assumes large proportions, on account of the looseness of the surrounding tissues, which give the vessel but little support. Again, pressure upon the accompanying vein leads to considerable œdema of the limb on the distal side, and likewise pressure upon the surrounding nerves occasions pain, or marked interference with their function.

An aneurysm upon the **brachial artery** is usually seen at the bend of the elbow, and is dependent as a rule upon traumatism. Not infrequently it is of the nature of an arterio-venous aneurysm, the outcome of a wound of the median basilic vein and of the brachial artery, over which it lies.

An aneurysm of the **abdominal aorta** does not as a rule give

marked evidence of pressure symptoms except upon the subjacent lumbar vertebræ and the nerves associated with them. It tends to increase forwards, this being the line of least resistance.

The **popliteal artery** has the following anatomical peculiarities which may explain the frequency with which it becomes the site of a sacculated aneurysm. It is constantly being bent in flexion of the knee, whereby a certain amount of strain may be thrown upon it; it derives little if any support from the surrounding muscles and tendons, and it is close above a bifurcation.

The position of the vessel upon which an aneurysm forms determines many of the characteristic signs caused by dilatation of the artery and the pressure of the sac upon the surrounding structures.

The first structure to be pressed upon is its accompanying vein. The popliteal vein is the thickest vein of its size in the body, but is the most adherent to its artery; hence it follows that pressure upon it soon leads to considerable œdema of the limb on the distal side. The internal and external popliteal nerves may be involved in the pressure, causing interference with their function, the pain frequently induced being attributed to rheumatism. The popliteal surface of the femur, the posterior ligament on the knee-joint and the upper part of the tibia all come in for their share of pressure, and considerable erosion may occur.

It must be remembered in connection with a popliteal aneurysm that various swellings which occur in the popliteal space may lie superficial to the artery and obtain thereby communicated pulsation, which without careful differentiation may lead to an error in diagnosis.

In the treatment of an aneurysm anatomical considerations are of the greatest importance.

For an aneurysm situated in a limb there can be little doubt that the ideal treatment is to ligature the artery upon which the aneurysm is placed both on the proximal and on the distal sides of the sac, and to dissect out the sac itself. This, however, may

be a matter of considerable difficulty, seeing that the dilatation upon the vessel so markedly alters the normal anatomical relations. It may be under these circumstances by no means easy to find the main vessel on the proximal or distal side of the sac, and to apply the two needful ligatures to it. Again, the accompanying vein (or veins) may be so closely applied to or stretched out upon the aneurysmal sac that it is well-nigh impossible to prevent injury to or even removal of the venous channel. Hence it follows that gangrene may be almost invited by the procedure required for the removal of the sac. The remarks here given apply in a peculiar sense to a popliteal aneurysm.

A very much more simple, and at the same time not infrequently efficacious, operation is to ligature the main artery leading to the sac on the proximal side of the aneurysm. The vessel may be ligated fairly close to the sac or at some distance on the proximal side, the point chosen depending to a very great extent upon the length of proximal artery available. The operation of ligature of the artery on the proximal side at some considerable distance from the sac is probably the better of the two operations, and that for certain anatomical reasons. The artery will here be in its normal anatomical relations, will be less likely to be unsound, particularly in the matter of dilatation of its coats, and will be readily separated from the veins in its proximity. Further, ligature at this point will secure a freer collateral circulation. Since the artery remains pervious from a short distance below the ligature to the aneurysm, blood can enter it by collateral anastomosis, and so eventually reach the aneurysmal sac, but in a small stream and under diminished pressure. Hence it follows that there will be a greater likelihood of the clotting of the blood thus introduced into the sac, and so a greater probability of cure. It will also follow that the opening up of this collateral circulation will tend to diminish the possibility of subsequent gangrene.

Where there is not room for a proximal ligature to be applied to the artery feeding the sac, it may be advantageous to ligate

either the main trunk on the distal side of the aneurysm, or one or more of the branches into which it may divide. In order, however, that success may follow such a procedure it is necessary that there should be no large branch given off by the vessel between the sac and the point at which the ligature is applied. Perhaps nowhere in the body is this anatomical requisite so evident as in the case of the common carotid. Supposing an aneurysmal sac to be formed on the proximal part of the vessel, a distal ligature will effectually prevent the passage of blood beyond the aneurysm, seeing that there is no branch into which it can pass. Another disadvantage of a distal ligature, particularly as seen in the limbs, is the liability of gangrene following upon the occlusion of the artery. In instances of subclavian aneurysm in which there is little chance of applying a ligature upon the proximal side of the sac with any degree of safety, a distal ligature is apt to cause death of the limb beyond, or failure in bringing about a cure of the aneurysm. Therefore it has been considered advisable to as it were perform distal ligation, but at the same time to remove the part which has to be supplied with blood, by an amputation at the shoulder-joint.

Pressure has been known to cure many aneurysms, in fact it may be one of nature's methods of bringing about the cessation of the continued dilatation of the sac. From its anatomical relation the aneurysmal sac may itself cause pressure upon the artery feeding it on the proximal side, and thus diminish the amount of blood passing into the sac, and so allow the deposition of clot.

This proximal pressure has been carried out by the surgeon, but as a rule at a greater distance away from the sac than the spot at which nature applies it. Hence in a popliteal aneurysm digital pressure is best applied to the last part of the external iliac or the first part of the common femoral backwards against the horizontal ramus of the os pubis. If such pressure by the finger is carefully applied, the return of blood through the femoral vein will not be interfered with—an advantage not secured by the mechanical pressure of a tourniquet.

Digital pressure upon the artery at this spot, should it fail to bring about a cure of the aneurysm, has not in any way been a disadvantage in view of a subsequent ligature. First of all the vessel will not be tied at the point over which the digital pressure has been applied, but at a more distal spot, either at the apex of Scarpa's triangle or in Hunter's canal. Secondly, it will have opened up the collateral circulation, and thus have diminished the likelihood of gangrene subsequent to ligature. It is always easy to diminish the amount of blood passing into a limb, but difficult to increase it.

#### VARICOSE VEINS.

It is not every vein in the body which is subject to varicosity, but certain veins, chiefly from their anatomical position, are prone to become varicose. The superficial veins of the lower extremity, the spermatic veins, and the veins of the inferior hæmorrhoidal plexus are those which most frequently undergo the change which is termed varicosity.

**Varicose Veins of the Lower Limb.**—The anatomical reasons why the superficial veins of the lower extremity are so commonly varicose may be stated as follows: they have a long column of blood to support, in the adult often as much as four feet, though of course the weight is partly borne by the series of bicuspid valves present within the vein; their distance from the heart renders the *vis a tergo* of the systole less powerful than elsewhere, and distension likely; and their want of support, owing to their being surrounded merely by subcutaneous tissue and not by muscle.

There are two chief varieties of varicose veins in the lower limb. The first is that in which one vein, generally the *long* or *internal saphenous*, is varicose. This vein commences from the inner side of the venous arch on the dorsum of the foot, passes then in front of the internal malleolus, up the inner side of the leg, behind the internal condyle of the femur, along the inner side of the thigh, to pass through the saphenous opening and join the femoral vein close below Poupart's ligament.



The whole length of the vein is supplied with valves, the last of the series being usually just below the passage of the vein through the saphenous opening. Hence it is that in some cases of varicosity a fluid swelling is found in this region simulating somewhat a femoral hernia. In varicosity the position of the valves is frequently evidenced by the greater dilatation of the vein opposite them. The pain felt is in some cases due to pressure upon neighbouring filaments of the internal saphenous nerve.

The *short* or *external saphenous* vein may also be alone varicose. This vessel begins on the outer side of the dorsal venous arch, passes behind the external malleolus, up the outer and posterior part of the calf, to pierce the deep fascia in the popliteal space and empty into the popliteal vein.

In the second variety, varicosity is not confined to the internal or external saphenous, but affects almost the whole of the superficial venous tributaries of the limb, including the radicles beneath the skin. Ulceration of the skin is peculiarly liable to be associated with this form of varicosity. It occurs typically in the lower half of the leg, and usually close above the ankle and on the inner side. This selection of position for the varicose ulcer is probably due to the fact that the veins of the foot itself are supported by the wearing of boots or shoes, but just above the upper limit of the footgear much strain is thrown upon the veins, considerable stagnation occurs, failure in the nutrition of the skin results, and often precursory eczema puts in an appearance. Then it has to be borne in mind that the lower third of the leg is open to frequent injury, and once there is abrasion of the congested skin staphylococci gain entrance and play havoc with the part.

The exquisite tenderness of some varicose ulcers is due to the exposure of the fine nerve terminations by the ulceration of the surrounding tissues.

Copious hæmorrhage sometimes occurs by the erosion of a large vein, such as the internal saphenous, during the course of a varicose ulcer. Whilst the limb is vertical the blood lost comes

from the proximal portion of the vein, that is the long tube on the cardiac side, and this is owing to the fact that the valves have become incompetent because of the dilatation of the vein without any corresponding increase in the size of the valve itself. Hence it is that elevation of the limb tends to immediately bring about the temporary cessation of the flow of blood.

The operative treatment of varicose veins in the lower limb usually consists in the obliteration of lengths of the vein, so as to force the blood to be returned by the deep veins, which have free communication with the superficial. The operation of tying the internal saphenous just below the saphenous opening has as it were the effect of placing a competent valve high up, and of thus cutting off the weight of the column of blood.

**Varicocele.**—Several anatomical considerations show the reason why varicocele, and particularly left varicocele, is such a common affection.

The blood supply of the testicle, entering through the spermatic artery, is returned by the spermatic veins. These commence by a number of venous radicles in the hilum of the testis and soon form the pampiniform plexus in the anterior part of the cord. Passing upwards through the superficial abdominal ring they unite into a smaller number of veins in the inguinal canal, and entering the extra-peritoneal tissue at the site of the deep ring, they join to form the two *venæ comites* of the spermatic artery. On the right side, these unite to form a single vein which opens into the inferior vena cava at an acute angle, at a varying distance below the entrance of the right renal vein. On the left side, the two spermatic *venæ comites* join to form a single trunk which opens into the left renal vein at a right angle.

The length of the veins implies a considerable column of blood to support. Valves are either wanting or very imperfectly formed, probably owing to the fact that the vessels are in reality stretched veins, the testis pulling them out in its so-called descent. There is little or no external support in any portion of the length of the vessels. The *vis a tergo* is slight, owing to the great length and small calibre of the spermatic artery, in comparison with the large

sectional area of the veins. Moreover, the veins are tortuous, and have many inter-communications, and there is possibly some pressure exercised upon them by the action of the muscular fibres forming the roof of the inguinal canal.

The greater liability of the veins of the left side to be affected is often attributed to the following anatomical reasons. The entrance of the left spermatic vein into the left renal vein at a right angle, and the rush of blood from the kidney across the mouth of the spermatic vein are supposed to produce an obstruction to the upflow of the spermatic blood. This is distinctly questionable, for this cross current may act in the opposite way, namely, to create a species of suction. The left testis usually hangs lower than the right, and the left spermatic vein terminates higher than its fellow, but the difference in length between two venous channels is not sufficiently great to make it a marked predisposing cause of left varicosity. The left vein has the last portion of the sigmoid flexure in front of it, and in cases of severe constipation it may be that pressure is brought to bear on the vein by hard scybalous masses.

The true reasons probably for the frequency of varicocele on the left side are that the veins in the left cord are usually larger than those in the right, and there appears to be a persistence of numerous fœtal veins on the left, veins which disappear early in life on the right side.

In exposing the spermatic veins for their ligature, for the relief of varicocele, the vas closely accompanied by its artery and veins will be readily demonstrated lying deep to the spermatic vessels in the posterior part of the cord. The spermatic artery cannot as a rule be distinguished, and is probably usually ligated with the veins.

After ligature of the spermatic veins the blood from the testicle is chiefly returned through the deferential veins, passing eventually into one of the vesical plexuses.

## CHAPTER XIII.

### DISEASES OF THE LYMPHATIC SYSTEM.

#### LYMPHATIC VESSELS.

**Lymphangitis.**—It is only in the superficial lymphatic vessels of the subcutaneous tissue that evidence of inflammation can be discerned by the eye. These when inflamed appear as red streaks owing to the peri-lymphangitis which accompanies the affection. In some instances the distribution of the lymphatic vessels is thus clearly marked out, and the skin then becomes mapped with a fine network of reddened lymphatics. Owing to their proximity to the surface, an induration caused by the inflammatory products can occasionally be readily palpated, and is very characteristic. Because of the same anatomical relation, lymphorrhœa is seen from the ulcerations and fissures of portions of skin affected with filarial and other obstruction of the lymphatics.

The deep lymphatic vessels of the limbs commence in the bones, periosteum, ligaments, muscles and inter-muscular connective tissue, altogether beneath the deep fascia. It is on this account that inflammation of these vessels seldom gives evidence of its existence by signs superficial to the deep fascia, as is well observed in the early stages of acute infective periostitis.

The deep lymphatics within the abdomen, especially those running between the layers of the mesentery and omenta, are but little supported, and when there is any obstruction to the onward flow of lymph in them easily part with their contents to form an effusion in the peritoneal cavity, in the form of chylous ascites.

Where lymphatics lie in loose connective tissue, as for example

in that of the eyelids, inflammation leads to very rapid infiltration and very great swelling of the part.

**Injuries to lymphatic trunks.**—The thoracic duct rises well into the neck on the left side, arching over the apex of the pleural sac and the first part of the left subclavian artery. It is here exposed to injury most frequently from operative procedures at the lower part of the left posterior triangle of the neck. In a fair proportion of cases the duct enters by more than one terminal vessel into the junction of the left internal jugular and left subclavian veins, and it is possible, therefore, for a wound to involve only one division and thus cause but little trouble. In the thorax the duct lies along the front of the bodies of the lower seven thoracic vertebræ and then passes to the left side of the upper five bodies. Here it has numerous communications with the azygos veins, which is another reason why injury to the main trunk in the neck is sometimes followed by a minimum of inconvenience. Fracture of one or more thoracic vertebræ may lead to damage to the duct. Injury to the receptaculum chyli, as it lies upon the front of first and second lumbar vertebræ, may occur during operations at this level of the abdomen, or from gunshot-wounds.

#### LYMPHATIC GLANDS.

All lymphatic vessels either pass to or proceed from lymphatic glands. These structures, therefore, filter lymph received by the afferent vessels and transmit it afterwards through the efferent vessels to the vascular system. Hence the great liability for lymphatic nodes to suffer from the entrance and arrest of improper substances circulating in the lymph stream.

**Lymphadenitis**—*The Upper Extremity.*—The fingers and hand are provided with a very intricate and delicate reticulum of lymphatic capillaries, so that even a pin prick must necessarily open up many lymphatic spaces. Infection, therefore, of the superficial lymphatics is very common, and resulting lymphadenitis is extremely frequent. The lymph is conveyed from the palm and dorsum of the hand by vessels which correspond

fairly with the superficial veins. Those accompanying the ulnar vein terminate in the supratrochlear gland, those with the median in the antecubital glands, if they are present, while most of those following the radial vein ascend to the axillary nodes, except a few which, running with the cephalic vein, terminate in the deep infraclavicular glands. It will thus be evident that infection of the inner side of hand is apt to lead to lymphadenitis of the supratrochlear gland, and suppuration here frequently calls for treatment. Lymphangitis of the median set of lymphatics should involve the antecubital glands, but suppuration in front of the elbow joint is uncommon. Infection of any portion of the rest of the skin of the upper extremity may lead to adenitis of the outer set of the axillary lymphatic nodes. The supratrochlear gland cannot be felt normally, but when it is enlarged it may be readily palpated, but should not be mistaken for the ulnar nerve, in front of which it lies. It can be easily distinguished by its oval shape, and by its mobility in all directions, the nerve on the other hand being elongated as a cord, and only moving from side to side. Symmetrical enlargement of the supratrochlear glands is often confirmatory evidence of constitutional syphilis.

*The Lower Extremity.*—The same intricacy of the peripheral lymphatic capillaries is seen in the lower extremity as in the upper.

The lymph is conveyed from the foot by vessels which correspond fairly to the superficial veins. Thus the short saphenous vein is accompanied by lymphatic vessels passing from the heel and back of the leg, and entering a gland close beneath the deep fascia at the termination of the vein in the popliteal space. This node may be the seat of inflammation in cases of blistered heel. There are also some four or five glands deeply placed along the popliteal vessels which may become secondarily involved in septic conditions. Infections occurring on the toes, the dorsum of the foot, the front and inner side of the leg, and the whole of the thigh are apt to lead to lymphadenitis in the groin, where there are situated two sets of lymph nodes. One, the vertical set, near the saphenous opening, lying above the deep

fascia, is that which is primarily concerned with the lower extremity, and is affected in inflammations of the regions just mentioned. They also receive lymph from the perineum, and the inner portion of the buttock. The other, the horizontal set, lying superficial to the aponeurosis of the external oblique muscle above Poupart's ligament, is that which receives lymph from the following parts—the inner glands from the external genitals of both sexes, including all those structures lying superficial to the symphysis pubis and the deep perineal fascia (thus it is here that the common bubo resulting from venereal disease is seen), the middle glands from the lower half of the abdominal wall, and the outer glands from the buttock.

*Cervical Region.*—Perhaps no part of the body is more prone to be the seat of lymphadenitis than the neck. There are numerous sets of cervical glands, but it must be confessed that their arrangement and the number of nodes in each is by no means regular or constant. Still, certain groups can be made out, and a knowledge of them is of great practical utility. Reviewing these from above downwards, they may be given as anterior auricular, posterior auricular, occipital, submandibular, sub-mental, superficial cervical, deep cervical, retro-pharyngeal and supra-clavicular. When a patient is seen with enlarged glands in the neck, the region where these glands are placed should be an indication as to the source from which the irritation causing their increase in size has been derived. On the other hand when a patient presents himself in whom there is some disease of the lips, the tongue, the scalp, &c., which may lead to implication of the lymphatic glands, it is needful to know which set to examine to determine whether or not such involvement exists. Thus it is convenient to review here the groups of glands and see where they derive their lymph, and then under the various diseases to state to which set the lymph passes.

The anterior auricular glands, situated in front of the ear, and upon and within the substance of the parotid gland, derive lymph from the frontal and the parietal portions of the scalp, and the upper part of the face. The posterior auricular, placed behind

the pinna of the ear, receive lymph from the auricle, and the posterior part of the parietal region.

Some of the submandibular placed near the middle line are called sub-mental and receive lymph from the lower lip, the chin, and the floor of the mouth. The lymphatic glands placed in the region of submandibular salivary gland are concerned with the tongue, cheek, floor of the mouth and the mandible.

The deep cervical glands placed along the carotid sheath derive lymph from the tongue, pharynx, tonsils, larynx, trachea and œsophagus.

The retro-pharyngeal, present in children, are atrophic in the adult. They drain lymph from the cervical portion of the vertebral column, and the posterior aspect of the pharynx proper.

The supra-clavicular glands in the lower part of the posterior triangle of the neck derive secondarily, but sometimes primarily, lymph from the anterior thoracic wall and the mammary gland. Those on the left side may be involved in secondary deposit in cases of malignant disease of the abdomen and thorax through their connexion with the thoracic duct.



## CHAPTER XIV.

### DISEASES OF THE DUCTLESS GLANDS.

#### THE THYROID GLAND.

**Enlargements of the thyroid gland** are usually spoken of as goitres. These may be of various kinds, such as parenchymatous, cystic, adenomatous or malignant. The goitre of Graves's disease is sometimes termed vascular, but this does not accurately describe the condition. From whatever cause the gland is enlarged, its anatomical relations have important bearings on the position and movements of the tumour and the symptoms it may produce. The almond-shaped lateral lobes with their isthmus give a characteristic horse-shoe shape to the gland which is retained in all instances of uniform enlargement. The right lobe is larger than the left, and this normal disproportion, being maintained in general enlargement, may lead to an erroneous impression that the right lobe is more increased in size than the left. A middle lobe may sometimes be detected, passing upwards on the front of the larynx—this is the pyramidal lobe which is usually attached to the hyoid bone by the fibrous remnants of the thyro-glossal duct. The well-recognised clinical facts that thyroid tumours follow the up and down movement of the larynx during deglutition, and also accompany it when moved laterally, are accounted for by the investment of the gland in a capsule which is derived from the deep cervical fascia and closely attached to the larynx. This fascial capsule is fixed in front to the anterior arch of the cricoid cartilage and also to the lower borders of the alæ of the thyroid cartilage. That part of the capsule which invests the inner and back parts of the lateral lobes also sends fibrous prolongations to the sides of the cricoid cartilage. These prolongations are known as the suspensory

ligaments of the gland, and when it is enlarged may appear as strong supporting cords. The recurrent laryngeal nerves lie in close contact with the outer or posterior surfaces of these ligaments. The arrangement of the capsule on the posterior aspect of the gland is important. As the fibrous coating is traced from the convex surface of each lateral lobe backwards it will be found to split into two layers, one of which remains in close contact with the gland, completing its posterior investment, whilst the other passes to the posterior surface of the pharynx and œsophagus, imprisoning those tubes together with the trachea in the hollow at the back of the gland. This part of the capsule, being dense and firm, forms a conspicuous object during operation, and if unwittingly followed backwards under the belief that it is the immediate posterior investment of the gland, may lead to the infliction of wounds on the œsophagus or trachea which are enclosed by it. Operations on the thyroid gland necessitate the opening up of the planes of cervical fascia, so that there is a liability to the occurrence of cervical and mediastinal cellulitis as complications.

Of the muscles which cover the thyroid gland, the sterno-mastoids are most superficial. When the gland is enlarged, these muscles are usually displaced outwards, so that the thyroid tumour appears in the interval between them. Occasionally the tumour presents at the posterior border of the sterno-mastoid displacing the muscle inwards. The sterno-hyoid and the sterno-thyroid muscles become flattened over the enlarged gland and may occupy grooves on its surface. The tension exerted on the omo-hyoid which also crosses the gland on its way to the hyoid bone, may cause the posterior belly of the muscle to form a prominent cord-like projection across the base of the posterior triangle of the neck.

The vascular relations of the gland are important. There is a rich plexus of veins immediately inside the capsule, and these it is inadvisable to wound. On each side the gland is in immediate relation with the carotid artery and internal jugular vein. Enlargement of the gland usually tends to displace these structures outwards, but there is a great difference in the degree of

THYROID GLAND.

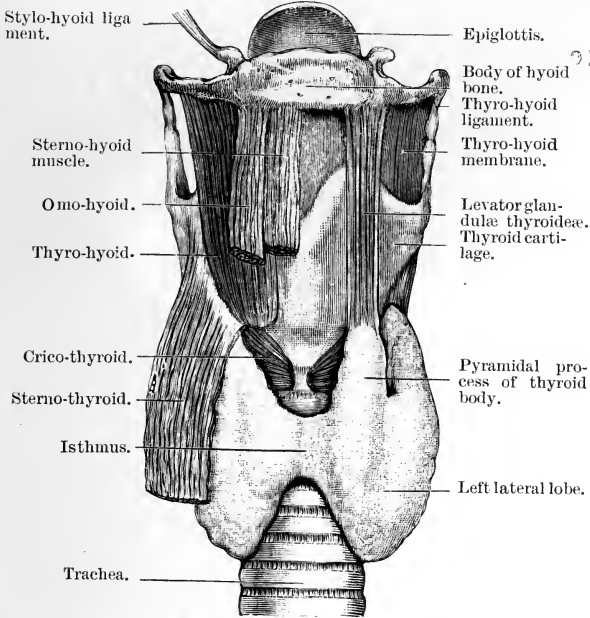


FIG. 38.—Thyroid body, with middle lobe and levator muscle. (From Morris's Anatomy.)

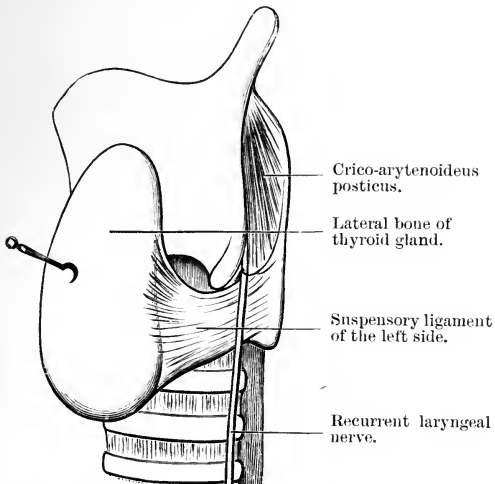


FIG. 39.—The suspensory ligaments of the thyroid body. (From Morris's Anatomy, after Berry.)



displacement of the artery and the vein. The vein is closely connected to the gland by its thyroid tributaries, and the traction on these to a great degree anchors the vein, causing it to be closely applied to the surface of the lateral lobe or even flattened over it. The artery is displaced without much restraint, for the superior thyroid arteries are tortuous and long. The same description applies to the inferior thyroid arteries which arise from the subclavian artery.

The superior thyroid arteries approach the apices of the lateral lobes whilst the inferior vessels enter the hilum which lies at the inner and back part of the lateral lobe close to the recurrent laryngeal nerves. It is advisable to ligature both the veins and the arteries outside the capsule, since by so doing wounds of the venous plexus are avoided, and the arteries are ligatured where their coats are thick, for they become attenuated after piercing the capsule. In addition, in the case of the inferior arteries there is less risk of damage to the inferior laryngeal nerves.

In addition to the internal jugular, the anterior and external jugular veins may be subjected to pressure and considerable engorgement result. Occasionally also the right and left innominate and the subclavian veins are compressed at the root of the neck.

It is often difficult to distinguish pulsation of the thyroid gland itself from pulsation communicated to it by the carotid trunks. The pulsation of the enlarged superior thyroid trunks can sometimes be recognised also. Venous and arterial murmurs may arise in the compressed veins and arteries.

The posterior border of each lateral lobe of the thyroid gland is in contact with the spine and prevertebral muscles, and may become fixed to these structures as the result of malignant infiltration. Under similar circumstances the gland may envelop the carotid artery without displacing it.

The posterior aspect of the gland is deeply concave, the whole being moulded on the trachea and œsophagus. The recurrent laryngeal nerves which lie in the sulcus between the trachea and œsophagus, are included in the bay or hollow thus formed on

the posterior surface of the gland. The left lateral lobe is normally in contact with a small portion of the front of the œsophagus where that tube deviates somewhat to the left at the root of the neck, whilst the upper extremities of the lateral lobes may actually embrace portions of the larynx and pharynx. Pressure on the œsophagus and dysphagia may thus result from thyroid swellings.

The pressure exerted by the enlarged gland on the trachea is of serious importance and may cause death. The trachea is usually compressed laterally and not often antero-posteriorly. The lateral compression is no doubt aided by the pressure exerted by the sterno-mastoid and infra-hyoidean muscles which cross the lateral lobes. The lateral compression is often associated with a certain amount of lateral twisting of the trachea on its long axis, and in unilateral enlargements of the gland the air tube may be displaced towards the opposite side of the neck. Sub-sternal goitres, being unable to enlarge forwards, compress the trachea antero-posteriorly.

Signs of pressure on neighbouring nerve trunks may be present. Alteration of the voice and various forms of laryngeal paralysis may result from pressure on the recurrent nerves. Contracted pupil, enophthalmos and narrowing of the palpebral fissure, indicate pressure on the cervical sympathetic which lies at the back of the carotid sheath. Numbness and tingling in the neck or arm may be produced by pressure on the branches of the cervical or brachial plexus. Irregularity of the heart is attributed to vagus compression.

The close relation of the cervical lymph glands to the lateral lobes of the thyroid gland gives rise to some difficulty in distinguishing malignant or tuberculous enlargements of the lymph glands in this position from actual disease of the thyroid.

The parathyroid bodies are difficult to recognise. Two of them may sometimes be found amongst the terminal branches of the inferior thyroid arteries, and the other two are usually situated at the lower extremities of the lateral lobes.

**Myxœdema** and **cretinism** are usually associated with atrophy

of the thyroid gland. In those cases where the gland is enlarged, its function is assumed to be in abeyance. It is not easy to form a definite opinion as to the presence of thyroid atrophy by an examination of the neck. The isthmus of the gland usually crosses the second, third, and perhaps the fourth, rings of the trachea. If these rings can be distinctly felt atrophy may be assumed, but in some persons the isthmus is congenitally absent, and in others, owing to its thick coverings, the trachea cannot be satisfactorily palpated. The lateral lobes, as a rule cannot be felt at all.

The general increase of bulk of the body in myxœdema is due to changes in the skin and subcutaneous tissues. Where the subcutaneous connective tissue is lax and abundant, the swellings are most apparent. Hence swelling may be found in the eyelids, face, submandibular region, the lower parts of the posterior triangles of the neck, and the backs of the hands and feet. Where the skin is firmly bound to the subjacent fascial structures, as in the palms, soles and scalp, the swelling is insignificant.

The swelling of the tissues of the limbs, by destroying the elasticity of the skin around the joints, interferes with the flexion movements of the elbows, wrists, fingers, and thighs. The finer movements of the hand suffer in particular. The mucous membranes as well as the skin are affected. The lips become stiff, swollen and everted, the buccal mucous membrane projects between the upper and lower teeth and is indented. The gums are swollen, and the lax tissues of the soft palate become infiltrated and interfere with the movements of deglutition and articulation. The tissues of the vulva and vagina may be similarly affected.

The epidermal appendages naturally suffer with the skin. The hair follicles, the sweat and sebaceous glands become infiltrated and destroyed. The nails may be cracked and furrowed, and the teeth fall out. It is possible that the peripheral sense organs are also involved.

The supraclavicular swellings of cretinism are by no means

constant, nor are they restricted to this variety of the disease. They may be met with in myxœdema and in association with some goitres, and are due to prominence of the supraclavicular fatty tissue. Their occurrence is favoured by the absence of superficial muscles and other resistant coverings in the situations where they are found.

#### THE THYMUS GLAND.

**Enlargement of the thymus** is met with in very diverse conditions. The chief of these are Graves's disease, rickets, myxœdema, acromegaly, leukæmia, and lymphadenoma. In the condition known as lymphatism, enlargement of the gland is associated with increase in the lymphoid tissue of the pharynx, bowel, and other parts of the body. The thymus may also be the seat of inflammation, tuberculosis, and new growths. It is important to bear in mind that wide variations in the size of the gland occur. The older estimates of its weight appear to be excessive. The average weight from birth up to the age of two years is from seven to ten grammes (quarter to half an ounce). After two, the gland remains stationary for some time, but a rapid diminution, accompanied by degenerative changes, occurs at puberty. Remnants of the gland are present to an advanced age.

Enlargement of the thymus is difficult to detect during life owing to the position of the gland behind the manubrium and upper part of the gladiolus. It may, however, give rise to the following signs: Fulness at the root of the neck, venous engorgement of the neck, dulness behind the sternum, cardiac irregularity or dilatation, and dyspncea. The fulness at the root of the neck is due to the fact that both lobes of the gland are prolonged for a short distance upwards along the sides of the trachea; indeed, the pointed extremity of each lateral lobe can be traced upwards in the fully-developed fœtus, under the lateral lobes of the thyroid gland to the thyro-hyoid membrane. The venous engorgement is explained by the relations of the left innominate





THYMUS GLAND.

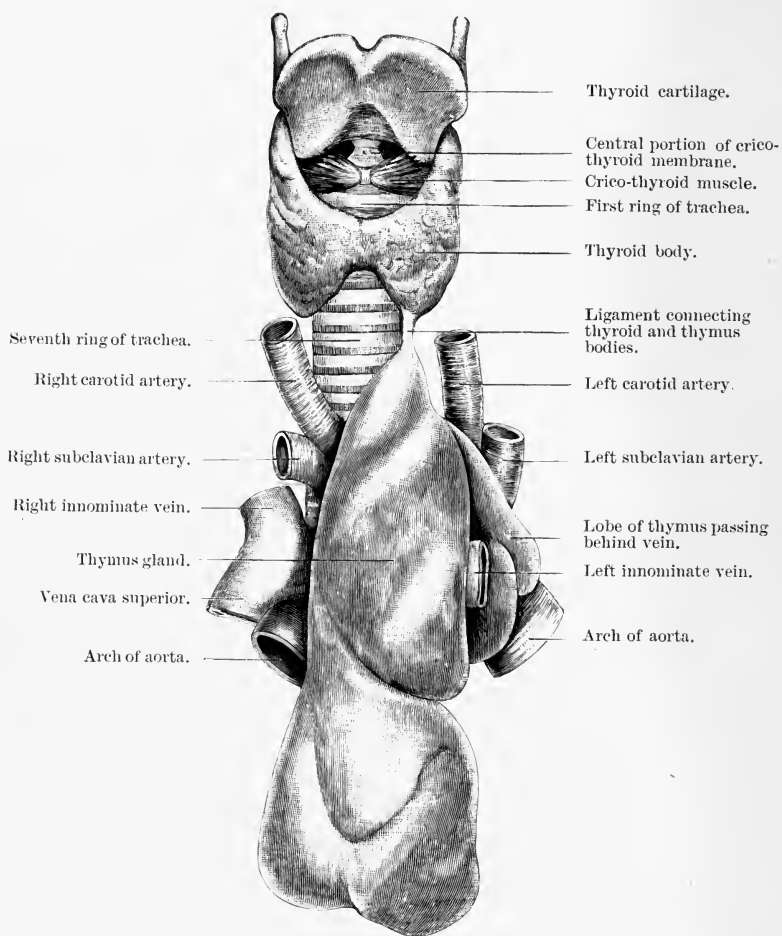


FIG. 40.—Thymus gland in a child at the age of two years showing some important relations. (From Morris's Anatomy.)

vein and superior vena cava to the back of the gland, where they lie in grooves. Dulness over the manubrium is due to the presence of the glandular mass immediately behind the sternum, in the fore parts of the superior and anterior mediastina. The gland reaches as low as the level of the fourth costal cartilage, a rib below the position where the upper limit of cardiac dulness may be expected in childhood. The cardiac hypertrophy which has been found in some instances has been attributed to compression of the ascending aorta or pulmonary artery. Compression of the vagi has been held responsible for certain irregularities of the heart's action. Dyspnoea is the most important symptom of all. There is great diversity of opinion as to the possibility of suffocation as the result of pressure by an enlarged thymus. It is urged that many of the sudden deaths which are attributed to suffocation are really due to syncope. The anatomical arrangement certainly seems to favour the possibility of tracheal compression when the gland is large. The space between the back of the manubrium and the front of the vertebral column measures little more than two centimeters (about seven-tenths of an inch), and is occupied by the œsophagus, trachea, great vessels and important nerves. Even if the size of the gland be insufficient to cause much compression of the trachea with the head in the normal position, if the neck be suddenly bent backwards the thymus and surrounding structures are further dragged up into the narrow thoracic aperture, and the trachea may be completely occluded. It is possible that this sequence of events might occur during life, owing to weakness of the flexor muscles of the neck, and the softness of the infant trachea.

Tuberculosis of the thymus must be carefully distinguished from caseation of the superior mediastinal glands, which are in very close relation with the thymus. Careful dissection is requisite to define the limits of the two structures. The tendency of the lobes of the gland to break down into a fluid consisting of lymphocytes has often given rise to an erroneous diagnosis of suppuration. The investment of the gland by the anterior

mediastinal fascia explains those instances in which inflammation has spread to the gland as a sequel of pleurisy, pericarditis, retropharyngeal and other abscesses in the neck, &c.

The thymus gland may be the site of epithelial, sarcomatous, lymphatic, fatty or other tumours. In this connection it is interesting to remember that it is developed as an epithelial structure, derived from the dorsal part of the third visceral cleft, represented in the adult by the space in front and on each side of the epiglottis. Later the gland is transformed into lymphoid tissue, whether by local growth or invasion from without is doubtful. Epithelial remnants remain in the form of the concentric corpuscles. The surrounding mesoblast supplies the capsule and the connective tissue stroma of the gland. Hence there are elements from which epithelial, connective tissue, or lymphoid neoplasms may arise.

#### THE SPLEEN.

**Injuries of the spleen** are not very common. The depth at which the organ lies in the left hypochondriac and adjacent part of the epigastric regions affords it protection. When enlarged the spleen becomes much more superficial anteriorly, and is liable to rupture by direct violence or vigorous muscular contraction. Normally the spleen lies under cover of the ninth, tenth, and eleventh ribs of the left side. Its long axis corresponds with the posterior part of the tenth rib, its posterior extremity lies an inch and a half away from the spinal column, and its anterior border reaches the mid-axillary line. It may be wounded when the ribs under which it lies are fractured and their broken ends driven inwards. Under such circumstances the diaphragm is penetrated, and the left lung and pleural sac, which overlie the spleen, may be damaged. The elasticity of the ribs and the yielding nature of the abdominal wall may allow the spleen to be severely crushed against the spinal column and lacerated without the occurrence of fracture or external bruises. The liver is often lacerated at the same time as the spleen. The

great vascularity of the spleen, due to the presence of numerous thin-walled blood sinuses, explains the profuse and often fatal hæmorrhage which results from ruptures and penetrating wounds. The anatomical relations of the organ sufficiently explain why penetrating wounds are often complicated by injuries to the pleura, lung, diaphragm, stomach, colon or

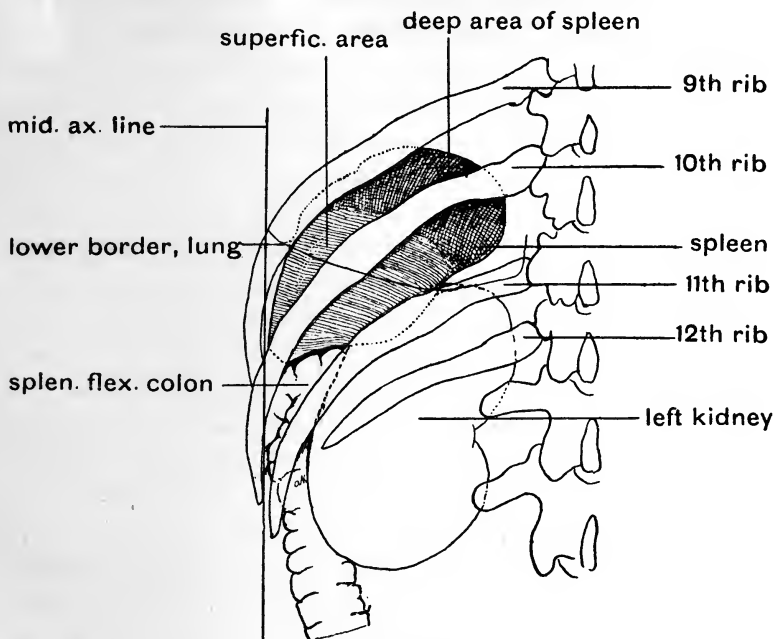


FIG. 41.—THE POSITION AND RELATIONSHIPS OF THE SPLEEN. THE DEEP AND SUPERFICIAL AREAS OF ITS DIAPHRAGMATIC SURFACE ARE EXPOSED. (Hughes and Keith.)

kidney. The greater sac of the peritoneum is of necessity opened by such injuries.

**Enlargement of the spleen** varies much in degree, and is met with in very diverse conditions. If the increase in size is very great, inspection may reveal the outline of part of the organ below the left costal margin, extending in the direction of the umbilicus. An enlarged spleen takes this direction by virtue of its surroundings, for its convex surface lies against the resistant

ribs, its renal surface is well supported by the left kidney, and its anterior extremity rests on the phrenico-colic fold. Consequently the organ is enlarged in the direction of least resistance, which is downwards, forwards, and inwards. If the phrenico-colic fold is unusually well developed its resistance may cause the main enlargement to take place in an upward direction, whilst if the fold is insignificant or yields to pressure, the spleen may drop directly downwards instead of being directed obliquely forwards towards the umbilicus. By palpation the margin of an enlarged spleen may be felt below the left costal margin. In infants the spleen is felt much farther back than in adults, probably on account of the greater mass of the liver in childhood. The presence of the notches serves to distinguish the spleen from an enlarged kidney. The latter, with which splenic enlargements are often mistaken, possess a more rounded outline, and is not notched. Another point of distinction is the fact that a large kidney must first fill the loin, this being the position in which it lies, whilst a large spleen presents itself anteriorly, for the reasons mentioned above. But a very large spleen may ultimately encroach upon the loin, and an enlarged kidney may ultimately present anteriorly, just above the umbilicus. The spleen lies in close contact with the diaphragm, and so shares in the respiratory movements, unless fixed by adhesions. The kidney is also in contact with the diaphragm, and so descends slightly on inspiration; this descent is exaggerated when the organ is enlarged.

The intervention of the lung above, the presence of the stomach and colon in the immediate vicinity, and the thickness of the muscular wall of the loin, much diminish the value of percussion as a means of determining splenic enlargement.

A large spleen may exercise pressure on the diaphragm, and so hamper the respiratory movements and induce partial collapse of the base of the left lung. It may also exercise pressure on the adjacent stomach or colon. Conversely collections of air or fluid in the left pleural sac, emphysema of the lung, or intra-thoracic tumours may cause depression of the spleen, and a sub-diaphragmatic abscess may have the same effect.

**Infarctions of the spleen** may be due to embolism or thrombosis of the splenic artery. The terminal branches of this artery have only a capillary connection with each other. When they are occluded a conical area of splenic tissue undergoes necrosis. The base of the cone is applied to the splenic capsule, and the apex is directed towards the hilus. A thin stratum next the capsule sometimes escapes necrosis, being nourished by capsular branches. Splenic infarcts are usually white, but occasionally they are red with blood which regurgitates into them from vessels in their neighbourhood.

**Passive congestion of the spleen** is due to increased pressure in the portal area. The splenic vein is a tributary of the portal trunk, and when this trunk is obstructed in any way enlargement of the spleen may result. The intimate connexion of the spleen with the portal system explains the rapid diminution in the size of splenic tumours, which may be brought about as the result of diarrhoea.

A **wandering spleen** is uncommon. The organ is occasionally found in regions very remote from its normal situation. It may become fixed in its new positions and cause considerable difficulty in diagnosis. The condition is sometimes a part of general visceral prolapse. The wandering spleen has a pedicle formed by the gastro-splenic omentum and lieno-renal ligaments, with the included vessels. Occasionally the tail of the pancreas retains its connexion with the organ, the whole gland becoming elongated and twisted. The pedicle of a wandering spleen may become twisted and produce acute symptoms. The splenic vessels in their normal condition are well supported at the back of the abdomen, but when the spleen is loose or enlarged they are subject to traction and may show signs of endarteritis, or small aneurysms of the splenic artery may occur. Communication between the artery and vein has been described, producing the characteristic murmur of arterio-venous aneurysm. Thrombosis also results occasionally.

**Accessory spleens** are very common in the gastro-splenic omentum and other peritoneal folds connected with the organ.

They may undergo compensatory enlargement when the operation of splenectomy has been performed. They sometimes form the starting point of tumours, and have been found embedded in the tail of the pancreas.

**Abscess of the spleen** is usually of embolic origin, but may invade the viscus from without. Primary abscesses may rupture into the left subphrenic space in which the spleen lies, or may discharge into the stomach, colon, or left pleural sac. An ulcer of the stomach may become adherent to the gastric surface of the spleen and give rise to a local abscess.

#### THE SUPRARENAL BODIES.

**Addison's disease** is nearly always due to tuberculosis of the suprarenal bodies. The infection is usually secondary to a lesion elsewhere and conveyed to the glands by the blood. The vascular supply to the suprarenals is very free, there being no fewer than three capsular arteries supplied to each. Since both bodies lie in contact with the spinal column, from which they are only separated by the crura of the diaphragm, it is not surprising that tuberculous caries of the lower dorsal and upper lumbar spine sometimes invades them by contiguity.

The semilunar ganglia and the solar plexus of the sympathetic are intimately connected with the suprarenals and may be involved by the spread of inflammation from them. At one time the symptoms of Addison's disease were exclusively attributed to implication of these nervous structures.

The receptaculum chyli is separated from the right suprarenal by the crus of the diaphragm, but there is no distinct evidence of obstruction to the lymph flow in Addison's disease.

A solitary thin-walled vein emerges from each capsule. That from the right passes directly into the inferior vena cava; that from the left pours its contents into the left renal vein. The efferent lymphatics accompany the veins and terminate in glands adjacent to the aorta. A few lymphatics perforate the crura of the diaphragm ending in small glands between the crura and



the spinal column. It is possible that obstruction of the venous and lymphatic trunks may produce the characteristic symptoms in those cases where disease of the suprarenals is said to be absent. Tuberculous peritonitis sometimes complicates Addison's disease, and may in some instances spread direct from the capsules. A part of the right suprarenal is in contact with the greater sac and a part of the left with the lesser sac. Tuberculosis sometimes spreads directly from the right capsule to that part of the liver which is in direct contact with it.

The small yellow fatty collections which normally occur in the cortex of the suprarenal bodies must not be mistaken for tubercles.

The cutaneous pigmentation of Addison's disease is due to infiltration of the cells in the Malpighian layer. In the mucous membranes the pigment is said to be more superficial. Those mucous membranes which from their position are subject to friction or irritation may be the site of the pigmentary deposits. A bluish stain may be seen where the margins of the lips come into contact. Brown smears occur on the inner sides of the cheek and lips where these touch the teeth. The sides and tips of the tongue may be pigmented for the same reason. Pigmentation may sometimes occur in the mucous membrane of the vulva and vagina. Pigmentation of the skin occurs chiefly in exposed parts, such as the face and backs of the hands. It is also evident in parts which are naturally pigmented and those which are exposed to friction, pressure or irritation. The natural pigmentation of the anterior axillary folds, areolæ of the nipples, lower part of the abdomen, groins, penis, scrotum and perineum may be exaggerated. The friction of yokes, waistbands, garters and corsets may determine deposits of pigment, and the same may be seen in parts which have been irritated by blisters. Scars as a rule escape.

**Accessory suprarenal bodies** of small size are not uncommon. Occasionally they are found embedded in the cortex of the kidney. They have also been recognised in the broad ligaments and spermatic cord, having been carried down from the neighbourhood of the Wolffian body by the descent of the ovary or testis.

**Primary tumours** of the suprarenal bodies may be carcinomatous, sarcomatous and possibly gliomatous. The central part of the suprarenal is derived from an ingrowth of the abdominal sympathetic, and this may account for the presence of gliomatous elements. The cortical part is developed within the Wolffian ridge. The epithelial and connective tissue elements of this may give origin to the other varieties of new growth mentioned.

Malignant tumours of the suprarenals lie high in the abdomen and may from their position simulate tumours of the liver, kidney or pancreas. The tumours are adjacent to the inferior vena cava, the suprarenal and the renal veins, which they may invade. Extension may occur from the right suprarenal into the liver. Growths on either side may invade the adjacent kidney, and if they open into its pelvis may be a cause of hæmaturia.

#### THE PITUITARY BODY.

**Acromegaly**, a condition characterised by overgrowth of the tissues of the extremities, and allied to gigantism, is generally associated with enlargement of the pituitary body. The enlargement may be simple or malignant. As a result, the sella turcica is enlarged and in part absorbed. The olivary and clinoid processes may disappear. The sphenoidal air cells may be opened up, or the base of the fossa entirely destroyed. The cavernous sinus and Gasserian ganglion have been invaded. Characteristic alterations in the visual fields arise from pressure on the optic commissure, which lies in front of the stalk of the pituitary body. As a result bitemporal hemianopia may be met with, or a more general contraction of the fields may occur.

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OPENINGS INTO THE NASAL CAVITY.

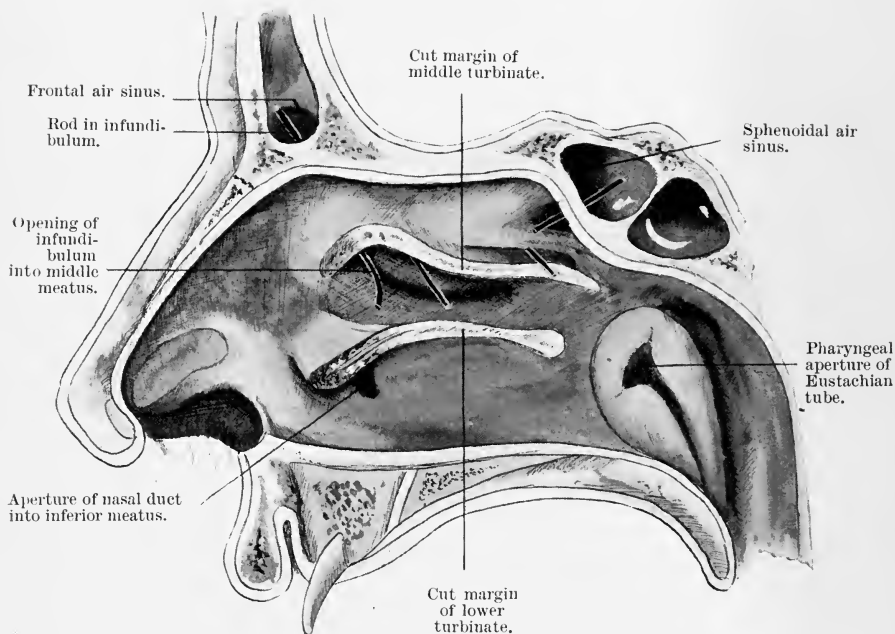


FIG. 42.--Dissection to show openings into the nasal cavity. (After Edward Taylor.)

## CHAPTER XV.

### DISEASES OF THE RESPIRATORY SYSTEM.

#### NOSE AND NASO-PHARYNX.

**Rhinitis.**—The air which is inspired through the anterior nares, across the nasal cavities, and by the posterior nares into the naso-pharynx, is passed over a large surface covered with mucous membrane. This membrane secretes mucus upon which should be deposited all solid particles, including bacteria, floating in the air; hence the liability of the upper part of the respiratory tract to infection and inflammation. This power of the nasal mucous membrane to catch undesirable material is very well demonstrated in towns during a fog.

The many accessory air cavities communicating with the nose may one or all become the site of infection by extension. The infundibulum, ending in the middle meatus, passes upwards into the frontal sinus, and the frontal headache so frequently present with a "cold in the head" indicates inflammation of the mucous membrane lining this air cavity. From the middle meatus, also, micro-organisms may pass through the small aperture into the antrum of Highmore, and may occasionally give rise to an empyema therein. The posterior ethmoidal and sphenoidal sinuses are sometimes infected through their openings into the superior meatus.

It is worthy of notice, also, that infection may pass directly along the branches of the olfactory nerves, through the cribriform plate of the ethmoid bone, into the interior of the cranium, there to set up a septic meningitis. In like manner the thin bony walls of the ethmoidal and sphenoidal sinuses may be eroded, and the overlying dura mater attacked.

The close proximity of the cranial cavity to the nasal fossæ

explains the ready manner in which a sharp pointed instrument introduced into the nostril may pass through the thin bone forming the base of the skull in this region, and carry with it septic material.

**Foreign Bodies in the Nostril.**—The difficulty of extracting foreign bodies introduced into the nose arises from the following anatomical facts:—First, the irregularity of the walls of the cavity; secondly, its many recesses; and thirdly, the swelling of its mucous membrane as a result of the irritation and inflammation produced by the body. For these reasons it is sometimes more easy to push the imprisoned substance backwards into the pharynx than to draw it forwards through the anterior nares.

**Polypi.**—The ordinary mucous polypi, or cedematous fibromata, are usually the result of chronic inflammatory changes in the mucous membrane of the ethmoidal region, and spring from that portion of the membrane over the superior and middle turbinate bones. Their presence tends to block the nasal cavity, and they enlarge in the line of least resistance—that is, downwards and forwards—until they may even protrude through the anterior nares.

**Septal Deviations.**—The bony and cartilaginous septum of the nose is hardly if ever absolutely in the middle line, but it is only when the degree of divergence from the perpendicular plane is so great as to cause a considerably increased space in one nasal cavity at the expense of the other that any real significance in the condition arises. The obstruction to the passage of air on the side to which the septum is deviated may be so great that it is well nigh impossible to pass a probe along it, and hence an operation becomes necessary.

**Adenoid Growths of the Naso-pharynx.**—The uppermost part of the ring of adenoid tissue about the naso-pharynx and the fauces is very frequently the site of hypertrophy, in the form of pedunculated masses. These constitute the so-called “adenoid growths.” The pharyngeal tonsil, situated in the mucous membrane forming the roof of the naso-pharynx between the two Eustachian orifices, is not infrequently chronically enlarged

and aids in the obstruction of the airway. The position of the funnel-shaped aperture of the Eustachian tubes on the lateral walls of the naso-pharynx explains the frequency of the extension of inflammation to the mucous membrane lining the tubes. Normally the cilia of the epithelium covering the membrane work towards the naso-pharynx, carrying secretions away from the tympanum and preventing the entrance of bacteria into that cavity.

The interference with nasal respiration caused by adenoid vegetations produces typical, and not infrequently serious, deformities. The nostrils and the nasal cavities may continue small and ill-developed from want of full use, and the arch of the palate remains higher than normal, with a tendency to crowding, and frequently misplacement, of the incisor teeth. The thoracic walls, from insufficient expansion, become considerably altered in shape, and exhibit a characteristic circular depression at the junction of the lower and middle thirds from the traction of the diaphragm and atmospheric pressure, whilst the upper part of the cavity seems to be abnormally enlarged.

#### LARYNX.

**Congenital malformations of the larynx** are rare, but the exaggeration of the normal peculiarities of the infantile larynx is the basis of the condition known as congenital laryngeal stridor. In these cases the epiglottis is much folded on itself vertically and the aryteno-epiglottidean folds are closely approximated. During inspiration the sides of the laryngeal aperture tend to be sucked inwards, and obstruction with a stridulous crow results.

**Catarrhal Laryngitis.**—The larynx, owing to its proximity to the nose, mouth and pharynx, is exposed to air-borne infection, to the action of irritant vapours, and to injury during the swallowing of corrosive fluids or rough solid bodies. The continuity of its mucous membrane with that of the fauces explains the spread of inflammation from the nose or throat. The laryngeal mucous

membrane being freely supplied with sensory nerves, laryngeal spasm may be the result of catarrhal inflammation.

**Oedema of the larynx** may be inflammatory, when it is either infectious or traumatic, or a passive œdema of local or general origin. It is most marked in situations where there is the most submucous tissue, *i.e.*, the aryteno-epiglottidean folds, the false cords, the ventricles, and the laryngeal aspect of the epiglottis. Oedema of the true cords is unknown, there being no loose connective tissues in this region. Oedema may cause considerable obstruction of the upper laryngeal aperture, and this is especially apt to be the case in childhood, since the larynx is then small. The laryngeal opening lies on the anterior wall of the pharynx, consequently when œdema interferes with its proper closure during deglutition, choking may occur, and at the same time pain may be experienced if the swelling is of inflammatory nature.

**Foreign Bodies in the Larynx.**—Many large foreign bodies, such as a bolus of food, obstruct the superior aperture of the larynx without entering its cavity. A small foreign body, having passed the upper strait, falls upon the edge of the true vocal cords, thereby causing sudden closure of the glottis. It may thus be immediately ejected. On the other hand, it may be forced into one of the ventricles of the larynx, or when the rima glottidis is opened, it may slip through during inspiration and drop into a bronchus. Sharp bodies, if they reach the interior, are apt to stick in the mucous membrane of the upper half of the larynx.

**Malignant disease of the larynx** usually occurs as squamous-celled carcinoma. The greater part of the laryngeal mucous membrane is covered with columnar ciliated epithelium, but on the true vocal cords the epithelial covering is squamous. Squamous epithelium also lines the vestibule or upper subdivision of the laryngeal cavity, the transition to columnar cells occurring at the widest part of the epiglottis in front, and a line or two above the false cords at the sides. Patches of squamous epithelium are also found on the inner surfaces of the arytenoid cartilages, and at the free borders of the false cords.

New growths of the larynx are usually primary, but may extend



to it from neighbouring parts. Owing to the virtual isolation of the laryngeal lymphatic system, laryngeal neoplasms never arise as metastatic deposits.

From a clinical standpoint carcinomata of the larynx are divided into two classes, extrinsic and intrinsic. The extrinsic varieties may arise on the posterior surface of the cricoid cartilage, in the arytenoid region, the aryteno-epiglottidean folds or on the posterior surface of the epiglottis. Intrinsic carcinomata usually arise from the posterior parts of the true cords, but may spring from the false cords, the ventricles of the larynx, or the subglottic region.

The commonest site of extrinsic carcinoma is the back of the cricoid cartilage, whilst intrinsic cancer, as just mentioned, usually arises from the vocal cords. Both these localities may be considered subject to friction and irritation, the back of the cricoid during deglutition, and the vocal cords during phonation.

The anatomical justification for the classification given above is found in the arrangement of the laryngeal lymphatics. The supraglottic region, the site of extrinsic carcinoma, is provided with a rich lymphatic network which is densest where the mucous membrane is thickest, and is in some degree continuous with the lymphatics of the pharynx and back of the tongue. The lymphatics of the glottic and subglottic regions, positions in which intrinsic carcinomata originate, are more scanty, and in the true cords are, for all practical purposes, non-existent. The subglottic network communicates in some measure with that of the trachea. The lymphatics of one-half of the larynx communicate freely across the posterior median line, but not at all freely anteriorly. Extrinsic cancer, guided by the course of the lymphatics, tends to spread towards the upper parts of the larynx and the pharyngo-laryngeal region. It may perforate the posterior part of the thyro-hyoid membrane near the exit of the lymphatic trunks. Early glandular enlargement is the rule, and the first to suffer is often a small gland on the thyro-hyoid membrane. Extension downwards in the region of the vocal cords is very slow.

Intrinsic cancer, on the other hand, is of slow growth, and glandular infection is rare. The growth tends to spread round to the opposite side or to the subglottic region rather than upwards.

The connexion between the lymphatics of the epiglottis and the base of the tongue affords an explanation of the tendency of cancer of the epiglottis to infect the tongue base.

The lymph glands beneath the sterno-mastoid muscle are those which finally receive the lymphatic of the larynx. These glands communicate below with the supra-clavicular, but not with the mediastinal groups. The efferent lymphatics of the supraglottic area reach the glands by perforation of the lateral part of the thyro-hyoid membrane. Of the subglottic lymphatic trunks, some pierce the thyro-hyoid membrane and others emerge between the cricoid cartilage and the trachea. On their way to the lower glands beneath the sterno-mastoid, the subglottic lymphatics may traverse small glands on the front of the larynx and trachea and along the course of the recurrent laryngeal nerves.

Of the clinical symptoms, hoarseness is easily explained by involvement of the vocal cords. Dysphagia is naturally most marked when the epiglottis or posterior part of the cricoid cartilage is invaded. The radiation of pain to the neck and ear is due to reflection along the superior laryngeal nerve to the auricular branch of the vagus. Laryngeal paralysis is of complex origin, but in some instances may be due to involvement of the motor filaments of the recurrent laryngeal nerve. Laryngeal anæsthesia is due to the implication of the sensory branches of the laryngeal nerves.

## THE BRONCHI AND LUNGS.

### BRONCHIECTASIS.

The bronchi which have a descending course are more likely to become dilated than those which pass upwards, since

secretions are more likely to stagnate in them. The direction of the larger bronchial branches of both upper lobes and of the right middle lobe is outwards, upwards, and forwards, whilst those of the lower lobes of both lungs pass downwards, inwards, and backwards. Consequently it is usual to find **bronchiectasis** limited to the lower and middle portions of the lungs, whilst the upper lobes, and particularly the territory of the vertical apical bronchi, escape. The greater length and narrowness of the bronchi of the left lower lobe may account for the greater frequency of bronchiectasis of the left side.

Since the connective tissue sheaths of the bronchi are continuous with the lobular septa of the lungs, and these septa in turn fuse with the visceral pleural envelope, it is evident that obliteration of the pleural cavity by adhesions will permit the inspiratory movements of the chest to make centrifugal traction on the bronchi. If these tubes are weakened by inflammation the traction may induce dilatation. It appears, however, that pleural adhesions are not at all common in the early stages of bronchiectasis, many cases of which originate in childhood as sequels to the lung inflammations of measles and of whooping cough.

Dilatation of bronchi is, in some instances, due to pressure on the roots of the lungs. The left bronchus being in intimate relation with the œsophagus, the aorta, and the bronchial glands, pressure on this tube is usually due to aneurysm, malignant disease of the œsophagus, or glandular swellings. The right bronchus is in relation with the back of the ascending aorta and the bronchial glands, so it is liable to pressure from an aneurysm or from glandular enlargements. Extreme dilatation of the left auricle in mitral stenosis has been known to cause compression of the left bronchus.

A **foreign body** lodged in a bronchus is sometimes the cause of bronchiectasis. There is some difference of opinion as to whether foreign bodies are more frequent in the right or the left bronchus. The right bronchus is the larger, and it would be expected that a small body would either fall directly into it, or, on account of the

deviation of the trachea to the right in the thorax, would strike the left wall of the tube and rebound into the right bronchus. The lung signs produced depend upon the point of lodgment of the foreign body. The body, if small, may slip past the bronchus of the upper lobe, and this is more likely to occur on the right side owing to the greater size of the main bronchus. Under such circumstances the upper lobe will remain unaffected.

The relation of the main or stem bronchi to the chest wall is of some interest in connection with bronchiectasis. They commence at the bifurcation of the trachea, and passing downwards and backwards, terminate for all practical purposes at the level of the eighth ribs. The bifurcation of the trachea lies to the right of the mid-line at the level of the disc between the fourth and fifth dorsal vertebræ. This point corresponds in level to the fourth dorsal spine posteriorly, and to the junction of the second right costal cartilage with the sternum in front (level of the sterno-manubrial joint). The stem bronchi become so small as to be negligible at the level of the eighth ribs posteriorly, the right two inches and the left three inches from the spines of the vertebræ. Viewed from the front of the thorax, these terminal points correspond to the right fifth rib in the parasternal line, and the corresponding rib on the left side just internal to the nipple line.

In children, the trachea bifurcates opposite the third costal cartilage in front and the terminations of the stem bronchi reach the same ribs as in the adult, but at half the distance from the vertebral spines.

The points at which the branches to the upper lobes arise can be found by measuring downwards along the lines of the stem bronchi. The eparterial bronchus, which supplies the right upper lobe, arises one inch below the bifurcation of the trachea, whilst the bronchus to the left upper lobe is two inches below the bifurcation.

The middle lobe of the right lung is solely supplied by the first right ventral hyparterial bronchus.

## BRONCHITIS AND BRONCHO-PNEUMONIA.

The mucous membrane of the bronchi being continuous with that of the nasopharynx and fauces, **bronchitis** may arise by extension of infectious processes from above. Air-borne irritants have direct access to the air tubes, and in certain conditions particles of food from the pharynx may be aspirated into the respiratory passages. The air tubes are supplied with blood by the bronchial arteries, whilst the pulmonary arteries distribute blood to the terminal bronchioles and the alveoli. The bronchial arteries differ from the pulmonary in that they are subject to vaso-motor control, consequently vascular changes may be induced in the bronchi by a variety of reflex causes. Lastly, the bronchial arteries, as part of the general vascular system, may themselves carry infection to the bronchi, and this explains the occurrence of generalised bronchitis in certain of the infectious diseases.

An impervious basement membrane lies beneath the epithelial layers of the bronchi, and this membrane probably plays an important part in the course of bronchitis. Leucocytes and foreign particles cannot penetrate it, as it is not traversed by lymphatics; consequently such particles and the secretions of bronchitis are not absorbed but are passed upwards by the ciliated stream and expectorated.

The walls of the lobular bronchioles are thin and their lumina are small. Inflammatory affections of these parts tend to assume a suffocative character and to spread to the air cells, giving rise to **broncho-pneumonia**. Although there is no lymphatic absorption from the bronchi, absorption can take place from the alveoli and air cells.

The bronchial veins play practically no part in returning blood from the lung, as at the most they only drain the tissues of the root. All the blood from the lung is returned by the pulmonary veins, and consequently is poured into the left auricle of the heart. Hence there is an anatomical reason for the early occurrence of passive pulmonary congestion in association with diseases of the

mitral valve. When bronchitis is accompanied by emphysema a considerable area of pulmonary capillaries disappears. The capillaries are derived from the pulmonary arteries which supply the alveoli, hence the pressure is raised in these vessels, so hypertrophy and dilatation of the right ventricle result.

The lungs of infants and young children differ markedly from those of adults. In early life the interstitial tissue forms a considerable part of the lung and is highly vascular; the alveoli are comparatively small and thick walled, and the extent of the bronchial tubes is proportionately greater than that of the air spaces. Consequently the breath sounds are not damped by the pulmonary parenchyma to such an extent as in the adult, but are harsh or even bronchial, and may give rise to a suspicion of broncho-pneumonia when none is present. In children, as a consequence of the anatomical conditions, vascular distension and interstitial infiltration form a marked feature in broncho-pneumonia, the small alveoli are much more easily plugged, and **pulmonary collapse** is readily induced by trivial inflammations.

The yielding character of the chest wall in infancy, and the feeble respiratory movements, also contribute to the production of collapse of the lung.

#### LOBAR PNEUMONIA.

A knowledge of the relations of the fissures of the lungs to the back, sides and front of the chest is essential for the determination of the site and the extent of the lesion in **lobar pneumonia**.

The great fissure, which separates the upper lobes of the lungs from the lower parts, extends from the second dorsal spinous process across the infraspinous fossa of the scapula, traverses the axilla near the fourth rib, and terminates in front close to the sixth costal cartilage near the parasternal line. The other parts of this great fissure lie on the inner surface and base of the lung, and, consequently, cannot be indicated on the surface of the chest. If the elbow on the side examined be well raised above the level of the shoulder, and the palm of the hand carried over

the opposite shoulder and applied to the spine of the scapula of that side, the other scapula becomes so tilted that its vertebral border practically corresponds in position with the great fissure of the lung.

The transverse fissure of the right lung, which separates the middle from the upper lobe, extends from the middle point of the great fissure to the vicinity of the fourth costal cartilage near the sternum.

At the back of the chest, the upper and lower lobes of the lungs can be examined. In the axilla, the upper, middle and lower lobes of the right side are accessible, and the upper and lower lobes of the left. In front, on the right side, the upper, middle and very small part of the lower lobes are superficial; on the left the upper and middle lobes.

Just internal to the lower part of the vertebral border of the scapula is a thinly covered portion of the chest wall. This area is sometimes known as the triangle of auscultation, and lies over the middle part of the lower lobe of the lung. Its boundaries are the outer border of the trapezius, the lower border of the rhomboideus major, and the upper border of the latissimus dorsi, just before that muscle crosses the angle of the scapula. Over this triangle the breath sounds are very distinct, and lung resonance is easily obtained.

High in the axilla, over an area corresponding to the first and second intercostal spaces, the upper lobe of the lung is very thinly covered, and the physical signs of apical pneumonia are often first discovered in this situation.

Pneumonia does not always involve a complete lobe, and so the outlines of the diseased area may not sharply correspond with the fissures of the lung; it is not uncommon to find a lower lobe completely consolidated and the lobes adjacent only partly invaded. Pneumonia, too, renders the affected lobe more bulky, and often induces partial collapse of the lobe immediately adjacent, this must be borne in mind in attempting to accurately localise the mischief. The visceral layer of the pleura nearly always shares in the inflammation, and, since the

pleural layers of the fissures are in close contact, they are easily agglutinated by pneumonic inflammation. Localised empyemata may form in a fissure thus sealed up.

Sometimes the pericardium is involved as well as the pleura, but it by no means follows that this is always an invasion from the adjacent lung. In some epidemics mitral and aortic endocarditis are associated with the lung disease, these are the first valves which come into contact with blood returned from the lungs, and this, together with the occurrence of complications in other parts of the body far distant from the lungs, must be attributed to blood infection. Delirium, meningitis, arthritis, albuminuria, nephritis, enteritis, and peritonitis may be cited as examples of this.

Obstruction to the circulation in the consolidated lung naturally throws a strain upon the right ventricle, which, being at the same time poisoned by the toxæmia, may undergo rapid dilatation.

Pneumonia being a disease of the alveoli and the contiguous structures the whole of the exudation in the lung may be absorbed and carried away by the lymphatics with which the alveoli are supplied, and none expectorated. This contrasts strongly with what occurs in bronchitis where lymphatics play little or no part in getting rid of the secretion.

#### PULMONARY PHTHISIS.

The channels by which infection may reach the lung, and the methods of dissemination in that organ, have been pointed out in the section on tuberculosis. Attempts to account for the primary localisation of phthisis at the apices on anatomical grounds have not yet produced any convincing explanation.

In making a diagnosis of **apical tuberculosis** from physical signs, the normal differences between the signs at the two apices must be borne in mind. The lungs either extend to an equal height above the clavicles or the right is a little higher than the left. The normal outline of the apex passes from the seventh cervical spine outwards and upwards to a point on the anterior



edge of the trapezius, an inch and a half above the clavicle; thence the curve passes downwards and inwards towards the clavicular attachment of the sternomastoid. The pulmonary resonance above the clavicle is obtained by direct percussion downwards upon the sloping anterior surface of the apical part of the lung; this surface lies under cover of Sibson's fascia and the scalene muscles. Even in health a flatness or deficiency in percussion resonance may be expected on the right side in most individuals, possibly because of the greater muscular development on this side of the body. At the right apex, too, the breath sounds are louder, the expiratory murmur more audible and, hence, apparently prolonged, the vocal resonance more marked, and the vocal fremitus more easily appreciated. In women and children the breath sounds at this apex may even possess a bronchial quality. There is an anatomical basis for these differences. The apex of the right lung is closely applied to the side of the trachea, owing to the deviation of that tube to the right; consequently tracheal sounds are easily communicated to the upper part of the right upper lobe, whereas on the left side the œsophagus in part, the left subclavian and common carotid arteries and the aortic arch all intervene between the trachea and the lung. In addition, the right main bronchus is rather larger than the left and sooner gives off the branch to the upper lobe.

It is important to bear in mind the positions in which tuberculous deposits may be expected in the lungs and the relations these spots bear to the chest walls. The primary and oldest lesion is usually found in the upper lobe, about one or one and a half inches below the extreme apex, and rather nearer to the postero-external aspect than any other part of the surface of the lung. From this focus the disease spreads directly backwards, giving rise to signs in the supraspinous fossa, and also downwards and forwards along the anterior aspect of the upper lobe, causing indications of its presence by abnormal physical signs above the clavicle or in the first, second and third intercostal spaces about an inch and a half away from the sternal edge.

More rarely the primary lesion lies further outwards, corresponding to the first and second interspaces below the outer third of the clavicle. It is exceptional for a lower lobe to be directly invaded across the interlobar fissure—the pleura appears to have sufficient power of resistance to prevent this. Secondary deposits in the other lobes are the result of aspiration from the primary focus.

The lower lobe is usually invaded before the mischief has extended very far in the upper lobe, the middle lobe being passed over. The first focus in the lower lobe is an inch or an inch and a half below the apex and the same distance from the posterior border. Here, again, extension takes place backwards towards the posterior border of the lung, corresponding to an area on the chest wall midway between the fifth dorsal spine and the vertebral border of the scapula, and also laterally along the line of the great fissure. This line is sufficiently well indicated by placing the hand upon the spine of the opposite scapula and raising the elbow above the level of the shoulder. The vertebral border of the scapula then corresponds fairly with the oblique fissure.

Infections of the opposite lung usually occupy positions similar to those of the lesions in the lung first involved, but occasionally the opposite upper lobe shows a deposit corresponding to the upper part of the axilla, just above the interlobar septum, hence the axilla as well as the apices should always be carefully examined. In children and women with thinly covered chests, bronchial breathing may often be heard over the manubrium sterni and upper part of the sternum, and also in the interscapular region. These points correspond to the termination of the trachea and the commencement of the main bronchi. At its point of bifurcation the trachea lies under the right edge of the sternum. The bifurcation corresponds to the level of the sterno-manubrial joint in front, and the tip of the fourth dorsal spine behind, these being the surface levels of the disc between the fourth and fifth dorsal vertebræ. It is sometimes stated that the bifurcation is on the same level as the root of the scapular .

spine, but since this is opposite the disc between the third and fourth dorsal vertebræ, the tracheal bifurcation is really lower down. The bifurcation is one vertebra higher in the infant than in the adult, but, nevertheless, it corresponds to the third costal cartilage in front instead of to the second, owing to the less obliquity of the ribs. The marked deviation of the lower end of the trachea to the right is due to the pressure exerted upon it by the aortic arch, and this leaves a distinct impress on the side of the air tube. In children a "cracked-pot" sound may often be obtained below the clavicle, especially on the right side; the thinness and flexibility of the chest wall and the proximity of the trachea and bronchi account for this phenomenon, the air being easily driven out by forcible percussion.

A systolic murmur can often be heard in the subclavian arteries of those who suffer from phthisis. In some cases, no doubt, this is hæmic, but the artery is in close relation to the pleural dome, which it actually indents, and so may be compressed by pleural adhesions or by a solid portion of the lung. The inflammatory thickening of the apical pleura may also entangle and paralyse certain nerves which lie adjacent to it. The recurrent laryngeal nerves, particularly that of the right side, the phrenics and the dorsal sympathetic fibres to the eye, are all liable to implication in this way. Unilateral abductor laryngeal paralysis, unilateral paralysis of the diaphragm or unilateral ptosis, myosis and ophthalmos may thus be produced.

When there is much **fibrous contraction** of the lungs, the heart comes more fully into contact with the chest-wall, and the area of visible pulsation is increased. Under these circumstances the conus arteriosus may beat in the second left interspace, being drawn upwards as well as uncovered. The shock of pulmonary valve closure can often be actually felt in this situation. On auscultation the loudness of the second sound will be evident. Contraction of the left lung allows the area of gastric resonance to encroach upon the chest, and contraction of the right allows the liver to pass upwards. The heart and mediastinum will be drawn towards the lung which is contracted, unless previously

fixed by adhesions. When lung contracts, compensatory emphysema or local retraction of the chest wall may be expected. The close adhesion of the visceral pleura to the lung, and its permeation by some of the pulmonary lymphatics, are sufficient to account for the frequency of pleurisy and of pleural adhesions in phthisis. Small groups of recent tubercles may often be found in the visceral pleura of those suffering from the disease.

#### PNEUMONOKONIOSIS.

In this group of diseases the inhaled dust forms a natural injection of the lymphatics of the lungs. The bulk of experimental evidence shows that no absorption of particles takes place through the mucous membranes of the trachea and bronchi, where the presence of an impermeable basement membrane cuts off the lymphatics of mucosa from those of the deeper structures. After the prolonged experimental inhalation of air loaded with fine particles, the latter are found in the peribronchial and perivascular lymphatics, in the lymphoid tissue in connection with these and in the bronchial glands, positions to which they are carried after they have been absorbed by the alveolar lymphatics. In pneumokoniosis the foreign particles lie in the desquamated epithelium and epithelial spaces of the alveoli, in the interlobular septa and the lymphatics which surround the pulmonary arteries and bronchi, in the deeper layers of the visceral pleura, in the lymphoid aggregations of the lungs and in the bronchial glands. There is practically no deposit in the superficial layers of the visceral pleura, for the pulmonary lymphatics do not run in these layers, and there is no deposit in the mucous membrane of the bronchi, since the ciliated epithelium passes the particles upwards and no direct absorption takes place into the deeper layers owing to the presence of the basement membrane.

#### EMPHYSEMA.

**Interstitial emphysema** is due to the rupture of an air cell into the supporting fibrous tissue of the lung. It is practically

limited to childhood, for only at that period of life does the interstitial tissue form any considerable bulk of the organ.

**Hypertrophic emphysema** is the form of emphysema most commonly met with. In this form the lungs are actually increased in bulk. The emphysematous dilatation of air cells is chiefly found in the thin and unsupported parts of the organs, *i.e.*, along the anterior edges, around the sharp basal margins and near the roots. The accompanying increase in bulk is accommodated in part by invasion of the spare pleural space and in part by the inspiratory position assumed by the thorax, together with exaggeration of the dorsal curve of the spine. The spare pleural spaces which can accommodate the enlarged lungs are the phrenico-costal and the pleuro-pericardial portions of the pleural sinuses. The phrenico-costal sinuses, which lie below the lower edges of the lungs between the diaphragm and the chest walls, are of considerable potential capacity, and attain a vertical measurement of nearly four inches in the axillary regions. In this position the lower border of the lung normally reaches to the eighth rib whilst the pleural reflexion corresponds to the tenth rib or intercostal space. The pleuro-pericardial sinus is a part of the left pleural sac which passes further forwards over the heart than the corresponding lung does. It is easy to see how the emphysematous lungs by encroaching on these sinuses cause the upper limits of the hepatic and splenic dulness to be depressed and may diminish or obliterate the area of cardiac dulness. When the chest wall assumes an inspiratory position, in addition to the increase in its diameters which gives rise to the so-called barrel outline, there is a certain amount of gliding upwards of the ribs beneath the skin so that the surface relations become altered. The nipple may lie on the fifth instead of the fourth rib and the cardiac impulse may appear in the sixth space. In emphysema, this altered relation tends to be permanent, and must be taken into account in estimating cardiac enlargement and displacement.

The destruction of alveolar septa which takes place in emphysema considerably reduces the capillary area of the

pulmonary artery, and leads to hypertrophy and dilatation of the right cavities of the heart, with signs of back pressure in the venous system.

**Compensatory emphysema**, as its name denotes, is a form of emphysema which occurs when for some other reason part of the pulmonary tissue becomes functionless. The whole of the opposite lung may become emphysematous when one lung is crippled, and under such circumstances the mobility of the mediastinum allows the active and over-distended lung to encroach considerably on its fellow. Similarly when one lobe of a lung becomes collapsed the remaining lobe or lobes of the same side may become emphysematous, and when part of a lobe is airless or cicatrised the pulmonary tissue immediately adjacent becomes over-inflated.

## PLEURÆ AND MEDIASTINA.

### PLEURAL INFLAMMATIONS AND EFFUSIONS.

Effusions into the pleura may be passive and non-inflammatory, or inflammatory.

**Passive effusions** occur in connexion with heart failure, the fluid poured into the sac accumulating there instead of being constantly absorbed by the lymphatics. Rarely a chylous effusion is found, the thoracic duct rupturing into the sac in consequence of injury or obstruction. In the posterior mediastinum the thoracic duct lies close to the right pleural cavity, whilst in the superior mediastinum and the root of the neck it is close to the left one.

**Inflammatory effusions** are frequent, and may be either serous or purulent. When pleurisy complicates a specific fever, such as rheumatism or scarlet fever, it is assumed that the infection is carried to the pleura by the blood stream. There are many systemic capillaries adjacent to the pleura. A uniform layer of capillary blood-vessels lies on the surface of the lung, beneath the visceral pleura, and is derived from the bronchial arteries. Another wide-meshed plexus lies in the subpleural connective

tissue of the mediastinum, and is supplied by the mediastinal and pericardiac branches of the aorta and by branches of the bronchial and internal mammary arteries. A similar plexus lies in the connective tissue beneath the costal pleura, being formed by branches of all the arteries of the thoracic wall. These plexuses establish a communication between the pulmonary and pleural vessels on the one hand and the vessels of the surface of the chest on the other; and this may explain the beneficial effects of counter-irritation applied to the chest in pleural and lung diseases.

In pyæmic pleurisy the infection is carried by branches of the pulmonary artery, having been brought to the right auricle by the systemic veins. Infarctions of the lung and superjacent pleurisy result.

The visceral, mediastinal, diaphragmatic, costal and apical regions of the pleura may all be invaded by the extension of inflammation from the structures which lie in immediate relation to them.

The intimate relations of the visceral pleura to the connective tissue of the lung; and of the parietal pleura to the connective tissue of the mediastina, chest walls, diaphragm and root of the neck explain the occurrence of pleurisy by extension of inflammatory mischief from these regions, and also account for the association of pleural and pericardial adhesions with mediastinitis. Extension of infection to the **visceral pleura** is common in abscess, infarction and gangrene of the lung. Pleurisy also frequently accompanies pneumonia. The lymphatics of the lung and of the visceral pleura pass into the same efferent trunks; and the latter also receive the lymphatics of the mediastinal tissues and of the mediastinal pleura. Lymphatic convection, as well as mere continuity of tissue, may therefore play a part in the production of secondary pleurisy. In phthisis, small aggregations of miliary tubercles may often be recognised on the visceral pleura close to the older lung lesions. These miliary deposits are probably situated in the lymphatics.

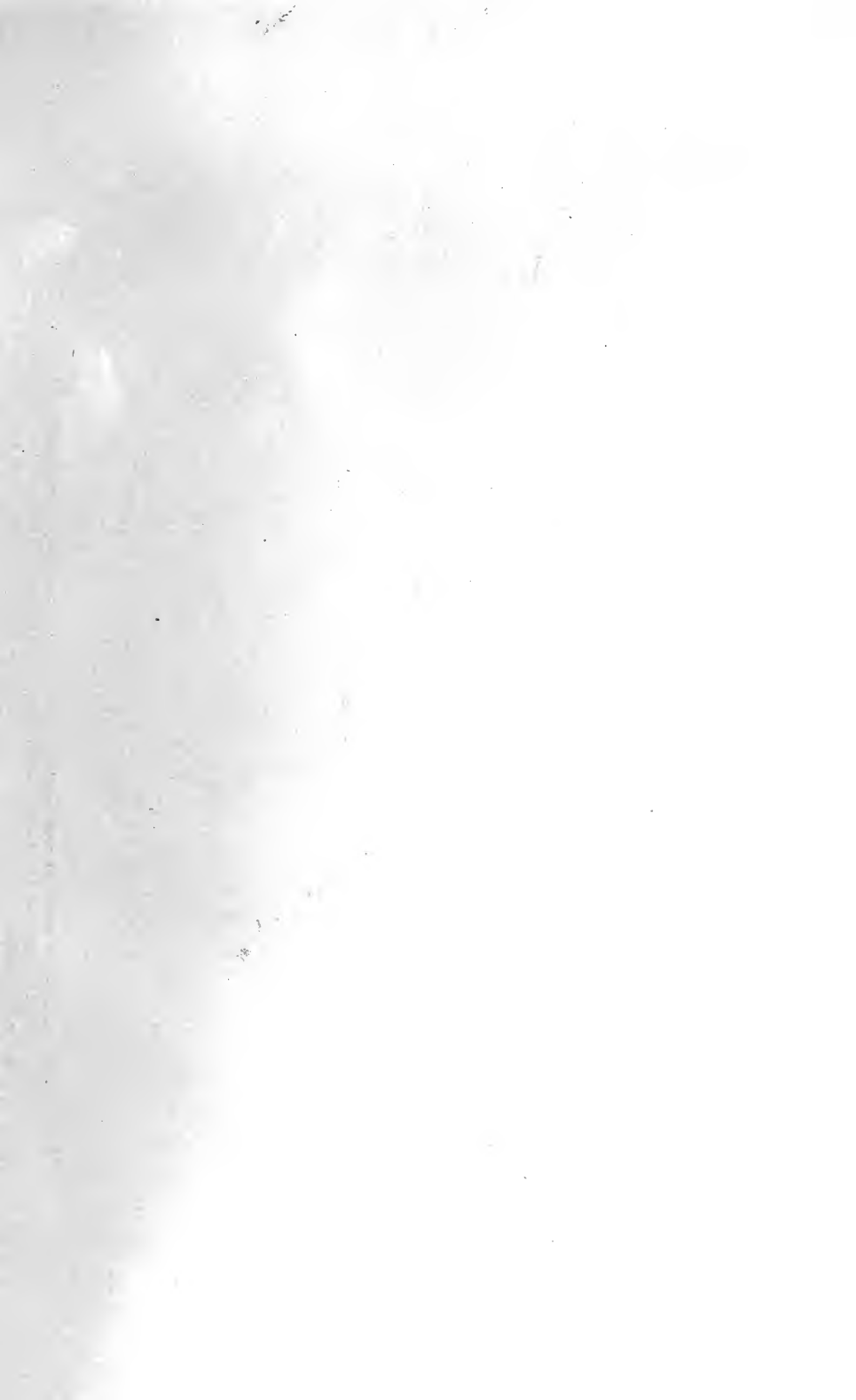
The structures which lie beneath the **mediastinal pleura** and

which, when diseased, may give rise to secondary pleurisy, are the aorta, the œsophagus and the mediastinal lymphatic glands. The transverse and descending portions of the thoracic aorta are in relation with the left pleural sac, and adhesive inflammation may accompany aneurysms of these parts of the vessel. The ascending aorta lies in relation with both sacs. The œsophagus in the posterior mediastinum abuts upon the right pleural cavity, which actually forms a small *cul-de-sac* between it and the spine. Above and below the œsophagus is in relation with the left pleural sac.

The **diaphragmatic pleura** has intimate relations with the peritoneum by lymphatic vessels which traverse the diaphragm. These channels are tortuous and narrow, so the diaphragm to a certain extent forms a barrier between the two serous sacs. Tuberculous, septic or malignant disease may invade the pleura by this route. Invasion may be facilitated by the occurrence of a defect in the posterior part of the diaphragm below the external arcuate ligament, in the region of the kidney. A small gap is not uncommon in this situation, especially on the right side. The fused subperitoneal and subpleural planes of connective tissue then form the only barrier between the peritoneum and the pleura. The base of the right pleura may be invaded directly by abscesses, hydatids or malignant growths of the liver, the diaphragm being perforated. The base of the left sac is separated by the diaphragm from the left lobe of the liver, the fundus of the stomach, the spleen and, when the stomach is contracted, the transverse colon near its splenic flexure. Consequently the left pleura can be invaded from any of these structures. The upper poles of the kidneys lie upon the lower sinuses of the pleura, the diaphragm intervening, so that perinephritic abscesses may infect the pleural sacs.

The **costal pleura** may be perforated by wounds of the chest or the ends of fractured ribs. It may also be invaded by tuberculous and other inflammations of the ribs or chest walls, and over-lying growths. Axillary abscesses occasionally give rise to pleurisy, and this may be explained by the fact that the axillary





PLEURAL DOMES.

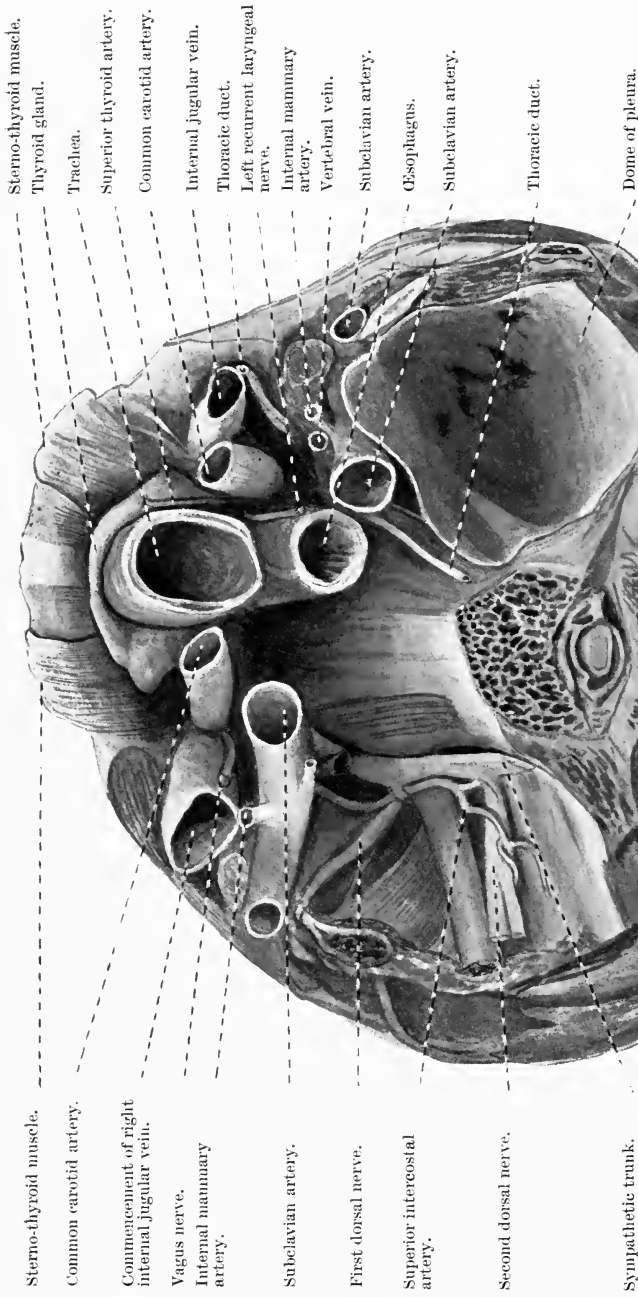


FIG. 43.—Transverse section at root of neck, immediately inferior to the pleural domes and observed from below. The specimen has been partially dissected and the pleural dome of the right side removed, to expose the structures in relation with it. The dark patches on the pleura of the left side indicate pulmonary adhesions. (Museum, Royal College of Surgeons of England.)

connective tissue is continuous over the first rib with the connective tissue which invests the pleural sac. The perforating branches of the intercostal vessels and nerves also carry with them sheaths of the same connective tissue which have been prolonged along the main vessels and nerve trunks from the posterior mediastinum.

Owing to the laxity of the subjacent connective tissue, the costal pleura is much more easily stripped from the parietes than the visceral pleura is from the lung.

The gangliated cord of the sympathetic lies over the heads of the ribs in the connective tissue beneath the costal pleura, and so the fibres which pass through the first and second dorsal rami to ascend in the cervical chain to the eye may become damaged when the pleura is inflamed.

The **pleural dome** is occasionally infected by suppurative processes in the lymphatic glands and connective tissue planes of the neck. It is also liable to injury and infection from wounds and operative procedures immediately above the clavicle. Its relation to the subclavian artery is important in connexion with aneurysms and ligature of that vessel. The continuity of the axillary vascular and nervous sheaths with the connective tissue over the pleural dome has already been mentioned.

A close plexus of lymphatic vessels lies in the subpleural tissue. The lymphatic efferents of the visceral pleura pass into the superficial collecting trunks of the lung. Those of the costal pleura open into the intercostal trunks; the diaphragmatic pleura is drained by the diaphragmatic trunks and the mediastinal pleural lymphatics pass into the glands of the posterior mediastinum. The pleural stomata, which are found in the pleura covering the diaphragm and intercostal spaces, but not in that of the mediastinum and ribs, facilitate the absorption of fluid. The lymphatic trunks are provided with valves which direct the flow away from the pleura. Thus the fluid which normally transudes into the sacs from the blood-vessels is constantly removed by the lymphatics through the agency of the respiratory pump. In pleurisy the presence of inflammatory

products prevents absorption, and in exceptional cases the effusion may exercise pressure on the lymphatic trunks. Aspiration of the fluid facilitates absorption by relieving pressure on the lymphatics and rendering more free the respiratory movements.

Pleural effusions are naturally limited by the boundaries of the pleural sacs. The costo-diaphragmatic reflections of the pleural sacs are decidedly lower than the lower edges of the lungs. The pleural reflection is about two inches below the border of the lung in the nipple line, nearly four inches below it in the mid-axilla, and about an inch and a half below it in the line of the lower angle of the scapula. The left pleural sinus overlies the stomach, and a change from resonance to dulness in this area is very characteristic of the presence of fluid in the pleural sac. The resonant gastric area is known as Traube's semilunar space. The boundaries of this space are, the lower border of the left lung above, the sloping lower edge of the left lobe of the liver to the right, the left costal margin below, and the anterior border of the spleen to the left. The space is crossed midway between its upper and lower borders by the costo-diaphragmatic reflection of the left pleural sac, consequently fluid in the sac will encroach on the area of gastric resonance much more than a solid lung does.

The lowest part of the pleural sac lies in the mid-lateral line of the body. It corresponds to the tenth rib or intercostal space, and can be readily found by drawing a horizontal line round the trunk at the level of the lower part of the first lumbar spine. This is the part of the pleura in which fluid can be early found, and from which it is late to disappear. (See also page 409.)

When effusion occurs into the pleural sac, the negative pressure in the sac is diminished and the mediastinum, being flexible, very soon undergoes a suction displacement to the opposite side. The heart lies in the mediastinum, and is displaced with it, affording an index of its displacement. At the same time the lung will become retracted towards its fixed point, which is its root. In most cases of pleural effusion the manometer still shows a negative pressure to exist within the thorax on the

affected side, the lung is probably retracted only in the neighbourhood of the fluid, and the position of the dulness does not shift appreciably with alterations in the posture of the patient. The root of the lung lies opposite the fifth, sixth and seventh dorsal vertebræ, and it is at this level, near the vertebral column, that the signs of retracted lung may sometimes be obtained in large pleural effusions. Only when the amount of fluid is very considerable will actual displacement of the liver or spleen occur.

A knowledge of the surface markings of the lung fissures (see pneumonia) is essential for the diagnosis of fluid collections, usually purulent, between the lobes. Such collections give rise to abnormal signs in the lobes which adjoin the fissures, and variations in these signs, such as shifting from one lobe to the other and back again, should arouse suspicions of an interlobar empyema.

The dome-like projection of the abdominal space into the base of the thorax allows the peritoneal cavity to invaginate the floor of the chest, the lungs and pleuræ fitting like a cap on the convexity of the projection. Hence peritoneal friction, produced between the liver or other abdominal viscus and the diaphragm, may be heard over areas which are in direct surface relation with the pleural sacs, for the latter extend downwards between the parietes and the peritoneum. An aspirator needle, too, may inadvertently be passed right through the pleural sac and withdraw fluid from the peritoneal cavity. Moreover, the action of the diaphragm will impart a respiratory rhythm to the peritoneal friction. Any of these occurrences may give rise to difficulty in diagnosis. The intimate relation of the pericardial to the pleural sacs, the former being clothed laterally by the mediastinal pleura and overlapped by the costo-mediastinal reflections in front, explains how inflammation of one sac may easily extend to the other, and also accounts for pleural friction with a cardiac rhythm. A large pericardial effusion, with retraction of the lung, may easily simulate pleurisy, and even be tapped without discovery of the error.

The pleural sacs in health are potential only, the parietal and visceral layers being in apposition. When roughened by inflammation the gliding of one layer over the other, which accompanies the respiratory movements, causes a friction sound. The accumulation of fluid between the layers will obliterate the sound by separating the rough surfaces. The normal proximity of the layers accounts for the ease with which pleural adhesions form, for the respiratory movements are lessened or absent over the inflamed areas. The inflammation of apical pleurisy may extend to the surrounding subpleural connective tissue and cause compression of the vagus, phrenic or sympathetic nerves which are in intimate relation with the pleural domes. The association of mediastinitis with pleurisy has already been referred to.

The narrowness of that part of the pleural cavity which extends between the diaphragm and the chest wall renders it inadvisable to make the incision for drainage of an **empyema**, in the lower part of the sac. After the pus has been evacuated, the diaphragm rises and approaches the chest wall, and so tends to block up the opening. Incisions in the scapular line, with resection of a part of the ninth rib, are free from this objection and afford efficient drainage of the sac in recumbency. The seventh or eighth ribs are sometimes resected, but although they are uncovered in this position when the arm is elevated, they are overlapped by the angle of the scapula when the arm is lowered. For this reason, and on account of the presence of the *latissimus dorsi*, some prefer to make the incision a little more forward.

Empyemata, if left alone, may open into a bronchus, and the danger of this undesirable occurrence has been much exaggerated. They may also point externally. The weakest part of the chest wall is in the interchondral spaces, where the intercostal muscles are deficient and the anterior perforating branches of the internal mammary afford a track. But the actual places at which a neglected empyema may present are various. Sometimes it presents near the nipple, sometimes in the lumbar

region, and sometimes it travels downwards between the vertebral and costal attachments of the diaphragm.

Sudden death sometimes follows aspiration or irrigation of the pleural sac. This is attributed to reflex inhibition of the heart through the vagus filaments which are distributed to the lung.

## PNEUMOTHORAX.

The parietal and visceral layers of the pleural sacs are normally in intimate apposition and coherent. When air by any means gains access to the pleural sac, or exceptionally when gases are generated in it by certain gas-producing bacteria, the pleural layers become separated from each other, and **pneumothorax** results.

*The visceral pleura* is in the more direct relation with the air, and it is by perforation of this layer that pneumothorax is commonly produced. In perhaps ninety per cent. of the cases the perforation is due to pulmonary tuberculosis, but it is sometimes caused by an empyema which has eroded the lung and discharged into the air passages, and is occasionally the result of abscess, gangrene or septic infarction of the lung. It is conjectured that the thin wall of an emphysematous air cell may sometimes rupture when the alveolar air pressure is suddenly raised by violent muscular effort. Pneumothorax has also been caused by wound of the visceral pleura and lung during paracentesis, but this is rare. The visceral pleura is often wounded by the ends of fractured ribs. Laceration of the lung or the tearing of a bronchus near the lung root may allow air to enter the sac. The application of a crushing force to the chest is usually responsible for such accidents, and fractures of the ribs usually coexist, but in children the thorax is so elastic that the lung may be lacerated, or even a bronchus torn across without any fracture taking place.

*The parietal pleura* is sometimes opened in its costal portion by perforating wounds, and pneumothorax may be brought about in

this way, but the firm cohesion of the pleural layers renders it possible for a small penetrating wound to damage both pleura and lung without the necessary access of air to the pleural sac. The same applies to compound fractures of the ribs.

Air is sometimes aspirated into the pleural sac by respiratory efforts during the careless performance of paracentesis.

The *apical* portion of the parietal pleura projects into the neck, and consequently air may enter the sac as the result of wounds inflicted on it during a difficult tracheotomy, or operations at the root of the neck, such as ligature of the subclavian artery. This part of the sac may also be wounded by the splinters of a fractured clavicle.

The *mediastinal* portion of the parietal pleura may be perforated by abscesses of the mediastinum, and if these communicate with the air or food passages, the pleura may become inflated. Malignant disease of the œsophagus may produce a similar result.

The *diaphragmatic* portion of the left parietal pleura is immediately above that portion of the diaphragm, which forms the roof of the stomach chamber, and is sometimes perforated by malignant disease of the stomach or transverse colon. When this occurs, the gas from these organs may invade the pleura. Abscesses of the liver or other sub-diaphragmatic collections which already communicate with the exterior of the body may effect a further communication with the pleural sac, and thus afford a channel for the access of air to the latter.

The lower part of the pleural sinus may be wounded during operations on the kidney, and air may also enter the pleura of the right side as the result of operations in the right sub-diaphragmatic region.

Any opening in the pleura must also involve the sub-pleural planes of connective tissue, and so pneumothorax may be accompanied by emphysema of the connective tissue spaces beneath the parietal pleura, or of the connective tissue of the mediastinum.

When air has free access to the pleural sac the pleural tension,



or negative pressure in the sac, is diminished, and the lung, by virtue of its elasticity, immediately retracts, unless adhesions prevent it. The mediastinum is drawn over to the opposite side, and the lung of this side also retracts somewhat in consequence. The dislocation of the cardiac impulse is an index of the displacement of the mediastinum. The inflation of the pleura causes the chest to assume an inspiratory position, and its resonance encroaches on the cardiac, hepatic or splenic areas of dulness. When the pressure in the sac rises above that of the atmosphere the diaphragm becomes depressed, carrying downwards the viscera in contact with its under surface.

Pneumothorax may be simulated on the left side by a stomach much distended with gas. The stomach and colon may also form the contents of a diaphragmatic hernia, and under these circumstances, if filled with gas, may give rise to signs of air in the left pleural sac. Repeated examination may throw light on the case by showing that the resonant area is at times replaced by dulness, presumably because some fluid has been able to pass into the incarcerated stomach or bowel.

It is exceptional to find air in the pleural sac, without some serous fluid or pus, for the lesion which admits the air also opens up a track for infection.

#### MEDIASTINAL EMPHYSEMA.

The mediastinal connective tissue surrounds the main air passages, and is prolonged into the root of the lung, where it becomes continuous with the connective tissue basis of that organ. Air may gain access to its spaces by perforation or rupture of the trachea, or a bronchus, or by the formation of a communication between the air cells of the lung and the surrounding connective tissue planes. Such communications may occur in connection with destructive pulmonary inflammations, extreme emphysema, or rupture of a cell during a paroxysm of whooping cough.

The deeper connective tissue planes of the neck are continuous

with those of the mediastina. The connective tissue of the retropharyngeal space, which lies between condensed layers known to anatomists as the prevertebral and retropharyngeal fasciæ respectively, is directly continuous with the connective tissue of the posterior mediastinum surrounding the œsophagus. Perforation of the latter may give rise to emphysema in these layers. The connective tissue which surrounds the trachea is encased by a condensed lamina known as the pretracheal fascia, and this fascia blends with the fibrous pericardium after encasing the innominate vein. As the result of difficult tracheotomies or wounds of the cervical fascia, air may be sucked or driven into the layer of connective tissue which surrounds the trachea under the pretracheal fascia. It then passes into the superior mediastinum, inflating the connective tissue spaces in this region, travels onwards in the connective tissue on the front of the pericardium, and laterally invades the connective tissue sheaths of the pulmonary roots, ultimately reaching the interstitial connective tissue of the lungs. Doubtless a valvular arrangement of the wound leads to this inflation of the connective tissue by violent respiratory efforts. Similar results may follow the passage of a tracheotomy tube into the connective tissue between the pretracheal layer and the trachea.

The extension of the emphysema may occur in the reverse direction. When an air cell ruptures into the connective tissue of the lung the air first gains access to the connective tissue of the lung and its root, and then spreads to the mediastinum, pericardial connective tissue, and ultimately to the neck, whence it may overflow into the subcutaneous connective tissue also.

Invasion of the mediastinum by gas-producing bacteria occasionally produces a spreading emphysema.

The presence of air in the extra-pericardial tissue may closely simulate pericardial friction; the air in the adjacent connective tissue of the lung gives rise to crackling sounds similar to the consonating crepitations of pneumonia; air in the connective tissue on the front of the thorax gives rise to a crackling which can be heard with the stethoscope and appreciated by the hand.

## MEDIASTINAL INFLAMMATION AND SUPPURATION.

**Mediastinal inflammation** is frequently associated with pleural and pericardial inflammations or adhesions. The direct continuity of the mediastinal tissue with the connective tissue of the lung and pericardium explains this association. After reaching and ensheathing the root of the lung the connective tissue spreads into the parenchyma of the organ on the one hand and extends over its surface as an extremely thin subpleural layer on the other. In like manner the mediastinal tissue ensheaths the fibrous pericardium and extends forwards in the narrow interpleural space behind the lower part of the sternum. It is also directly continuous, both in front and behind, with the layer of loose connective tissue between the parietal pleura and the chest wall.

There are many lymphatic glands in the mediastinum, and it is possible that both acute and chronic inflammations may at times spread from these. The bronchial group of these glands is very apt to be tuberculous.

When the mediastinal tissue becomes indurated the structures most likely to be compressed are the systemic and the pulmonary veins. This is accounted for by the thinness of their walls. The pulmonary artery, the aorta, and the trachea and bronchi, having thicker walls, suffer less. Although there is only a fraction of the circumference of the inferior vena cava in the thorax, yet it has been obstructed by mediastinal induration. The phrenic nerves, the vagi, and their recurrent laryngeal branches, all lie in the mediastinal connective tissue planes, and may suffer compression. The same holds for the sympathetic chain which lies over the heads of the ribs in the subpleural layer. It is exceptional for dysphagia to occur in chronic mediastinitis, although the œsophagus lies in the mediastinum.

**Mediastinal suppuration** may originate from structures which lie in the spaces or reach the mediastinum by extension from other parts. The mediastinal glands, the thymus, and the œsophagus may all originate the mischief. Extension may

occur from the adjacent vertebræ, ribs, or sternum. Occasionally dermoids and hydatids of the mediastinum suppurate. Pus may reach the spaces from above by burrowing down in the retro-pharyngeal space or around the trachea. When the mediastinitis is secondary to perforations of the œsophagus or of œsophageal pouches, it is especially likely to become gangrenous.

The course of the pus is determined by anatomical relations. It may invade the subpleural tissue or the interstitial tissue of the lung by continuity. It may burst into the œsophagus or air passages, or into the pleural or pericardial sacs. Lying around the large blood-vessels it may soften and erode their walls. The connective tissue planes may direct it to the root of the neck, or it may come forwards in the interchondral spaces by tracking along the investing sheaths of the anterior intercostal vessels. The continuity of the mediastinal tissue with the subperitoneal tissue around the aorta may direct the abscess through the aortic opening of the diaphragm, or anteriorly it may pass through the interval between the sternal and costal portions of the same muscle.

Should an attempt be made to open the mediastinum from the back it is necessary to bear in mind the relations the pleural sacs bear to it. Below the eighth dorsal vertebra on the right side a small *cul-de-sac* of pleura intervenes between the œsophagus and the spinal column. On the left side the posterior mediastinal pleura passes direct from the left side of the aorta to the bodies of the vertebræ. Hence there is less risk of opening the pleural sac if the space be approached from the left side of the vertebral column in this region.

#### MEDIASTINAL GROWTHS.

There are many structures in or near the mediastina from which new growths may originate. The most important are the groups of lymphatic glands, the œsophagus, the trachea, and bronchi. Tumours may arise in the residue of the thymus or spring from the periosteum of the vertebræ, ribs, or sternum.

Accessory thyroids are rarely found in the superior mediastinum, and dermoid cysts sometimes lie behind the mesosternum. Gumata may also grow in mediastinal tissues.

The mediastinum is easily invaded along the connective tissue sheaths of the bronchi by new growths which originate in the lung. The bronchial connective tissue sheaths contain lymphatic vessels and blood-vessels, and are directly continuous with the mediastinal connective tissue. Pulmonary new growths may also give rise to detached secondary mediastinal growths, which consist of infected lymphatic glands.

A primary carcinomatous growth is theoretically most likely to originate in the posterior part of the superior mediastinum or in the posterior mediastinum. The presence of the epithelial structures of the œsophagus, trachea, bronchi, and thoracic duct in these situations accounts for this. Sarcomata arise chiefly from the mediastinal lymphatic glands, but may grow also from the periosteal boundaries of the space, that is from the vertebræ, ribs, or sternum.

The positions and relations of the glandular groups are of great importance in the consideration of the pressure symptoms induced by mediastinal tumours.

The cardiac group of glands lies on the front of the arch of the aorta, and is therefore in the superior mediastinum. Isolated glands prolong this group upwards on each side to the terminations of the right and left lymphatic ducts—that is, to the junction of the internal jugular and subclavian veins. The main mass of glands lies in close relation with the innominate veins and the three great branches of the aortic arch. The trachea lies posterior to them. The upward prolongation of the right side follows the superior vena cava. Enlargement of the glands may cause venous obstruction, inequality of pulses, and dyspnoea.

The posterior mediastinal glands are grouped round the œsophagus. Some are between the œsophagus and the pericardium, and some lie close to the thoracic duct. These glands are particularly likely to suffer in malignant disease of the gullet.

The middle group of thoracic glands is called peritracheo-bronchial, and is by far the most important. It lies at the junction of the superior and posterior mediastina, and on account of the practical importance of its relations, is divided into four sub-groups, although these are continuous with each other. The groups comprise (1) the glands around the right bronchus, (2) the glands around the left bronchus, (3) the glands which lie in the bifurcation of the trachea, and (4) the glands which follow the bronchi into the lungs and are embedded in the pulmonary tissue.

The glands around the right bronchus are wedged in between the termination of the trachea and the lung. When enlarged they may press upon the superior cava which lies in front of them, or the azygos vein which arches over them, or the vagus which passes down behind them. They are not far removed from the right pulmonary artery, and, extending as high as the concavity of the subclavian artery, they are favourably situated to compress the right recurrent laryngeal nerve, which turns round that vessel.

The glands around the left bronchus occupy a corresponding position to those around the right. They extend upwards into the concavity of the aortic arch to reach the left recurrent laryngeal nerve, whilst the vagus is behind them. Both these nerves and also the pulmonary artery may be compressed by enlargement of this sub-group.

The glands in the bifurcation of the trachea abut upon the pericardium, which separates them from the back of the left auricle. The posterior pulmonary plexus and the œsophagus lie behind them. These glands, when infiltrated with malignant growth, have invaded the pericardium and have compressed the œsophagus.

The bronchial glands which follow the air tubes into the lung may be traced as far as the fourth sub-divisions of the bronchi. Disease of these glands may be mistaken for primary disease in the lung tissue.

The enlargement of certain external groups of lymphatic

MEDIASTINAL GROWTH.

Right common carotid artery.

Right subclavian artery.

Right vagus nerve, somewhat displaced by the growth which presses against it.

Section of vena azygos major.

Cut surface of growth.

Right lung.

Trachea.

Right recurrent laryngeal nerve.

Left lung.

Cut surface of growth.

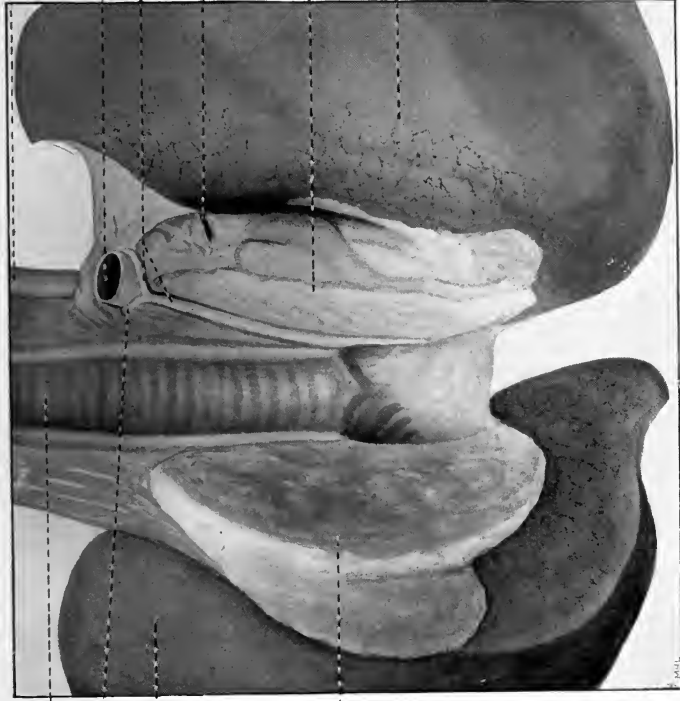


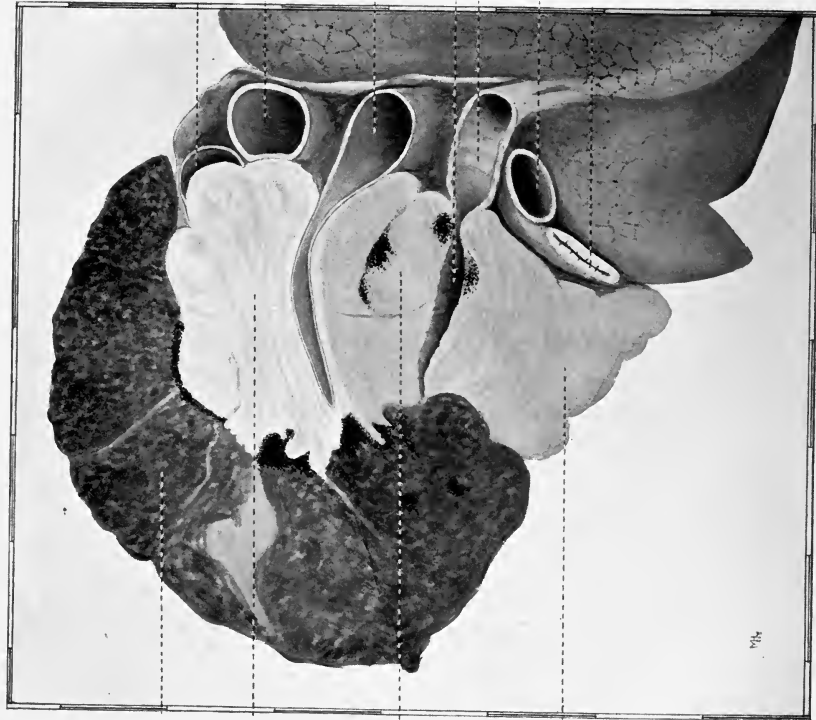
FIG. 45.—Posterior view of a mediastinal lymphosarcoma which had caused right recurrent laryngeal paralysis. The growth has been divided vertically and the two halves pulled apart. The intimate relation of the right half to the vagus, at the point where the recurrent laryngeal branch leaves the main trunk, is well seen.







MEDIASTINAL GROWTH.



Collapsed and infarcted right lung.

Lympho-sarcomatous growth, showing indistinctly the outlines of lymphatic glands.

Lymphatic glands outlined by pigment.

Lympho-sarcomatous growth.

Commencing invasion of superior vena cava by growth.

Ascending aorta, slightly compressed laterally but not invaded.

Pulmonary artery, compressed but not invaded.

Right bronchus, partly destroyed.

Bifurcation of trachea.

Descending thoracic aorta.

Esophagus.

FIG. 46.—Lympho-sarcomatous growth arising in mediastinal glands, showing the manner in which the different structures in the mediastinum are involved.

glands may aid the diagnosis of both primary and secondary mediastinal growths. The supraclavicular, the axillary, and the inguinal glands are accessible, and the abdominal glands can be palpated if enlarged to any great extent. The connection of the supraclavicular and axillary glands with the lymphatics of the mediastinum is not direct. The supraclavicular glands receive no efferents from the mediastinal groups, but the ducts of both groups open at the junction of the internal jugular and subclavian veins. When the supraclavicular glands are invaded in connection with malignant disease of the thorax it is probably by retrograde infection. A small gland which lies deeply behind the clavicular origin of the left sterno-mastoid muscle, and is sometimes known as Virchow's gland, is usually the first gland involved by malignant infection from below, whether from lung, pleura, œsophagus, or stomach. It has also been pointed out that growth may reach the posterior triangle by travelling along the sheath of the subclavian artery.

The axillary glands are in indirect communication with the lymphatics of the parietal pleura. The efferents from the parietal pleura pass into the intercostal lymphatic trunks, and the latter, by means of trunks which accompany the perforating branches of the intercostal arteries, communicate with the axillary glands. The axillary glands are in turn continuous with the infraclavicular or subclavian group.

Lymphosarcomata, arising in the thoracic lymph glands, have a tendency to creep along the mediastinal connective tissue surrounding the air tubes and great vessels. They envelop the trachea and bronchi, and so invade the lung from its root. Descending in the sheaths of the pulmonary artery and aorta, they invade the pericardium. Extending towards the heart around the superior vena cava and the pulmonary veins, they may actually invade the auricles. They may also invade the myocardium by travelling along the sheaths of the coronary arteries. They have a tendency to fungate into veins but not into arteries, and masses of loose growth have been found in the cavities of both sides of the heart. Passing upwards in the

sheaths of the great branches of the aorta, the neoplasm may find its way to the root of the neck and into the posterior triangles.

Carcinomata are said to be more destructive in their growth, infiltrating, and so destroying, rather than surrounding, the vessels and nerves with which they come in contact.

Any of the nerves in the thorax may be involved in mediastinal growths. The recurrent laryngeal nerves have already been mentioned. The fact that the phrenic nerves and the vagi completely traverse the chest affords many chances for their compression. Implication of one phrenic nerve will produce unilateral paralysis of the diaphragm. Disturbance of the branches of the vagus probably accounts for the asthmatic paroxysms of dyspnoea which sometimes accompany mediastinal tumours, the vagus being the motor nerve to the bronchial muscles. A growth in the superior mediastinum may involve the brachial plexus, or pressing on the orbital fibres of the sympathetic produce ptosis and myosis, but these occurrences are rare. In both superior and posterior mediastina the intercostal nerves and even the spinal column may be attacked. The intervertebral foramina allow the growth, by tracking along the vessels and nerves, to reach the interior of the spinal canal.

**Venous obstruction** is a common and striking phenomenon of mediastinal growth, but may also occur in connection with aneurysm and mediastinitis. The veins most likely to suffer are the superior vena cava and the two innominates. The azygos veins are sometimes occluded. The inferior vena cava rarely suffers; its extremely short course in the thorax and its remoteness from the principal groups of glands no doubt account for its comparative immunity. Obstruction of the superior vena cava produces congestion in the areas of its tributaries. The parts involved are the head, face, tongue, fauces, ears, neck, arms, and chest. The circulation within the cranium is obstructed, the nasal mucous membrane is turgid and inclined to bleed; even exophthalmos and conjunctival hæmorrhage may result. If only one innominate or one subclavian vein be obstructed the area of congestion is more restricted. In some instances of compression

of one innominate vein, it is said that the external jugular vein of the obstructed side remains distended during inspiration, whilst the vein on the opposite side is emptied by the thoracic suction.

When the superior vena cava or its large tributaries are obstructed, the feeding veins become distended, and relief can only be obtained by diversion of blood into the inferior cava. This necessitates the opening up of collateral channels. These channels lie, some within the body cavity, some in the depth of the body walls and some on the surface. In rare instances the deeper collateral channels are sufficient to relieve the congestion, and no enlargement of the surface veins can be detected; in others a congested network of superficial veins appears carrying blood from above downwards. The following are the chief possible channels of collateral circulation (Fig. 47, p. 338):—

1. Within the body cavity the azygos veins, which receive the blood from most of the intercostal spaces, are connected below with the ascending lumbar veins and so communicate with the lumbar, renal, or iliac tributaries of the inferior vena cava.

2. Within the rectus sheath the deep superior epigastric veins which accompany the epigastric branches of the internal mammary artery, communicate with the deep inferior epigastric veins which accompany the deep epigastric artery. Thus the innominate vein is linked with the external iliac vein. The lumbar tributaries of the inferior vena cava also communicate with both the superior and inferior epigastric veins.

3. On the surface of the chest and abdomen, mesial, lateral, and posterior groups of anastomosing veins exist. These when enlarged form a striking clinical picture. The mesial superficial veins consist above—that is, on the chest and epigastrium—of a plexus of vessels derived from the anterior intercostal tributaries of the internal mammary veins, and near the free margin of the thorax, from tributaries of the deep superior epigastric veins. A large vein may often be seen running vertically from the ensiform cartilage to the umbilicus. This may be called the superficial superior epigastric vein. It

communicates below with the venous circle of the umbilicus, above with the internal mammary vein, and receives tributaries from the superficial network of the epigastrium. The blood from all these superior veins is received below by the superficial inferior epigastric veins, which accompany the superficial epigastric artery, by perforating tributaries of the deep inferior epigastric veins which accompany the deep epigastric artery, and by branches of the lumbar veins. Thus the blood can find its way from above into the internal saphenous veins, the external iliac veins, and the inferior vena cava.

The lateral group of superficial anastomosing veins is formed as follows. On the side of the lower part of the thorax is a long trunk known as the thoracico-epigastric vein. This communicates above with the long thoracic (external mammary) vein, a tributary of the axillary vein. The long trunk communicates below, usually in a plexiform manner, with the superficial inferior epigastric vein, or with the superficial circumflex iliac vein, or with both. On the side of the chest wall above, the costo-axillary veins, which are lateral perforating vessels from the seven upper intercostal spaces, unite and pass into the lateral main trunk, whilst below the same trunk communicates with the superficial lumbar veins. This long anastomosis links the axillary and intercostal veins with the internal saphenous vein and the inferior cava. The posterior group of anastomosing superficial veins consists of large inter-communicating tributaries of the lumbar and intercostal veins on the back. Engorgement of these veins has been supposed to indicate obstruction of the azygos major trunk.

Pressure on the air tubes causes the most serious **dyspnœa**, and also gives rise to various secondary changes in the lungs. The dyspnœa may be paroxysmal, and then is probably due to implication of nerves. The right vagus and the left recurrent laryngeal nerves lie at the sides of the trachea, the vagi pass behind the roots of the lungs, and the left recurrent laryngeal is for a short distance in immediate relation with the thoracic part of the trachea. The forward projection of the bodies of the

COLLATERAL VENOUS CIRCULATION.

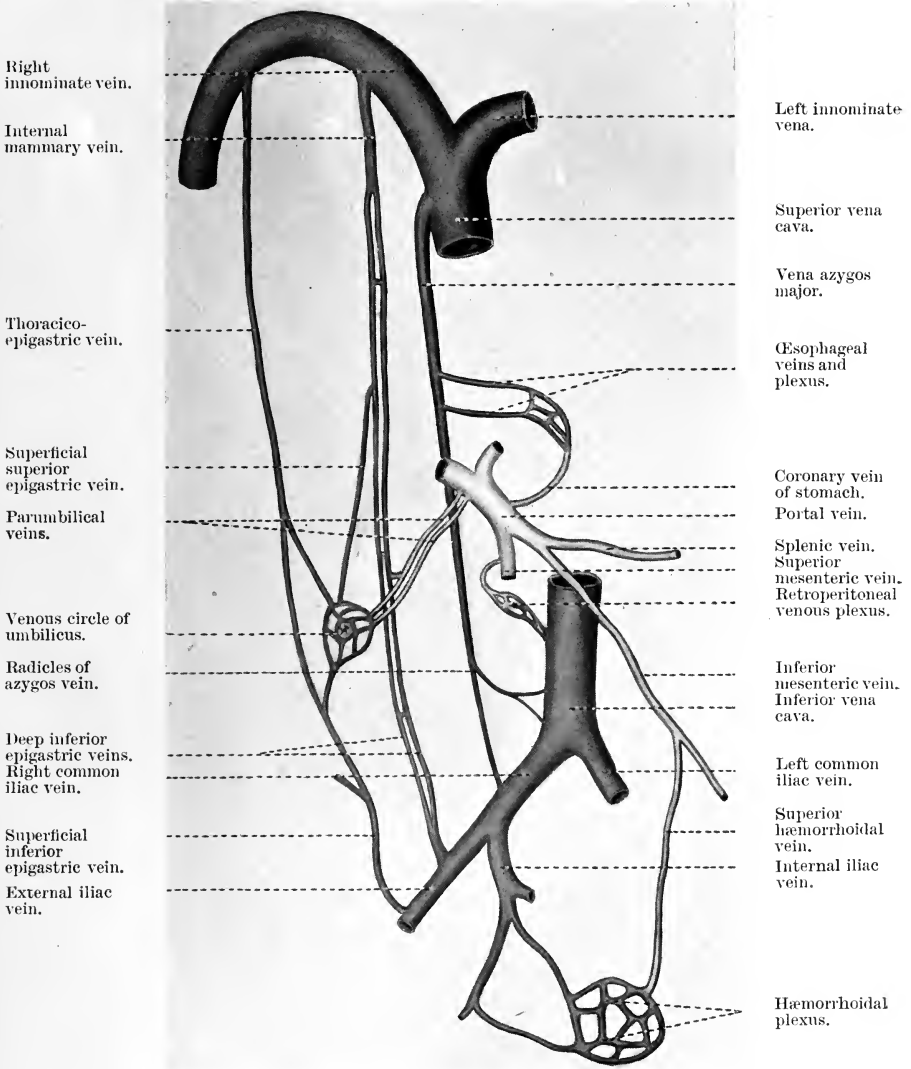


FIG. 47.—A diagram to show the communications which exist between the superior and inferior caval veins, and also those which occur between the portal and systemic veins. (After O. Schultze, much modified.)





vertebræ into the thorax considerably narrows the antero-posterior diameter, especially above, where the trachea and bronchi lie. Hence a growth in the superior mediastinum is in a very favourable position to produce pressure on the air passages, and, moreover, it is in this locality that the most important groups of lymphatic glands, the residue of the thymus and aberrant thyroids are most likely to be found.

Dyspnoea may also be the result of **pericardial** and **pleural effusions**. The relations of the serous sacs to the mediastinum and its contained glands, the fact that the efferent lymphatics and veins of the sacs pass to the mediastinum, and the continuity of the connective tissue basis of the sacs with the mediastinal tissue, sufficiently account for the passive effusions and inflammatory affections which are found associated with growth in the thorax.

#### TUBERCULOSIS OF MEDIASTINAL GLANDS.

In childhood the peritracheo-bronchial group of lymphatic glands often becomes tuberculous. The cardiac group of glands may also become involved. The glands first mentioned receive the lymphatics of the lung and visceral pleura, of the heart and pericardium, and in part of the thymus and œsophagus. The serial prolongation of the glands into the lungs explains the frequent occurrence of islets of caseation near the roots in children. The anatomical relations of the glands have been described already. They are in a favourable position to produce vagus irritation and so give rise to a dry cough. Occasionally the recurrent laryngeal nerves are implicated, and laryngeal paralysis observed. The glands may interfere with the air entry into a whole lung or one of its lobes, and if very large may produce dulness, with increased conduction of the voice and breath sounds, in the interscapular or upper sternal regions. A venous hum heard over the manubrium sterni when the head is fully retracted is supposed to indicate pressure on one of the venous trunks of the mediastinum. Traction diverticula of the

oesophagus are believed to be initiated by disease of these glands. The inflamed glands have been known to perforate any of the following structures, which are in close relation with them: trachea, main bronchi, oesophagus, pericardium, aorta, pulmonary artery, pulmonary vein, or thoracic duct. They may produce chronic mediastinal suppuration. By opening in two directions they have given rise to communications between the oesophagus and the air tubes, or the air tubes and the mediastinum.

## CHAPTER XVI.

### DISEASES OF THE ALIMENTARY TRACT, PERITONEUM, AND DIGESTIVE GLANDS.

#### THE LIPS.

THE lips are well supplied with blood from the coronary arteries of the facial, and this, together with their mode of development, may account for the frequency with which they are the site of *angiomas* or *nævi*. Their exposed position renders them liable to injury and subsequent inflammation, and should a *fissure* be produced, the contraction of the circular fibres of the orbicularis oris frequently interferes with healing because of the want of rest thereby induced, in a manner similar to that caused by the action of the external sphincter in cases of anal fissure.

The presence of many and large mucous glands explains the incidence of *mucocèles* or retention cysts.

A *carbuncle* occurring on the upper lip is always a serious affection, because of the possibility of thrombosis spreading from the facial to the ophthalmic vein through the communication at the internal canthus of the eye, and so backwards to involve the cranial sinuses, particularly the cavernous, whereby severe cerebral symptoms may be produced.

A *primary syphilitic infection* may occur on either lip from contact.

*Carcinoma*, in the form of epithelioma, occurs almost exclusively in the lower lip, a reason for this fact being that the lower lip is the one which is irritated in the use of a pipe.

The lymphatic glands which are affected in inflammation and epithelioma of the lips are first the submental and submandibular, and afterwards the deep glands along the carotid sheath,

and particularly those near the bifurcation of the common carotid.

*Wounds of the lip* may bleed profusely from the coronary branches of the facial artery, but the hæmorrhage can be readily controlled temporarily by compressing these vessels between the finger and thumb. The arteries lie closer to the mucous membrane than the skin surface. In suturing it is important to remember that care must be taken to approximate the edges of the wound accurately, so as to bring the red margin of the lip exactly into line.

**Hare-lip.**—A common congenital deformity is that known as hare lip. In the development of the face the median fronto-nasal process as a whole should become joined with the bilateral maxillary processes, the deeper bony parts articulating with one another and the superficial cutaneous portions fusing. Failure of the junction of the skin edges of the lower part of the cleft between the above-mentioned processes constitutes hare-lip. This may vary in degree from a slight notch in the red margin of the upper lip, to a cleft extending right up into the anterior naris. As a rule, in a hare-lip the red margin is continued up on the sides of the gap. Moreover, the cleft may be unilateral or bilateral; if bilateral, the median portion, consisting of the skin covering the premaxillary bones, may be carried forwards on the end of the vomer. The premaxillary bones contain the incisor teeth, and in dealing with this part during the closure of the hare-lip, it has to be remembered that if the bone is forced back, there is a tendency for these teeth, when they appear, to be rotated backwards in such a manner that they will not close properly upon the lower incisors. Hare-lip may be associated with a cleft palate.

*Median hare-lip* is extremely rare. In this variety the notch between the internal nasal prominences of the fronto-nasal process remains unobliterated, and the cleft may be prolonged by a furrow which runs on to the tip of the nose.

After closure of a hare-lip there is a possibility that asphyxia may arise, for the dressing still further diminishes the opening of the anterior nares.

## THE MOUTH.

**Cleft Palate.**—A cleft may involve both the soft and the hard palates, or the soft palate alone. Very rarely, in the congenital form, the hard palate is cleft without the soft. The cleft is practically median until it reaches the spot where the intermaxillary suture divides laterally into that between the premaxillary bone and the maxilla. It is uncommon to get a complete cleft of the palate unaccompanied by a hare-lip, though it is extremely frequent for a hare-lip to be present without a cleft of the palate.

The higher the arch of the palate, the easier as a rule is the usual operation for closure, on account of the greater breadth of the muco-periosteal flap that can be separated. The attachment of the velum palati to the ridge on the posterior aspect of the palate bone indicates the necessity for a free division at this spot, so as to allow the two freshened edges of the soft palate to be brought together by sutures without tension.

## THE GUMS AND TEETH.

An **epulis** usually has its origin from the lining membrane of the alveolus of a tooth, and although the prominence of the growth appears upon the gum, its pedicle lies hidden within the tooth socket. In the majority of cases it is therefore necessary to remove the tooth, and cut out the epulis together with that part of the socket from which it is growing.

An **alveolar abscess** is present when caries of a tooth causes the formation of pus within an alveolus, and a knowledge of the anatomical relations of the part explains many points in the progress of the inflammation and its treatment. Purulent matter will find its exit along the line of least resistance. It most commonly therefore tracks upwards or downwards along the side of the tooth, and elevating the mucous membrane of the gum, forms a typical "gum-boil," which as a rule discharges itself into the cavity of the mouth.

On the other hand, the pus may find an exit through the substance of the bony tissue of the jaw. In the case of the mandible, if it reaches the external surface of the bone, it raises the periosteum and collects either between that membrane and the osseous tissue, or, perforating the membrane, forms a cavity in the superficial soft structures. It will thus be seen that an abscess of this character consists of pus in two cavities with a narrow track between. The one cavity, the alveolus, is small; the other cavity, external to the bone, may be many times greater (Fig. 48).

In order to obtain a satisfactory evacuation of the purulent fluid, it is desirable that the offending tooth should be extracted, to drain the alveolar cavity, and that an incision should be made from within the mouth, keeping close to the bone, to open the external collection. By this internal incision a disfiguring scar in the skin may be prevented. Unless the knife is kept practically in contact with the bone, there may be some danger of wounding the facial artery, near the anterior border of the masseter.

In the maxilla, pus may find its way through the bone in two directions, either upwards into the antral cavity or inwards beneath the muco-periosteum of the palatal process of the maxilla. Pus formed in connexion with the second molar is that which most commonly induces **antral empyema**, owing to the fact that the fangs of this tooth may actually project through the bony floor of the cavity and slightly raise its muco-periosteum. Occasionally the roots of the first molar or second bicuspid also perforate the floor of the antrum. Pus in the antrum may find its exit by the side of the affected tooth into the mouth, or through the opening of the antrum into the middle meatus of the nose, or occasionally through the anterior wall of the cavity into the tissues of the cheek. It is only, however, in a certain number of instances that the purulent fluid will flow into the nasal cavity, because the aperture in the natural state with all the bones and the mucous membrane present, is really a small one, and may be readily occluded by the swelling of the soft tissues. Further, the opening lies at

ALVEOLAR ABSCESS.

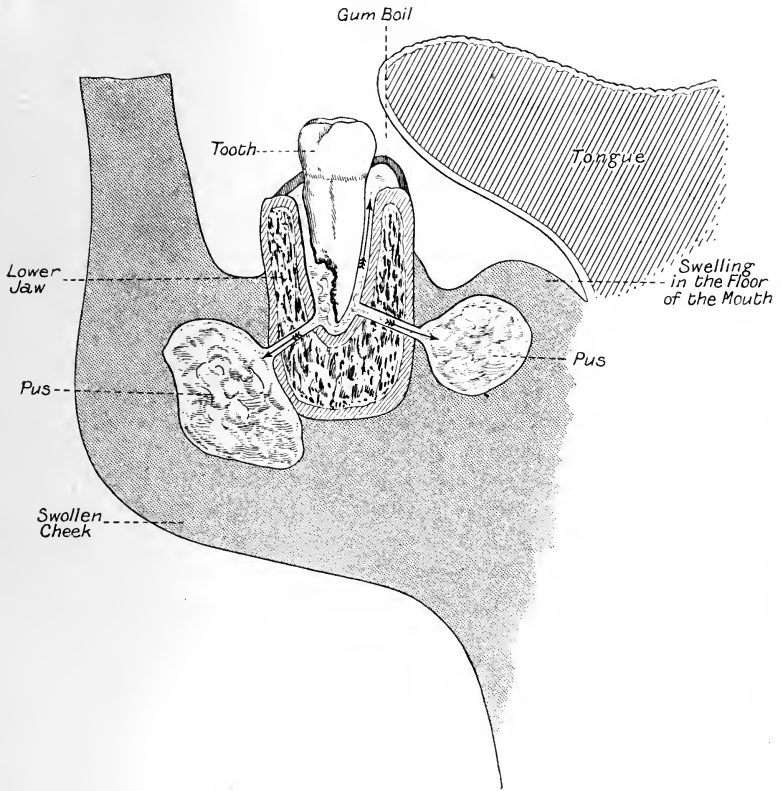


FIG. 48.—Diagram to illustrate how pus may track from an alveolus.





nearly the highest point of the cavity, and unless the head is inclined to the opposite side, pus must fill the antrum before it will overflow into the nose.

In incising an abscess beneath the muco-periosteum of the hard palate, the presence of the somewhat large anterior palatine artery, lying in close apposition with the inner border of the alveolar process, should be remembered, as hæmorrhage is likely to be troublesome if the vessel is injured.

The alveolar processes of the maxilla and the mandible may be expanded by cystic or solid new growths. **Cysts** may be derived from the original epithelial tooth elements, while the common new growth is that of the character of a **myeloma**. In both there will be a tendency for a considerable thinning of the compact bone, frequently giving rise to a characteristic parchment-like crackling.

#### THE TONGUE.

**Wounds of the Tongue.**—A deep, incised wound of the tongue, caused most commonly by the closure of the teeth upon it, is apt to bleed rather freely, on account of the vascularity of the organ. If the ranine artery be cut across, it will be needful to apply a ligature, but in the majority of cases a stitch inserted deeply into the substance of the tongue by means of a fully curved needle will control the hæmorrhage by the pressure of the two surfaces of the wound one upon the other.

**Glossitis.**—The exposed position of the surface of the tongue and its liability to irritation and injury, either from the passage of food substances over it or from its oft-repeated movements against the teeth, renders superficial inflammation extremely common.

*Dyspeptic ulcers* are apt to occur at or near the tip of the tongue, probably from the incessant worrying of this portion of the mucous membrane by contact with the none too clean and often roughened posterior surface of the incisor teeth.

The ordinary *superficial dental ulcer* of the tongue is seen more

frequently on the lateral margin of the organ, and corresponds to the position of a jagged bicuspid or molar, and it is here again that epithelioma most frequently originates.

The small ulcers seen upon the frænum of the tongue in some cases of whooping-cough may perhaps be explained by the traction of this band of tissue across the two lower central incisors during a paroxysm.

*Interstitial glossitis* of a gummatous character is most commonly found in the middle line of the tongue towards the base, which is possibly due to the fact that there is little if any arterial anastomosis from one half of the tongue to the other, the practically extra-vascular area permitting the development of the infective granuloma and its subsequent caseation.

**Epithelioma of the Tongue.**—Unfortunately this is by no means an infrequent disease, and a thorough knowledge of the anatomical considerations concerning it is of the utmost importance in its early and radical treatment.

The neoplasm commences as a rule in that part of the tongue which is most subjected to irritation: hence, as has been mentioned, the side of the tongue is one of the most frequent sites. The disease spreads locally in the substance of the organ, and if left untouched, sooner or later involves the floor of the mouth and subsequently the mandible. This local spread accounts for the fixation of this naturally freely moveable organ, and when the loss of mobility is well marked, and therefore protusion greatly interfered with, the possibility of a complete extirpation of the disease is extremely doubtful.

The lymphatic glands are apt to be early enlarged. The submandibular and the deep carotid glands are those which are first the sites of secondary deposits. The impossibility of determining the actual implication of the glands in the early stages of the disease renders it highly desirable, if not absolutely necessary, that all the lymphatic nodes of the region should be dissected out at the time of the operation upon the tongue, or very soon afterwards. The submandibular glands lie in close connection with, and even sometimes partially buried in, the submandibular

salivary gland. Hence it is not altogether easy to thoroughly eradicate them without at the same time extracting the salivary gland. No hesitation should be felt in doing so as the other glands are amply sufficient to keep the mouth moist, and the wound does not as a matter of experience become infected from the mouth along that part of Wharton's duct which is left behind.

The same incision which is made for the removal of the glands before excision of the tongue can be utilised for ligature of the lingual artery. If this vessel is tied where it lies under cover of the hyo-glossus muscle, it should be recollected that its direction is much more nearly vertical than horizontal, and great care must be taken not to miss the artery and pass through the middle constrictor, and so open into the pharynx and provide a pathway for infection into the cervical wound. Ligature of the lingual artery beneath the hyo-glossus does not cut off the blood supply to the dorsum of the tongue near its base, seeing that the dorsalis linguæ branch arises behind the posterior border of the hyo-glossus. It is well, therefore, if the tongue or half of it is to be removed far back, to ligature the lingual soon after its origin from the external carotid. In cases of primary or secondary hæmorrhage from the lingual when it has been tied in the floor of the mouth, arrest of the bloodflow may be easily brought about temporarily by drawing forward the base of the tongue with the finger and pressing it against the body of the mandible. This temporary arrest may need to be followed by ligature of the external carotid. The artery should be secured distal to the origin of the superior thyroid branch, so as to eliminate the free communication between the inferior and superior thyroid arteries.

#### THE TONSILS.

**Tonsillitis.**—The palatine tonsils are placed in an extremely exposed position, for they may be infected both by the passage of air or food over them. The air which is unnaturally drawn

in through the mouth may be highly charged with bacteria, and these micro-organisms may pass directly on to the surface of the tonsil, covered with its mucilaginous secretion. Few, if any, of these micro-organisms are deposited in their passage through the mouth. If they had passed over the mucous membrane of the devious passages of the nose, few would have entered the pharynx. Again, the passage of food through the fauces, guarded laterally as it were by the tonsils, explains the liability for these organs to meet with injury and infection.

Rapid absorption occurs from the lymphoid tissue, and the lymph carrying septic material passes to the glands of the concatenate chain, and these filters arrest the progress of the bacteria. Thus it happens that they are so frequently the site of tuberculous infection. One of the highest of these glands, lying immediately under cover of the angle of the jaw, when enlarged in tonsillitis, has been sometimes mistaken for the tonsil itself.

The enlargement of the tonsil induced by acute inflammation may seriously obstruct the entrance to the pharynx; hence it is that severe pain is caused by the act of deglutition, partly owing to the movement of the pillars of the fauces and partly to the pressure of the bolus of food upon the inflamed structures.

The sensory nerve supply of the tonsil is derived from the glosso-pharyngeal nerve, which runs downwards and forwards just external to it. The tonsil and the posterior third of the dorsum of the tongue are both supplied by the glosso-pharyngeal nerve, which may explain the chronic furring of the tongue in cases of chronic tonsillitis, but this is more likely to be due to the mouth breathing.

When acute tonsillitis terminates in suppuration, the purulent matter should be evacuated. There is very little, if any, danger of wounding any important vessel in this small operation. It is generally stated that the internal carotid artery is not far distant, but as a matter of fact even in children the amount of space between the tonsil and the vessel is very appreciable, and injury of this arterial trunk is very unlikely to occur unless the

point of the knife is plunged almost directly outwards. The vessel which in point of fact lies closest to the tonsil is the main trunk of the facial artery, and in cases where external pressure is applied just below the angle of the mandible, to push the tonsil inwards during the operation of tonsillotomy or tonsillectomy, this artery may possibly be in some degree of danger. The actual vessels from which blood flows after tonsillotomy are the tonsillar branches of the ascending pharyngeal, of the facial, of the dorsalis linguæ, and sometimes branches from the internal maxillary.

In order to engage the tonsil satisfactorily in the ring of a guillotine, it is well to remember that the front portion of the organ is overlapped to a certain extent by the anterior pillar of the fauces, containing the palato-glossus muscle, and therefore the posterior portion of the tonsil should be first caught by the instrument.

#### DISEASES OF THE ŒSOPHAGUS.

**Malformations and Diverticula of the Œsophagus.**—A complete interruption of the œsophagus a short distance above the bifurcation of the trachea may be met with. The upper end of its lower segment then opens into the trachea. This malformation does not, as was once supposed, indicate a failure of the buccal invagination of epiblast to fuse with the fore-gut, but is probably due to obliteration of part of the gullet by traction of the outgrowing lung bud, which springs from the alimentary tract in this situation. Congenital strictures may occur in the same situation and are due to the same cause.

Pressure pouches occur in the mid-line posteriorly at the junction of the œsophagus and pharynx. These diverticula are at first lodged in the retro-pharyngeal space but later present themselves at the left side of the neck. The part of the œsophagus from which they arise is weakened owing to the divergence of the longitudinal muscular fibres towards the front in the form of two lateral bands which become attached to the back of the cricoid cartilage. Although known as pressure pouches of the

œsophagus it is possible that some of these diverticula really arise in the pharynx and are due to defective closure of pharyngeal clefts.

Traction diverticula are usually found on the anterior wall of the œsophagus, near the tracheal bifurcation. They are attributed to the contraction of inflamed and adherent bronchial glands. Inflammatory adhesion of the œsophagus to the right lung has been known to have the same effect. Œsophageal diverticula are prone to ulceration, and thus communications may be established between the œsophagus on the one hand and the trachea, bronchi, pleura, pericardium or mediastinal spaces on the other.

A **pericœsophageal abscess** lies in the surrounding mediastinal tissue which is continuous above with the retropharyngeal space. The abscess may arise in connexion with caries of the vertebræ, suppurating lymph glands or inflammations and perforations of the œsophagus itself. The natural tendency of the abscess is to open into the œsophagus or pharynx, but it sometimes perforates the trachea or may be guided by the connective tissue planes towards the contiguous pleural sacs. The mediastinal abscess of caries often presents posteriorly, following the connective tissue sheaths of the posterior branches of the intercostal arteries. These sheaths are continuous with the mediastinal tissues surrounding the aorta. Retropharyngeal abscesses sometimes track through the thorax into the abdomen. By pressure of mediastinal abscesses on the vagus or air tubes a dangerous form of paroxysmal dyspnoea may be induced.

There are three places where the œsophagus is somewhat constricted and at which **malignant disease** is most likely to commence. The first place is quite at the commencement, opposite the cricoid cartilage and six inches from the incisor teeth. The second place is supposed to indicate the level at which the lung bud originally arose from the gullet; it is a short distance above the point where the left bronchus crosses the latter and lies about nine inches from the incisors. The third constriction is at the œsophageal opening of the diaphragm, at least fifteen

inches from the teeth. The actual opening of the œsophagus into the stomach is an inch lower down. The œsophagus pierces the diaphragm at the level of the tenth dorsal vertebra, at a point immediately to the left of the tip of the ninth dorsal spine.

In the neck the œsophagus is loosely connected by areolar tissue with the posterior membranous wall of the trachea; the recurrent laryngeal nerve of each side ascends in the groove between the two. The carotid sheaths and the lobes of the thyroid gland lie laterally, the relation being more intimate on the left side owing to the deviation of the œsophagus to the left at the root of the neck. A malignant growth of the cervical portion of the œsophagus may cause œdema of the larynx, simulating an inflammatory condition, or actually invade it. The trachea may be compressed or perforated; one or both recurrent nerves may be paralysed; the thyroid gland may be invaded, or, more rarely, the growth may ulcerate into the adjacent blood vessels or give rise to suppuration in the prevertebral connective tissue, the layers of which separate the œsophagus from the vertebral column and the longus colli muscles.

The thoracic portion of the œsophagus traverses the superior and posterior mediastina. The trachea lies in front above, but lower down the air tube deviates to the right and the left bronchus crosses the gullet. Either this bronchus or the lower part of the trachea may be compressed or perforated by a new growth in this situation. Below the trachea, the pericardium, and lower still the diaphragm, are in anterior relation and are sometimes invaded. The lateral relations of first importance in the thorax are the lungs and pleuræ. The œsophagus is in much more extensive relation with the right pleural sac than with the left, because the thoracic aorta intervenes between it and the latter. The former sac not only forms a continuous lateral relation, but below the level of the eighth dorsal vertebra becomes posterior as well. There is therefore a greater probability of invasion of the right pleural sac and lung than of the left. Gangrenous processes may be the outcome of such invasion, since the food passages easily convey infections. The left pleural sac is in

relation with the œsophagus in two positions, in the superior mediastinum and in the posterior mediastinum just before the tube traverses the diaphragm.

The relations of the thoracic aorta to the œsophagus vary considerably at different levels. The transverse aorta crosses it to gain its left side. The descending aorta at first lies to the left, but in the lower part of the thorax the relations of the two are gradually reversed, so that the œsophagus passes in front and finally lies even to the left of the aorta. Of the large arteries arising from the aortic arch only the left subclavian comes into relation with the œsophagus. Ulceration of a growth into the aorta or subclavian is rare. Occasionally the thoracic duct, the azygos veins or the sympathetic chain are implicated. The thoracic duct at first lies to the right, being separated from the œsophagus by the aorta, higher up it lies behind and finally crosses to the left as it enters the superior mediastinum. The left recurrent laryngeal nerve lies between the œsophagus and the transverse aorta. It may be involved here by cancer of the œsophagus.

When cancer invades the middle and lower parts of the tube the mediastinal and lumbar glands become enlarged; when the upper third is involved the glands of the mediastinum and those at the root of the neck become implicated. A gland which lies in the posterior triangle just above the left clavicle is of great significance in this respect, but the same gland may be enlarged when the malignant disease involves the stomach, pleura or lungs. Enlarged glands may actually obstruct the œsophagus at some distance from a primary carcinomatous ulcer which is causing no symptoms.

**Fibrous strictures** of the œsophagus may result from the action of corrosive poisons. They are found at the upper or lower ends as a rule. At these points the tube is narrow and the passage of fluids somewhat delayed, whilst their passage along the rest of the gullet is rapid.

**Foreign bodies** impacted in the œsophagus tend to become fixed where the tube is narrow, that is to say at either end or at a point nine inches from the incisor teeth, just above the



position of the left bronchus. The subsequent formation of a mediastinal abscess or ulceration into the aorta, pericardium, left bronchus, trachea, pleura or lung tissue are sufficiently explained by the relations of the gullet to these structures.

**Rupture** of the œsophagus is rare and is practically limited to the lower third of the tube. The rent, which is usually longitudinal, may open directly or indirectly into either pleural cavity, causing pneumothorax. Mediastinal emphysema, suppuration or hæmorrhage may also occur. The lower part of the gullet differs from the upper in that it consists of involuntary muscular fibres and is firmly fixed to the diaphragm, also simple ulceration usually occurs in the lower third of the tube. All these factors may favour rupture during the act of vomiting.

**Idiopathic dilatation** of the œsophagus has been variously attributed to pressure by a dilated aorta or hypertrophied heart or spasm of the cardiac orifice. It may, however, occur without evidence of the presence of any of these conditions, and is possibly due to paralysis of the longitudinal muscular fibres and consequent inability to open the cardiac sphincter.

#### DISEASES OF THE STOMACH.

**Gastric Ulcer.**—Since quite two-thirds of the stomach lie under cover of the liver and left costal arch only a small portion of the stomach, which includes the pylorus, can be palpated. The area of superficial tenderness may afford some indication of the position of the ulcer. The stomach is innervated from the seventh, eighth and ninth dorsal segments. The seventh segment supplies the cardiac end and is distributed to a skin field which includes the ensiform region, extending round to the back across a spot opposite the eighth and ninth dorsal spines, below the lower angle of the scapula. Superficial tenderness may be found in both these regions when the cardia is the site of ulceration. The ninth segment innervates the region of the umbilicus. The eighth segment is intermediate in position. When this segment is involved a tender spot may be found in front or in the infra-axillary region. As a general rule the nearer the superficial

tenderness is to the ensiform cartilage the nearer the ulcer to the cardiac orifice. (See Figs. 20—23, p. 226.)

The idea that the stomach is a flaccid bag, suspended by the attachment to it of the œsophagus and the gastro-hepatic omentum, is erroneous and misleading. When empty the viscus appears as a contracted pyriform tube of which the cardiac end is the larger. The tube is somewhat bent on itself at the junction of its cardiac and pyloric segments. The long axis of the cardiac portion is directed from the œsophageal opening, downwards, forwards and to the right, that of the pyloric portion is directed more transversely to the right to terminate in the duodenum. When food is ingested, the more flaccid cardiac portion probably relaxes quickly to receive it, whilst the relaxation of the thicker pyloric segment is delayed; finally the whole organ, with the exception of its terminal inch, which may be called the pyloric canal, becomes further enlarged. This explains the early onset of pain after food when the ulcer is in the cardiac portion, and the delay of onset when the pyloric portion is involved. The sharp angle in the lesser curvature, which corresponds to the junction of the flaccid cardiac and tonically contracted pyloric segments may have some influence in determining the occurrence of ulcers near this part of the lesser curvature. When hour-glass stomach occurs as the result of ulceration, the stricture is usually rather to the pyloric side of the middle of the viscus. The position of such a stricture may represent the line of junction between the cardiac chamber and the more contracted pyloric segment. The ulcers and cicatrices which result from the swallowing of corrosive fluids also tend to involve the pyloric segment of the organ. This may be explained by the fact that such fluids pass quickly through the cardiac portion to remain longer in contact with the pyloric segment, which lies at a lower level and offers more resistance to their passage.

The stomach when distended expands away from the lesser curvature, which remains more or less fixed by the attachment to it of the œsophagus, the duodenum and the left pancreatico-gastric fold. The right pancreatico-gastric fold, carrying the

hepatic artery, in a similar manner serves to anchor the duodenum and so indirectly fixes the right end of the lesser curvature of the stomach. As the stomach expands it completely fills a well-defined space in the upper and left part of the abdomen known as the stomach chamber. This chamber has a sloping floor on which the stomach rests, a dome-like roof against which the anterior surface abuts, and an anterior wall which is in contact with the anterior surface and greater curvature. When the stomach is empty a part of the transverse colon near the splenic flexure turns up into the stomach chamber.

The inflammation which accompanies gastric ulceration may extend to surrounding structures, and the stomach thus contract adhesions to different parts of the stomach chamber. Adhesions readily form between the under (posterior) surface of the stomach and the shelf on which it rests, since in this position they are well supported and not easily disturbed by gastric or respiratory movements. Adhesions between the stomach and the dome or the anterior wall of the stomach chamber are formed with less facility and more easily ruptured since they are constantly exposed to traction in consequence of alterations in the size of the stomach caused by the ingestion of food or fluid, and also by the respiratory excursions of the diaphragm, liver and anterior abdominal walls. Consequently, although ulcers of the posterior wall are more common, ulcers of the anterior wall perforate more frequently. The latter ulcers, owing to the difficulty with which firm adhesions form, are sometimes spoken of as "undefended."

The structures which form the stomach bed are the front of the left kidney and its adrenal body, the gastric surface of the spleen, the upper surface of the pancreas, the transverse colon and its mesocolon. Interposed between these structures and the posterior wall of the stomach is the lesser sac of the peritoneum, except over a small area near the cardiac end, where the stomach is in direct contact with the diaphragm above the left adrenal body. Adhesions of the posterior surface of the stomach partly obliterate the lesser sac and usually bind the stomach to the pancreas, but sometimes the spleen or other structures, such as the lymphatic

glands along the top of the pancreas and in the gastro-splenic omentum, become adherent. By an extension of the destructive process the pancreas may become exposed in the floor of an ulcer and eroded or inflamed or even suppurate. The same may happen in the case of the spleen. Posterior ulcers rarely perforate directly into the lesser sac. The roof of the stomach chamber, to which ulcers of the anterior wall may adhere, is formed by part of the under surface of the liver and the left cupola of the diaphragm. The anterior wall of the stomach chamber, against which part of the anterior surface and greater curvature of the distended stomach rest, is formed by the anterior abdominal wall between the costal margin on the left side and the liver on the right. Adhesions may occur in this area also. Perforation of an ulcer of the anterior wall of the stomach must involve the greater sac unless protective adhesions have formed and the perforation occurs into them.

It is interesting to note that a part of the under surface of the heart, near the apex, is only separated from the stomach by the pericardial sac, the left cupola of the diaphragm, and the peritoneum; in rare instances an ulcer of the anterior wall of the stomach has been known to perforate the pericardium and even the heart itself. Adhesions in the region of the pylorus have been known to obstruct the bile ducts, or attach the pylorus to the gall-bladder, inducing attacks of cholecystitis and colic.

The gastric hæmorrhage which results from ulceration may be arterial or venous. Hæmorrhage which proves fatal is nearly always arterial, a large vessel being involved. The larger vascular trunks in the wall of the stomach lie in the submucous tissue beneath the mucous membrane. The large arteries which form the vascular circle along the borders of the stomach are sometimes attacked, and erosions or small aneurysms may result. The arteries of the lesser curvature, particularly the coronary, may suffer in this way. The pancreatico-duodenal and the right gastro-epiploic arteries are, from their position, more likely to be perforated by ulcers of the pylorus or duodenum than of the stomach. The splenic artery, although not directly applied to the stomach

wall, lies in the bed of the stomach chamber, and may be opened by ulcers on the posterior wall which have contracted adhesions. The coronary veins may be eroded, or even the splenic or portal trunks, although at first sight, on account of the interposition of the pancreas, erosion of the two latter would appear impossible. Hæmorrhage from veins is uncommon, and when attacked by a gastric ulcer they are much more likely to be concerned in the production of portal pyæmia or secondary abscesses. As rarities may be mentioned perforation of the hepatic artery, of the abdominal aorta, and even of the left ventricle of the heart.

A gastric perforation is occasionally circumscribed by adhesions which have formed as the ulcer approached the peritoneal surface. In such cases a more or less localised abscess may result, and the patient escape general peritonitis. Such localised abscesses may lie in the dome of the stomach chamber between the stomach and left wing of the diaphragm, or between the stomach and its bed. They may come forwards and present in the epigastrium, being limited below by omental adhesions to the anterior abdominal wall. Fistulæ are rarely formed as the result of simple ulceration. They may appear in the epigastrium or unite the stomach with the colon, small intestine, or even gall-bladder. Rarely an adherent ulcer perforates into the subperitoneal tissues, giving rise to suppuration or emphysema.

The cicatrices left by ulcers may produce a bilocular or hour-glass stomach, and may, if situated in the pyloric region, give rise to obstruction to the exit of contents and to dilatation.

**Carcinoma of the stomach** arises from the epithelium of the gastric glands, and so may be either columnar- or spheroidal-celled. It is most frequent in the pyloric region, but may originate at the œsophageal opening or in some other part. The pylorus and adjacent part of the greater curvature are the only portions accessible to palpation. The transpyloric line (Fig. 49, p. 362), which bisects the distance from the top of the manubrium sterni to the pubic symphysis, indicates the level of the pylorus, but the weight of a tumour or the drag of a distended stomach may dislocate the pylorus considerably. The

tumour may exhibit considerable mobility or contract adhesions. The adjacent aorta may communicate pulsation to it. Being muscular it may sometimes be felt to harden and relax, and at times gastric contents are felt to bubble through it. The peristalsis of the stomach may be distinguished from peristalsis of the transverse colon by the fact that the wave of the former passes from left to right, whilst that of the latter passes from right to left. In some cases the direction of gastric peristalsis is reversed.

The arrangement of the lymphatic vessels and glands of the stomach has an important bearing on the spread of gastric carcinoma. The lymphatics commence in the submucous coat and follow the track of the vessels in the subserous tissue. Three distinct lymphatic areas may be recognised, the superior gastric, the inferior gastric, and the area of the fundus. The superior gastric lymphatics drain the upper halves of both surfaces of the stomach from the pylorus to the junction of the central third with the fundus. Their efferent trunks follow the coronary vein along the lesser curvature, lying between the two layers of the lesser omentum; they traverse the superior gastric glands which lie in this situation, and near the cardiac orifice turn backwards, in the pancreatico-gastric fold, to enter the glands of the cœliac group. They leave the stomach where the coronary artery reaches the lesser curvature. The inferior gastric lymphatics drain the lower halves of the pyloric and central regions. They traverse the inferior gastric glands of the greater curvature, and their trunks, accompanying the right gastro-epiploic vessels, pass below the pylorus into the pancreatico-duodenal fold to reach the cœliac glands. The lymphatics of the fundus accompany the vasa brevia of the splenic artery in the gastro-splenic omentum, and pass to the glands near the hilus of the spleen. These glands in turn discharge into the cœliac glands. In the lesser omentum a few lymphatic trunks pass directly from the pylorus to the glands in the portal fissure.

Carcinoma of the *pylorus* involves both upper and lower lymphatic areas. It extends chiefly along the lesser curvature,

towards the point where the lymphatic trunks leave the stomach. It shows little tendency to pass beyond the pylorus to the duodenum, the lymphatic communication not being at all free in this situation. Extension along the greater curvature is slight, since the lymphatic trunks soon pass away to the cœliac glands. The glands in the portal fissure may be directly implicated and compress the portal vein and bile ducts.

Carcinoma of the *greater curvature* infects the inferior gastric glands and may cause diffuse infiltration of the great omentum or perforate the transverse colon, causing a gastro-colic fistula. Sometimes it compresses the vessels of the transverse colon, causing local necrosis of this portion of the bowel.

Carcinoma of the *cardiac orifice* can only infect one or two glands of the lesser curvature, since the lymphatics of this region soon reach the cœliac glands. The growth may invade the œsophagus and spread along it in longitudinal columns corresponding to the course of the lymphatics. There is a free communication between the subserous and submucous lymphatics of the stomach and the œsophagus.

Enlarged cœliac glands sometimes form a nodular tumour. They are in a position to receive pulsation from the aorta, occlude the inferior vena cava, or invade the spinal column. They are early infected in gastric carcinoma, and any radical operation should include their removal. Their ducts join with those from the intestines and discharge into the receptaculum chyli which lies on the right side of the second lumbar vertebra under cover of the right crus of the diaphragm. By backward propagation the lumbar, mesenteric, sacral, iliac and even the inguinal glands may be involved. Forward infection invades the thoracic duct, the glands of the posterior and superior mediastina and even those at the root of the neck, particularly one on the left side above the clavicle, known as Virchow's gland.

Miliary carcinomatous deposits may occur on the under surface of the diaphragm, and in the superficial lymphatics of the liver, being derived from the lymphatics of the portal fissure. The diaphragm may be traversed and the right pleura invaded

Large secondary growths in the liver are due to infection carried by the portal vein direct.

Carcinoma of the stomach has a tendency to become adherent to neighbouring parts, and invasion through the medium of the adhesions may occur. The pyloric branch of the hepatic artery is sometimes eroded.

The gastric growth soon ulcerates through the mucous membrane, and presents as a foul ulcer owing to its exposure to the gastric contents. A diffuse infiltration of the stomach wall may occur, giving rise to what is known as the "leather bottle stomach." Growth at the pylorus is in a position to cause obstruction, and so lead to dilatation, whilst growth at the cardia may hinder the entry of food into the stomach, and considerable atrophy of the organ result.

**Dilatation of the stomach** is due to various causes. Acute dilatation is a form which comes on abruptly, and is very fatal, but is fortunately rare. In atonic dilatation the muscular wall is at fault. Habitual over-distension with food or drink may have the same result. Obstructive dilatation may be due to changes in the walls of the pyloric portion, or in the duodenum or to pressure from without. The pyloric sphincter involves nearly an inch of the stomach wall adjacent to the pyloric orifice, and is capable of great dilatation. Foreign bodies with a diameter of over three-quarters of an inch have passed through it. Malignant disease, cicatrices, and benign growths may narrow its lumen. It is sometimes the seat of congenital hypertrophy, which also obstructs the exit.

Since the pylorus rests on the neck of the pancreas and is overlapped by the liver, it is not surprising that it is occasionally obstructed by tumours of these organs. It also lies in close relation with the under surface of the gall bladder, and as the result of cholecystitis or of pyloric ulceration the two may become adherent. The pressure exerted by a movable and displaced kidney upon the duodenum or pylorus may also give rise to intermittent obstruction to the exit of gastric contents. Obstruction may be caused by tumours of the omentum,



enlarged mesenteric or retroperitoneal glands, or even by aneurysms of the abdominal aorta or celiac axis.

Very diverse estimates as to the normal capacity of the stomach are current. It may perhaps be fixed as being from thirty-five to forty ounces in the adult. In children up to six months of age the gastric capacity may be approximately stated to be an ounce more than the age of the child in months; at birth it is barely an ounce, at one month the capacity is two ounces, and at six months it is seven ounces. At twelve months, however, the capacity is but nine ounces, and at eighteen months it is twelve ounces.

Dilatation of the stomach is inferred from an increase in its superficial area, as determined by physical examination. It is important to remember that when the stomach is contracted a part of the transverse colon may lie in front of it. Sometimes the actual outline of the stomach can be seen; at others it must be determined by auscultatory percussion. Inflation is often necessary, and the valvular mechanism of the cardia, coupled with the obstruction at the pylorus, renders this easy of accomplishment by effervescing mixtures or by an air pump and stomach tube. Since the cardiac end of the dilated stomach accurately fits the left dome of the diaphragm, the fundus of the distended stomach rises to the same level as this dome, and lies a little above and internal to the position of the cardiac impulse. The proximity of the stomach to the heart is held accountable for the palpitation and dyspnoea sometimes produced by gastric distension. The cardiac sounds may be echoed by the air-containing stomach, and its encroachment on the base of the lung may simulate pneumothorax in childhood.

The greater curvature of the stomach emerges from beneath the left costal margin at the level of the ninth costal cartilage (corresponding to the position of the gall bladder on the opposite side). The lower border of a normal stomach when inflated should not extend lower than a line joining the tips of the tenth costal cartilages, or two inches above the umbilicus. (Fig. 49, p. 362.)

The position of the pylorus is best defined by the transpyloric line. This is a horizontal line which bisects the distance between the episternal notch and the top of the pubic symphysis. The pylorus normally lies in this line, being practically mesial if the stomach is empty but lying two or three inches to the right if the viscus is distended.

The weight of the dilated stomach tends to depress that part of the stomach bed which is formed by the transverse colon and mesocolon. The stomach may thus indirectly exercise enough pressure on the duodeno-jejunal flexure or the terminal portion of the duodenum to obstruct the passage of its own contents.

**Prolapse of the stomach, or gastroptosis,** may also be a cause of dilatation, a sharp bend being formed at the superior duodenal flexure through the agency of the peritoneal ligaments attached at this spot. The stomach is normally retained in its position in the stomach bed not only by the support of the muscular walls of the abdomen and the structures which form the bed, but also by strong ligamentous bands. At each end of the lesser curvature vertical and horizontal ligamentous supports are attached. The left support is formed by the firm attachment of the cardiac end to the diaphragm in the neighbourhood of the œsophageal opening, by the adjacent gastro-phrenic omentum which passes vertically, and by the left pancreatico-gastric fold, which is horizontal. The pancreatico-gastric fold is continuous with the gastro-phrenic omentum and conveys the coronary artery to the stomach. The right support is also a double fold; this is attached not to the stomach but to the first curvature of the duodenum. It consists of the vertical hepato-duodenal ligament, which is the right free edge of the lesser omentum and the horizontal pancreatico-duodenal fold which passes from the region of the head of the pancreas forwards to join the duodenum at the same point as the hepato-duodenal ligament, the two becoming continuous. The central portion of the lesser omentum is of extreme tenuity and obviously of no importance as a gastric support. The main factors are the strong ligaments

## TOPOGRAPHY OF ABDOMEN.

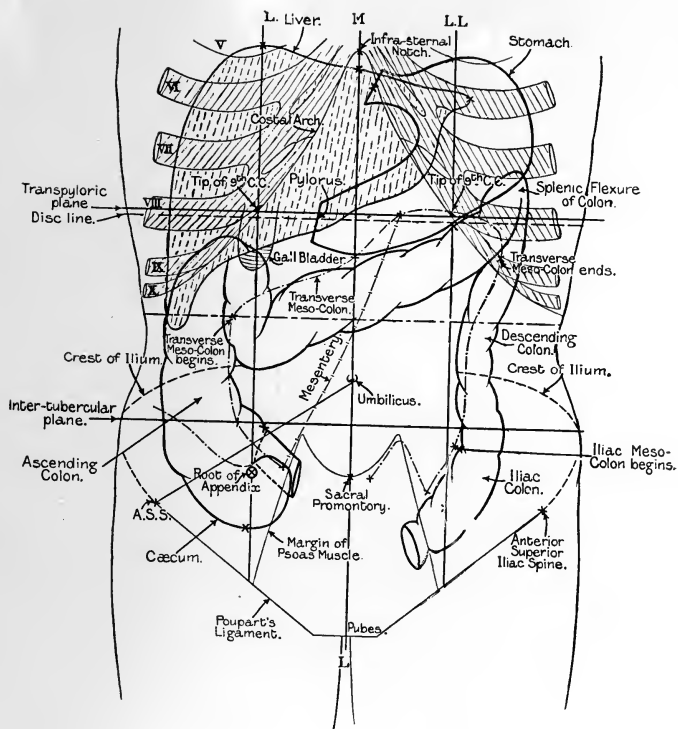


FIG. 49.—Diagram showing the disposition of the liver, the stomach, the large intestine and the lines of peritoneal attachment in the regions of the abdomen. M.L., middle line; L.L., lateral lines; Disc line represents the level of the disc between the first and second lumbar vertebra. (From Ellis's Anatomy, after Christopher Addison.)



already described. The two horizontal portions of these ligaments are easily demonstrated by opening the lesser sac below the stomach and turning that viscus upwards. The folds can then be seen to spring from the posterior abdominal wall in the neighbourhood of the cœliac axis, and skirting the orifice of the Spigelian recess of the lesser sac of the peritoneum, pass forwards, just above the pancreas, to the cardiac end of the stomach and to the duodenum about an inch beyond the pylorus. The coronary and the hepatic arteries can be felt in the substance of the folds.

The sharp V-shaped bend which develops in the lesser curvature of a prolapsed stomach is due to its being slung up by its two ends. The lesser curvature in such cases, instead of lying under cover of the liver, may be distinctly visible, and sometimes a part of the pancreas can be felt above it, simulating a malignant tumour. In extreme prolapse the stomach may form a prominence below the level of the umbilicus.

#### DISEASES OF THE INTESTINE.

**Duodenal Ulcer.**—The first part of the duodenum is the commonest site for ulcers. This part of the bowel begins at the pylorus, opposite the first lumbar vertebra. With the stomach empty, the duodenum is directed first to the right and then backwards beneath the liver, but with the stomach full it passes directly backwards. It is therefore very movable. Its relations have important bearings on the complications which may arise. The quadrate lobe of the liver lies above and in front of it. The head and neck of the pancreas support it. Behind it the portal vein passes upwards to the liver, and the gastro-duodenal artery and bile duct pass downwards, the artery lying about an inch to the right of the pylorus. The termination of this part of the duodenum lies immediately to the right of the inferior vena cava. The relations to the peritoneum are exactly like those of the stomach. A diverticulum of the lesser sac passes to the right behind the bowel, whilst the remainder of its circumference

is in relation with the greater sac. Duodenal ulcer, however, like gastric ulcer, does not tend to perforate into the lesser sac and probably for similar reasons, *i.e.*, the formation of adhesions which obliterate the small posterior diverticulum. Ulcers of the first part have, however, a great tendency to perforate anteriorly into the greater sac and to produce either general peritonitis or a localised collection of pus in the right kidney pouch or subphrenic space. Various vessels have been known to be eroded. Posteriorly the portal vein, the hepatic artery, or its gastro-duodenal branch may suffer. The superior pancreaticoduodenal branch of the latter has been perforated, and ulceration has been known to extend sufficiently to the left to open branches of the splenic artery, and even the aorta. (Fig. 52, p. 416.)

The second part of the duodenum is sometimes the site of ulceration, which is usually above the level of the biliary papilla. This part lies on the right side of the spine, and reaches as low as the third or even the fourth lumbar vertebra. The transverse colon crosses its middle; it comes into contact above with the neck of the gall bladder and below with the coils of small intestine; any of these may be opened by an ulcer, and a bimuscular fistula result. Posteriorly it is in relation with the inner part of the right kidney, with its vessels and ureter; but these are not known to suffer. Moulded on its right or convex aspect is the duodenal impression of the liver, and below this the hepatic flexure of the colon. Its concave left aspect is closely applied to the head of the pancreas, and is in intimate relation with the bile and pancreatic ducts, the papilla of Vater, or common orifice of these ducts, being found three and a half or four inches beyond the pylorus. Ulcers near the papilla are of especial importance, since they may cause complete cicatricial occlusion of both the bile and pancreatic ducts, giving rise to very characteristic symptoms. The inferior vena cava is in close relation with the duodenum behind the head of the pancreas. The second part of the duodenum is only covered by peritoneum in front and on its convexity. When there is no transverse mesocolon, the transverse colon may be in direct contact over a

considerable area. The bearing of the peritoneal and colic relations on the possibility of perforation into the greater sac and into the transverse colon is obvious.

As the result of duodenal ulceration cicatricial stenosis may occur, or the presence of dense external adhesions may cause considerable obstruction.

**Pressure** on the duodenum sometimes leads to obstruction. The pressure may be due to a movable right kidney, and is then intermittent. A stomach much distended with fluid may exercise pressure on the terminal portion of the duodenum which lies behind it. Collapse of the small intestine may cause the superior mesenteric vessels to become tightly drawn across the transverse portion of the duodenum, and may thus obstruct it.

**Malignant disease** of the duodenum is very uncommon. A carcinoma of the first part appears as a movable tumour, with symptoms very like those of pyloric cancer. If the obstruction is considerable no regurgitation of bile is possible. Carcinoma is more likely, however, to arise in the neighbourhood of the papilla of Vater. The tumour is then fixed, even in its early stages, for this part of the duodenum is not movable, being retroperitoneal. Obstruction of the biliary and pancreatic ducts will complicate the intestinal obstruction which results. Retroperitoneal growths arising in the lymphatic glands sometimes encircle and infiltrate the retroperitoneal portion of the duodenum, and may compress the bile duct and pancreatic duct at the same time.

**Malformations** of the duodenum are rare. When the duodenum is prolapsed, traction by the bile duct may draw out a small diverticulum. Diverticula may also arise from ulceration and yielding of the wall. A diverticulum sometimes projects above the biliary papilla and passes towards the head of the pancreas; this has been attributed to the influence of one of the outgrowths from which the pancreas is developed. External adhesions at the base of an ulcer may also drag out the duodenal wall in the form of a pouch.

**Rupture** of the duodenum usually results from the passage

of a wheel across the abdomen, whereby this portion of the bowel is subjected to a shearing force. Being fixed on the front of the spine, it cannot slip away, and is lacerated. The rent may be entirely extraperitoneal.

**Malignant disease** of the *small intestine* is rare. When a distinct tumour is present, and this is more likely to be the case with sarcoma than with carcinoma, the length of the mesentery may allow the growth to gravitate towards the lower part of the abdomen. It may become adherent in this position, and by ulceration a fistulous communication may be established with some part of the colon, the urinary bladder, or other hollow viscus. Pedunculated growths in the interior of the jejunum or ileum sometimes give rise to extensive chronic intussusceptions, an occurrence which is favoured by the length and freedom of the mesentery.

Malignant disease of the *colon* usually occurs in the form of an annular carcinomatous infiltration leading to stricture. Occasionally it is in the form of a tubular infiltration. In both cases the interior, being exposed to the passage of the bowel contents, tends to ulcerate and become septic. Malignant disease is prone to arise in those parts of the colon which present abrupt bends or are exposed to pressure by bony edges. It is often met with on the brim of the true pelvis, or opposite the crest of the ilium; it is less common at the splenic or hepatic flexures, in the transverse colon or the cæcum. (Fig. 49, p. 362.)

The growth opposite the brim of the true pelvis lies at the junction of the iliac and pelvic portions of the colon. It may become adherent to the left iliac vein, causing œdema of the left leg, or compress the left ureter, giving rise to left renal colic and hydronephrosis. Since the ovarian fossa and ovary are close by, it is not surprising that the growth may become adherent to the latter. Hæmorrhoids may occur from obstruction to the veins in the bowel wall. Perforation at the site of the growth will give rise to a pelvic abscess or a spreading general peritonitis.

The growth opposite the iliac crest on the left side occurs at



the point of junction of the descending and iliac portions of the colon.

Carcinoma at the splenic flexure cannot as a rule be palpated, since this part of the colon is deeply situated under cover of the ribs. Any tendency to prolapse of the growth is counteracted by the phrenico-colic ligament. The growth is in close relation with the spleen and the front of the left kidney.

Similar considerations apply to growth at the hepatic flexure. The growth lies deeply, and the hepato-colic ligament which is occasionally present may keep it up. It is in relation with the right lobe of the liver, the gall bladder, and the duodenum.

Carcinoma of the transverse colon is usually associated with prolapse of this part. The average length of this segment of the large bowel is twenty inches, and the length of the mesocolon is five inches from origin to attachment. The prolapsed growth tends to become adherent to the pelvic colon, the urinary bladder, or the uterus. Occasionally it becomes adherent to the stomach, to the lower border of which the transverse colon is closely applied.

Carcinoma of the cæcum is rare, and commences in the neighbourhood of the ileo-cæcal valve. It may become adherent to the external iliac vein or the right ureter. Carcinomata of the ascending or descending colon or cæcum occasionally ulcerate into the retroperitoneal tissue, since these parts of the bowel may be devoid of a complete peritoneal investment. Carcinoma at the junction of the iliac and pelvic segments of the colon may do the same.

Secondary deposits may be expected in the lymphatic glands and also, since this part of the bowel belongs to the portal area, in the liver. In some cases the general circulation is invaded, and metastatic deposits occur in the lungs, bones, and other parts. Involvement of the glands is slow. The lymphatics of the ilio-pelvic colon terminate in glands around the inferior mesenteric artery. The lymphatics of the transverse colon and its two terminal flexures are numerous, and pass alongside the middle colic artery to the glands of the

superior mesenteric chain. They communicate freely with the lymphatics of the great omentum, and so come into relation with the lymphatics of the lower border of the stomach. The lymphatics of the ascending and descending colon also gain the superior mesenteric chain. The glandular apparatus of the descending colon is very poorly developed, which may account for the slowness with which glandular infection occurs when the disease is in the lower parts of the bowel.

Malignant disease of the *rectum* is more common than in any other part of the alimentary tract. It may either originate in the glandular epithelium of the crypts of Lieberkühn or in the squamous epithelium of the anal canal. Squamous epithelium extends up to the lower ends of the columns of Morgagni, and, according to some authors, along these columns also. Local extension of rectal growths is liable to involve important pelvic structures, for the prostate, urinary bladder, vesiculæ seminales, and terminations of the vasa deferentia, lie in the closest contact with the anterior rectal wall. In the female, the vagina and uterus are apt to be invaded, and fistulous communications may be thus established. By backward extension rectal growths become adherent to the sacrum and coccyx.

**Ulceration of the Small Intestine.**—*Tuberculous* ulcers commence in the Peyer's patches or the solitary follicles. These ulcers tend to extend transversely round the bowel, following the course of the lymphatic vessels. The lymphatic collecting trunks in the mesentery sometimes become caseous, and can be traced to the mesenteric glands, which may also be infected. Since both the Peyer's patches and the solitary follicles are best developed in the lower ileum, the ulcers are best marked in this region. They are especially common just above the ileo-cæcal valve, owing to the obstruction offered by that structure to the onward passage of the intestinal contents.

*Typhoid* ulcers also originate in the lymphoid structures, and are best marked in the lower part of the ileum. They are produced by sloughing or necrosis of Peyer's patches and follicles, and have not the same tendency to extend transversely

that tuberculous ulcers exhibit. An important collection of lymphatic glands lies around the termination of the superior mesenteric artery at the ileo-cæcal angle. These glands are involved secondarily to ulceration of the lower part of the ileum, and by suppuration may give rise to peritonitis, since they are immediately beneath the peritoneum.

In *summer diarrhœa* and various *infectious fevers* other than typhoid, considerable swelling and even ulceration of the lymphoid tissue of the intestine may occur.

Ulcers of *vascular* origin, girdling the intestine, are sometimes met with as the result of embolism, thrombosis, syphilis, and amyloid disease. The distribution of the arterial twigs which supply the intestine is the basis of the transverse arrangement of these ulcers.

**Ulceration of the Large Intestine.**—Although there are no Peyer's patches in the large bowel, lymphoid tissue is plentiful, particularly in the cæcum and vermiform appendix. Ulcers may arise in this tissue in many infective conditions, such as typhoid fever, tuberculosis, and dysentery. So-called stercoral ulcers are particularly apt to form in the cæcum as the result of chronic obstruction of the bowel, and certain metallic poisons, like mercury and antimony, may, during the process of excretion by the colon, originate ulceration.

#### INTESTINAL OBSTRUCTION.

(*Other than that due to Malignant Disease.*)

**Strangulation by a band** is an accident which is almost exclusively limited to the small intestine, this part of the bowel being more easily ensnared than the colon owing to its greater mobility, smaller calibre, and coiled arrangement.

Bands are usually adventitious structures which have resulted from past inflammation or operation. They occur most frequently in the lower part of the abdomen, in connection with the vermiform appendix, the hernial orifices, the Fallopian tubes, and

the mesenteric glands. The parts of the small intestine most likely to become entangled are those which lie in relation with these structures.

A Meckel's diverticulum sometimes acts as a band, producing obstruction. The diverticulum, which is the remains of the vitelline duct, usually arises from the small intestine within three feet of the ileo-cæcal valve. It may retain its connection with the umbilicus, or its extremity may be free or acquire adhesions to other structures.

**Strangulation in a peritoneal slit** is sometimes observed. The aperture is usually in the lower part of the mesentery of the ileum, and is surrounded by an arterial arch formed by the ileo-colic branch of the superior mesenteric artery and the lowest of the vasa intestini tenuis. Slits may also occur in other broad peritoneal folds, such as the great omentum, the broad ligaments, the falciform ligament, the phrenico-colic ligament, or even the mesentery of the appendix.

**Diaphragmatic hernia** may be congenital, or acquired. The *congenital* variety is due to persistence of the original communication between the pleural and peritoneal cavities. The opening is in the dorso-lateral region of the diaphragm, and usually occurs on the left side, since the presence of the liver on the right side aids the closure of the right communication. The serous membranes of the two cavities being continuous, the extruded viscera lie free in the pleural cavity. The stomach usually passes into the cavity, dragging in the colon and spleen by virtue of their omental connections. The liver and pancreas sometimes enter as well. The displacement of the stomach may lead to acute torsion of the small omentum and partial strangulation.

The *acquired* forms may be traumatic or spontaneous. *Traumatic* hernia is due to wounds or ruptures of the diaphragm. *Spontaneous* diaphragmatic hernias have definite sacs. They may pass through the œsophageal opening or between the sternal and costal portions of the diaphragm, never, it is said, through the openings for the aorta or vena cava. In enteroptosis, the œsophageal

opening of the diaphragm may be so displaced downwards that it actually encircles part of the stomach below the œsophagus.

**Duodenal hernia** may occur in two forms, the right and the left. Of these the *left* hernia occupies a peritoneal fossa which lies to the left of the duodeno-jejunal junction. The mouth of the sac contains in its left margin the inferior mesenteric vein and usually the left colic artery. These two vessels form by their intersection the vascular arch of Treitz. The hernial sac burrows in the retroperitoneal tissue in a direction outwards or outwards and upwards. In extreme instances the whole of the small intestine may lie in the sac. When the opening of the sac is in view only the issuing coil of intestine can be seen; the entering coil being entirely concealed, appears to gain the interior by perforating the sac wall. As the hernia extends in the retroperitoneal tissue it passes behind the attachment of the ascending and descending colon to the parietal peritoneum, so that the colon becomes closely applied to the front of the sac. Behind the sac lie the psoas and the kidney with the renal vessels. The compression to which the inferior mesenteric vein is exposed at the mouth of the sac may give rise to hæmorrhoids and bleeding from the bowel.

The *right* duodenal hernia is so called because it lies to the right of the spine. The orifice of the sac lies behind and to the left, on the lumbar vertebræ. The hernia appears to originate in a small fossa in the mesentery of the upper part of the jejunum, lying immediately below the transverse portion of the duodenum. The orifice of the sac lies within the concavity of the curve formed by the superior mesenteric artery as that vessel arches downwards and to the right. The aperture may be dragged downwards to the right iliac fossa by the weight of the intestine, and so confounded with hernial orifices in this region. As the hernia develops it establishes relations with the back of the ascending and transverse colon.

**Hernia into a fossa about the cæcum** is either ileo-cæcal or retro-colic. The ileo-colic fossa is not known to be the site of hernia. The ileo-cæcal fossa lies between the mesentery of

the appendix behind and the ileo-cæcal fold in front. Small intestine may become entangled in this fossa, and the appendicular artery then lies behind the mouth of the hernial sac. Hernia into the retro-cæcal fossa usually goes by the name of retro-colic, since the sac lies behind the lower part of the ascending colon. The outer boundary of the fossa is known as the retro-colic fold or suspensory ligament of the cæcum. The inner boundary is a downward prolongation of the mesentery of the small intestine towards the iliac fossa, which is sometimes called the inferior ligament of the cæcum, and may reach to the inguinal canal or the broad ligament.

**Intersigmoid hernia** lies in a pouch behind the root of the mesentery of the pelvic colon. The common iliac artery and the ureter lie behind the mouth of the fossa.

**Hernia into the foramen of Winslow** has been met with. Some part of the small intestine or colon passes through the foramen into the lesser sac of the peritoneum, and is strangulated. The stomach lies in front of the sac, and the presence of the hepatic artery, portal vein, and bile duct in the constricting ring may render relief of the condition impossible.

Gross congenital malformations of the peritoneum are often associated with the different forms of internal hernia. Indeed, the occurrence of hernia into the foramen of Winslow is impossible apart from such abnormalities. Either the cæcum and ascending colon fail to migrate in the usual manner towards the right iliac fossa, or an abnormal length of mesentery, or a mesentery common to great and small bowel is present.

A **volvulus** or **twist** is only likely to occur in those parts of the bowel which are provided with a long fan-shaped mesentery. This condition is normally fulfilled by the pelvic colon and the coils of small intestine, but as an abnormality the ascending colon may have an unusually long mesocolon, or there may be a common mesentery for the small intestine, cæcum, and a variable length of the colon. The pelvic colon is the part usually twisted; the mesentery is of the required shape, and the bowel is constantly changing its position with the varying

distension of the bladder and rectum. A volvulus occasionally involves the small intestine.

The cæcum may be obstructed in consequence of its becoming acutely flexed on the colon; the colon and cæcum may become twisted round their longitudinal axis, or the ascending, transverse, and descending colon may form one large volvulus. Strangulation is not so acute when the intestine is merely bent at an angle instead of becoming twisted on its mesenteric axis. In acute strangulation the vessels are obstructed, the bowel becomes greatly congested and swollen, and the peritoneal coat, which is the least distensible, is apt to tear. Peritonitis is an early complication.

**Intussusception** usually occurs in the neighbourhood of the cæcum, but it is also found in the jejunum, ileum, colon, and rectum. It appears most likely to occur where a part of the intestine which is more or less free joins a part which is comparatively fixed; consequently the favourite sites for intussusception are the cæcal region, the lower end of the ileum, where that becomes comparatively fixed before entering the colon, and the junction of the pelvic colon with the rectum. In the ileo-cæcal variety, which up to recent time has been held to be the commonest, the ileo-cæcal valve, followed by the small intestine, becomes prolapsed into the colon. In the ileo-colic form the lower part of the small intestine becomes prolapsed and engaged in the jaws of the valve. This form rarely attains a great size of itself, but the end of the ileum, tightly gripped in the jaws of the valve, is apt to form the starting-point of a new intussusception of the ileo-cæcal form. A compound intussusception results, and this is now said to be the commonest variety. Another form of compound intussusception may arise when an enteric invagination reaches the valve, and, being unable to pass through it, pushes it on in front. Whenever the ileo-cæcal valve is prolapsed the cæcum and appendix become drawn into the receiving sheath, the general rule being that intussusceptions increase at the expense of the receiving sheath. The mesentery being also drawn into the sheath by the portion of intestine to

which it is attached, intense engorgement of the intussuscepted bowel occurs, leading to the passage of blood and mucus, and sometimes to sloughing of the intussusceptum. The traction on the mesentery also confers a characteristic curve on the intussusceptum, and may cause its orifice to be tilted against the side of the receiving layer. Complete obstruction of the bowel or sloughing of the receiving layer at the point of pressure may result. The tumour, consisting as it does of muscular bowel, may sometimes be felt to harden and relax during examination. The highly œdematous and inflamed apex of an enteric intussusception may easily be mistaken for the ileo-cæcal valve. A polyp is often the starting-point of an intussusception in the adult, and in childhood inflamed Peyer's patches or invaginated saccules of the colon are believed to act in a similar manner.

Intussusceptions during their progress follow the direction of the colon, and in some cases protrude from the anus. For an intussusception arising in the cæcal region to do this it is obvious that either an extensive mesocolon must be present, or the colon and its mesentery must be stripped away from the posterior abdominal wall.

A moment's reflection will show that the easiest way to reduce a tight intussusception is by compression from below aided by traction of the outer layer downward over the intussusceptum, thus causing reduction by gradually unrolling the outer tube in the reverse way to that in which the invagination was brought about. Upward traction on the intussuscepted layer causes the unfolding to occur at the apex of the entering tube; this process is resisted, and tends to throw the inner tube into folds, further wedging it in the bowel. But intestine prolapsed through the ileo-cæcal valve usually has to be reduced by upward traction.

**Obstruction of the Bowel by Gall Stones.**—A gall stone large enough to become impacted in the bowel must of necessity have entered it by ulceration, and not by passage along the bile ducts. The relations of the gall bladder render it possible for a stone to enter either the duodenum or the colon in this way, but entry into the duodenum is by far the commoner and more serious



occurrence. Once in the lumen of the bowel the point of impaction is largely determined by the size of the stone, for there is a gradual diminution in the calibre of the intestine from the duodenum to the ileo-cæcal valve. The ileo-cæcal valve itself may offer considerable resistance to the passage of a stone. A stone in the colon may become impacted in the pelvic segment or near the anus, the calibre of the pelvic colon and rectum being much less than that of the upper portions of the large bowel. Comparatively small stones may induce obstruction by setting up spasm of the muscular coat of the bowel. A gall stone by its weight may drag down the coil of small intestine which contains it, and even be felt in the pouch of Douglas.

Impaction is not the only way in which gall stones produce obstruction. A volvulus may result from their presence in the bowel. Adhesions of the gall bladder may obstruct the bowel or the obstruction may be due to acute peritonitis.

**Cicatricial strictures of the intestine** arise in various ways. Tuberculous ulcers tend to encircle the gut, and strictures may result in the lower part of the ileum, where lymphoid tissue is very abundant and tuberculous ulcers, consequently, are commonest. Dysenteric ulcers frequently cause stricture. Strictures are met with in the rectum, pelvic colon, and sometimes at the hepatic and splenic flexures, all of which are positions in which the bowel wall presents sharp bends. Cicatrices may also occur in those parts of the bowel which have been strangled in hernial orifices.

**Compression by adhesions** usually affects those segments of the intestine which are the least movable, such as the ascending and descending colon and the hepatic and splenic flexures.

#### CONGENITAL OBSTRUCTIONS OF THE FOOD PASSAGES.

Congenital occlusion of the *œsophagus*, gives rise to vomiting, choking, inanition, and hunger in the new born. The obstruction cannot be due to a failure of the oral depression to meet the anterior end of the primitive gut, for it lies below the pharynx,

which is itself a derivative of the fore gut. The occlusion is usually just below the level of the cricoid cartilage, and is determined by the outgrowth of the lung bud from the primitive intestinal tube. A fistulous communication between the œsophagus and trachea frequently exists below the obstruction. Rarely the œsophagus is stenosed just above its entrance into the stomach.

Hypertrophic stenosis of the *pylorus* is probably a congenital condition, and is characterised by a great hypertrophy of the muscular coat of the pylorus and pyloric canal.

Occlusion or obstruction of the *duodenum* is sometimes found in the neighbourhood of the common bile duct, and may be due to the outgrowth of the liver, or pancreas buds in this position.

Stenosis of the *duodeno-jejunal flexure* is rare. It has been attributed to developmental changes in the peritoneal folds of this region.

Occlusion or stenosis of the *ileum* may be found at the site of Meckel's diverticulum.— This diverticulum arises within three feet of the ileo-cæcal valve, and is the residue of the vitelline duct. It may or may not retain its connexion with the umbilicus.

Multiple congenital strictures of the small intestine occur rarely. No satisfactory explanation of these has been offered.

Congenital dilatation of the *colon* is associated with obstinate constipation, and may be met with without organic obstruction of the rectum or anus.

**Malformations of the rectum** include congenital strictures, imperforate conditions, absence of the anus or the rectum, or both those structures, and openings of the rectum in abnormal situations.

An obstruction may be found at the junction of the anus and rectum in the form of a membranous fold. The fold is probably a remnant of the cloacal membrane. The anal valves, which lie at the lower ends of the columns of Morgagni, represent the cloacal membrane, the fusion of anus and rectum corresponding to the white line in this situation.

Congenital strictures of the rectum sometimes assume a

cylindrical form. These may arise in connexion with the out-growth of the allantois from the bowel, an explanation which brings them in line with similar strictures of the œsophagus, duodenum, and ileum.

Occasionally the rectum ends blindly two inches or more above the anus. The mere persistence of the cloacal septum is insufficient to account for so much separation of the two tubes. In such cases either a part of the rectum has been obliterated, and may still be represented by a fibrous cord, or the condition may be due to a complete failure in the down-growth of the post-allantoic portion of the gut.

If the rectum retains its connexion with the allantois, and the anal canal is not formed, the bowel may open into the bladder or upper part of the vagina. Abnormal communications with the urethra, or openings in the region of the vulva, indicate that the bowel has retained its communication with the front part of the uro-genital sinus. Communication of the rectum with a spina bifida suggests the persistence of a neurenteric canal.

Discharge of bowel contents from the *umbilicus* are in some cases due to a wound of a prolapsed coil of intestine during division of the umbilical cord. In other instances a Meckel's diverticulum is present and patent.

It has been suggested that a coil of bowel is sometimes ensnared in the umbilical ring during fetal life and so obliterated.

## APPENDICITIS.

The situation of the local tenderness and the local swelling caused by **appendicitis** will depend upon the position which the appendix happens to occupy.

The spot at which the vermiform appendix springs from the inner and back part of the cæcum may be indicated with sufficient accuracy by taking a point one inch below the intersection of the right Poupart and the intertubercular planes. (Fig. 49, p. 362, also Fig. 54, p. 429.) The "lie" of the appendix itself is inconstant, and cannot be predicted with accuracy.

In most cases the appendix lies internal to the long axis of the cæcum, but sometimes it is behind or to the outer side of it. When it is to the inner side of the cæcum it may lie under cover of the lower end of the ileum and mesentery of the small intestine, or be coiled up on the pelvic brim, or hang over into the cavity of the true pelvis. If it is behind the ileum and its mesentery, which is not an uncommon position for it to occupy, these structures will have to be raised in order to expose it, and the peritonitis which results from its inflammation is very apt to spread widely amongst the coils of the small intestine. In such cases the local swelling and tenderness will be felt in the right iliac fossa internal to the position of the cæcum. Inflammation of an appendix which lies coiled on the pelvic brim is in a favourable position to infect both the pelvis and the right iliac fossa; the inflammatory mass should be felt superficially just above Poupart's ligament. If the appendix hangs down over the brim of the true pelvis, the peritonitis which results from its inflammation involves the structures in the pelvis, and the inflammatory mass may only be detected by rectal or vaginal examination.

When the appendix is not internal to the long axis of the cæcum, it may be found in a retrocæcal pouch, or may extend upwards behind the ascending colon or lie in the sulcus between the cæcum and the right flank. An appendix in a retrocæcal pouch may give rise to a localised and deep-seated abscess which is difficult to find. The difficulty is even greater when the appendix is actually bound down behind the ascending colon. In such cases it has probably become entangled during the descent of the cæcum, and imprisoned by the normal adhesion of the mesocæcum to the abdominal wall. The fact that all the longitudinal bands of the colon converge to the root of the appendix may afford considerable assistance to the surgeon in his search for an abnormally placed appendix. When the appendix lies behind the cæcum or colon, tenderness on deep pressure may often be found, not on the front of the abdomen, but immediately above the iliac crest posteriorly. In such cases

the retroperitoneal connective tissue may become invaded during the course of the disease. When the appendix lies in the peritoneal sulcus between the cæcum and the flank, the inflammatory mass may be found in the right loin, and is favourably placed to infect the right kidney pouch of the peritoneum and extend into the right subdiaphragmatic region.

In rare instances the cæcum fails to complete its migration from the umbilical region to the right iliac fossa. In such cases the abscess resulting from appendicitis may lie in the region of the umbilicus or neighbourhood of the right kidney. If the descent of the cæcum fails to stop at the proper point both cæcum and appendix may be found in the true pelvis. When the cæcum is loosely attached to the abdominal wall the appendix may wander into the left iliac region. It has also been found in the sacs of umbilical, inguinal, or femoral herniæ. Inflammation of an appendix in any of these abnormal situations may give rise to great difficulties in diagnosis.

The vermiform appendix is essentially a lymphoid organ. It abounds in lymphatic tissue and closely resembles the tonsil. This, together with the fact that it is a blind tube, explains its proclivity to inflammation and perforation. In common with other lymphoid aggregations, a certain amount of atrophy occurs after middle life. Appendicitis is most common before this retrogression occurs. The narrow lumen of the tube is easily obliterated as a consequence of inflammation, and retention cysts may arise as a consequence.

The lymphatics of the appendix, which arise in the lymph sinuses surrounding the lymphoid nodules, after traversing some insignificant glands in the meso-appendix, terminate in a group of glands near the ileo-cæcal junction, round the terminal branch of the superior mesenteric artery. These glands may form part of the inflammatory swelling in appendicitis. Suppuration in them is quite the exception. A group of retroperitoneal glands along the inner border of the ascending colon is frequently enlarged as well, as also are some glands which lie alongside the right iliac arteries. An anastomosis

between the lymphatics of the appendix and those of the right ovary has been described but needs confirmation. The appendix receives its blood-supply from the appendicular artery. This small vessel is derived from the terminal branch of the superior mesenteric, and usually passes behind the termination of the ileum to reach the meso-appendix. Running along the free edge of this fold it distributes a series of branches to the wall of the appendix. The meso-appendix is not continued quite to the tip of the appendix, and where it terminates the appendicular artery continues its course in close apposition to the muscular wall. The base of the appendix also receives twigs from the cæcal arteries, and, in the female, the ovarian artery is said to supply a small branch, but, like the intercommunication between the appendicular and ovarian lymphatics, the presence of this is not satisfactorily established. There can be no doubt that the chief factor in the production of gangrene is virulence of inflammation, but it has been supposed that obstruction of the appendicular artery by twists or adhesions may be a contributing cause. The great frequency of gangrenous processes near the tip of the organ may be due to the greater ease with which the terminal portion of the appendicular artery is involved by extension of the inflammation, the vessel being here in closest contact with the muscular wall.

The nerves of the appendix are derived from the superior mesenteric plexus. This plexus consists of branches from the cœliac plexus, the semilunar ganglia, and the vagus. Nevertheless, the wall of the appendix, like other parts of the intestine, is quite insensitive. Consequently absence of pain in appendicitis does not indicate that serious mischief is not in progress. In the early stages of appendicitis pain is often vaguely referred to the region of the umbilicus. This pain is supposed to be due to the drag exercised by the contracting cæcum and appendix on their peritoneal attachments to the posterior abdominal wall, for it is well known that tension on the root of the mesentery or mesocolon can elicit pain of this nature. This supposition is supported by the fact that the umbilical pain soon ceases,

presumably owing to paralysis of the gut induced by increasing inflammation. The occurrence of deep-seated local pain in appendicitis indicates inflammation of the sensitive parietal peritoneum or retroperitoneal tissues. Absence of this deep tenderness may be of serious prognostic import, indicating a profound toxæmia. Cutaneous hyperæsthesia, if present, for it is exceptional, corresponds in distribution to the tenth, eleventh, and twelfth dorsal segmental areas. It is a true reflected tenderness, and is not derived from the visceral peritoneum, which appears to be unable to originate sensory impulses.

The mass which is felt in the abdomen in cases of appendicitis may consist of inflamed cæcum and small intestine, the appendix itself, inflamed omentum, enlarged glands, fæcal matter, and pus. The disposition of the great omentum over the front of the intestinal coils accounts for the frequency with which it forms part of the wall of an appendix abscess.

Venous thrombosis may arise as a complication of appendicitis. The veins of the appendix are radicles of the portal system, and may be the starting-points of portal thrombosis or portal pyæmia. The proximity of the appendix to the right iliac veins, across which it may lie, sometimes leads to thrombosis or infection of these vessels, and in such cases thrombosis of the right femoral vein, extension of clot to the opposite common iliac vein, systemic pyæmia or pulmonary embolism, are dangers to be feared.

Arteries are rarely eroded during the course of the inflammation. If they are, hæmorrhage into the peritoneal cavity or into the bowel may occur. The right iliac arteries, the deep circumflex iliac, and the colic branches in the wall of the gut have been known to be the source of hæmorrhage in such cases.

The tracks followed by the pus when suppuration is localised and intraperitoneal, or becomes extraperitoneal, are described subsequently. (See page 383.)

In the attempt to avoid hernia as a sequel to operations on the appendix, advantage is taken of the arrangement of the muscular layers of the abdominal wall. The rectus sheath may

be opened, and that muscle pulled aside, so that when allowed to return it covers the incision in the posterior wall of the sheath, or access may be gained by separating the external oblique, internal oblique, and transversalis muscles in the direction of their fibres which decussate with each other. In all cases it is important to bear in mind that the outer edge of the rectus usually extends in the adult to within two inches of the anterior superior iliac spine.

### DISEASES OF THE PERITONEUM.

#### ACUTE PERITONITIS.

**Acute peritonitis** is usually the result of infection which has spread from some viscus which is invested by the visceral or abuts against the parietal layer of the membrane. In the female the orifices of the Fallopian tubes allow infection to spread from the genital passages. In some cases peritonitis results from blood-borne infection, and it may also follow penetrating wounds of the sac. The extension of the serous membrane into the dome of the diaphragm renders the sac liable to penetration by wounds of the lower part of the thorax, and its extension around the rectum and along the upper third of the posterior vaginal wall exposes it to injury in these situations. The membrane may also be infected along a patent funicular process.

The most vulnerable part of the visceral layer is that which invests the small intestine and appendix. The pelvic portion is more resistant to infection. It is exceptional for infection to spread through the parietal portion of the greater sac from without, but this sometimes happens, particularly in the region of the diaphragm. The lymphatic channels are better developed in this situation. The parietal portion of the lesser sac is liable to infection from the subjacent pancreas.

The great absorptive power of the peritoneum accounts for the rapidity with which toxic symptoms arise in peritonitis. The area of the membrane is said to be equal to that of the whole integument of the body, and it is looked upon as a large lymphatic space, although it is doubtful if its direct connexion



with lymphatics by means of stomata is so free as was at one time supposed. Unfortunately it matters little whether the toxic absorption is due to the agency of lymphatics or the blood vessels with which the membrane is freely supplied.

The nervous connexions of the peritoneum are important. It receives branches from the lower seven intercostal trunks through the great abdominal plexuses of the sympathetic. The same nerves innervate the abdominal muscles and lower intercostals. The movements of these muscles are therefore restricted in peritonitis, as also is that of the diaphragm.

The visceral layer of the peritoneum appears unable to transmit painful sensations, but the parietal layer is extremely sensitive. The pain of peritonitis is due to implication of the parietal peritoneum, and is as a rule deep seated and not referred to cutaneous areas.

#### INTRAPERITONEAL SUPPURATION.

(Fig. 50, p. 384.)

For the consideration of localised abscesses and the tracks followed by purulent collections within the peritoneum, it is convenient to divide the peritoneal cavity into certain regions.

1. The Subdiaphragmatic or Supramesocolic Region, which is included between the transverse mesocolon below and the diaphragm above. This space may further be subdivided into a part below the right wing of the diaphragm, a part below the left wing, and a part below the right lobe of the liver in the region of the right kidney.

2. The Lumbar Regions, situated laterally, and containing the ascending and descending portions of the colon. These regions are effectually separated by the prominence of the lumbar spine.

3. The Submesocolic Region, between the transverse mesocolon above and the mesentery of the small intestine below. This region is continuous laterally with the right lumbar region, of which it is virtually a part.

4. The Submesenteric Region, intervening between the mesentery and the true pelvis. It bears the same relation to the

left lumbar region that the submesocolic region bears to the right.

5. The Lesser Sac of the Peritoneum. This is a diverticulum of the greater sac arising in the region of the right kidney.

6. The Region of the Pelvis.

The mesocolon and mesentery constitute peritoneal partitions of which the mesocolon is more complete and important. The transverse mesocolon is attached to the posterior abdominal wall at the level of the second lumbar vertebra. Traced from right to left across the abdomen it first ascends as it crosses the lower part of the right kidney, the second part of the duodenum, and the head of the pancreas, after which it runs to the left with less upward inclination, along the anterior border of the body of the pancreas. The projection of the lumbar spine and a small upward prolongation of the superior layer of the mesocolon in this region sharply separate the right and left halves of the fold. Consequently extravasations of fluid above the right half are directed downwards towards the depression around the upper part of the right kidney.

The root of the mesentery springs from the posterior abdominal wall along an oblique line which extends from the left side of the second lumbar vertebra to the right iliac fossa. Starting at the duodeno-jejunal flexure, the line of attachment crosses the front of the terminal part of the duodenum, the aorta, the inferior vena cava, the right ureter, and the right psoas muscle to reach a variable point in the right iliac fossa. The direction may be indicated on the surface by drawing a diagonal line from a point on the transpyloric line, an inch to the left of its middle point, to the right iliac region. The mesentery is not so efficient in separating the submesocolic from the submesenteric regions as the transverse mesocolon is in cutting off the subdiaphragmatic space from the regions below.

**Subdiaphragmatic Region.**—The viscera in relation with this part of the greater sac are the stomach, spleen, liver, gall bladder, bile ducts, part of the duodenum, and upper part of the right kidney. An abscess in this region may be due to disease

INTRA-PERITONEAL SUPPURATION.

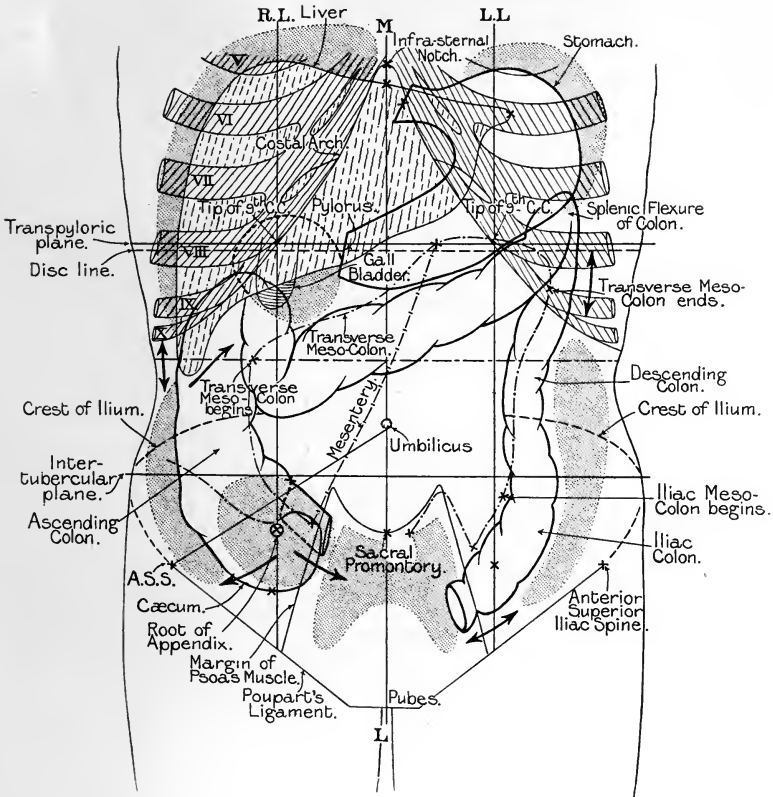


FIG. 50.—The shaded areas indicate the common positions of intra-peritoneal abscesses. The heavy arrows indicate the directions in which such abscesses tend to track. Abscesses among the coils of small intestine have been omitted.

The diagram is founded on one drawn by Christopher Addison to illustrate the topography of the abdomen (see page 362).



or injury of any of these structures. But the area may also become infected by pus which has travelled up alongside the ascending or descending colon. Conversely, the track of infection may pass from above downwards towards the pelvis.

A subdiaphragmatic abscess may lie between the liver and the diaphragm in the right cupola, around the spleen in the left cupola, or around the kidney in the right kidney pouch. The falciform ligament sometimes forms the right or left boundary of an abscess beneath the diaphragm. The area of percussion dulness or of tympanitic resonance when gas is present may be sharply limited by the position of the ligament. The latter is a crescentic fold, the remnant of the ventral mesentery of the embryo. Its convex border is attached to the under surface of the diaphragm, near to the right limit of the pericardium, and to the anterior abdominal wall an inch or more to the right of the middle line. Its concave border is attached to the superior and anterior surfaces of the liver. The fold extends downwards to within one or two inches of the umbilicus, and the round ligament lies in its free edge.

*An abscess under the right wing of the diaphragm* will tend to raise the diaphragm and depress the liver. Hence on the one hand, respiratory movements are restricted and the right lung becomes partly collapsed, whilst on the other the liver is depressed. Subdiaphragmatic friction, simulating pleural friction may be heard, and pain, tenderness, and even œdema of the right axillary region may be present.

*An abscess under the left wing of the diaphragm* lies in the greater sac, occupying part of the stomach chamber, and also the space between the spleen and the chest wall. Such abscesses are sometimes termed perisplenic. The retention of pus in this situation is considerably assisted by the presence of a well marked phrenico-colic fold. This fold is continuous with the left part of the great omentum, from which it is derived, and extends outwards from the splenic flexure of the colon to the diaphragm. Thus, when the patient is supine, there is a natural well in the left subdiaphragmatic region, containing the spleen

and a portion of the stomach, and corresponding to the well around the upper part of the right kidney. This well is ready for the reception of any fluid which may trickle into it. The physical signs produced by a collection of fluid here are similar to those already described as accompanying right-sided collections, with the exception that the left lobe of the liver alone may be depressed and the cardiac impulse may be displaced upwards. The subdiaphragmatic friction sound may have a cardiac or respiratory rhythm.

*A subdiaphragmatic abscess which lies beneath the liver*, and is consequently sometimes termed subhepatic, occupies a peritoneal well, known as the right kidney pouch. This belongs to the greater sac, and its boundaries are as follows. Above and in front, the right lobe of the liver and its ligaments; below, the hepatic flexure of the colon and its attachment to the posterior abdominal wall; internally, the peritoneum covering the descending duodenum and lumbar spine, and stretching forwards to the foramen of Winslow; externally, the parietal peritoneum of the lumbar region. The peritoneum covering the right kidney forms the floor of the pouch. This well has proved capable of holding nearly a pint of fluid before it overflows into other parts of the peritoneal cavity. With the patient supine, the right lumbar region slopes from below towards it; the right half of the transverse mesocolon forms an inclined plane directed to it, and the gall bladder and bile ducts lie in intimate relation with it. Fluid collections in this situation may travel from the appendix along the lumbar region, or from the pylorus and duodenum, being directed by the prominence of the lumbar spine and inclination of the transverse mesocolon, or may be the result of lesions of the gall bladder and its ducts. Abscesses in the liver and right kidney or in connection with malignant growth of the hepatic flexure of the colon may also be found in this part of the peritoneal sac. A large collection of fluid here may so push up the liver and impede the action of the diaphragm as to simulate a collection between the liver and the diaphragm, but the edge of the liver would be raised in

the former case and depressed in the latter. The subdiaphragmatic region may be almost entirely shut off from the rest of the peritoneal cavity in consequence of adhesion of the great omentum to the anterior abdominal wall. The upper part may also be shut off by adhesions between the abdominal wall and the liver or stomach.

**The Lumbar Regions.**—These regions communicate below with the pelvis. With the body supine they form inclined planes leading from the pelvic brim to the subdiaphragmatic space. Their separation from the latter is very incomplete. On the left side the phrenico-colic fold, and on the right a similar but more variable fold may form more or less effectual barriers. As the result of appendix suppuration pus may track upwards in the right loin, between the colon and the parietes, and infect the right kidney pouch or extend between the liver and the diaphragm. Even suppuration of pelvic origin may travel the same way. Conversely, suppuration in connexion with perforated pyloric or duodenal ulcers may be directed by the mesocolon and prominence of the spine into the kidney pouch, and then travel downwards in the right lumbar region simulating appendicitis. Lesions of the gall bladder may produce a similar result.

When perforation of the anterior wall of the stomach has given rise to a collection of fluid in the greater sac around the spleen, a well developed phrenico-colic fold may effectually seal off the infection from the left lumbar region. A badly developed fold may allow the lumbar region and even the pelvis to be infected. Pelvic collections and infections may travel upwards by the same route, following the descending colon.

**The Submesocolic Region** lies between the transverse mesocolon and the mesentery of the small intestine. Its apex lies at the point of convergence of these folds near the second lumbar vertebra. The prominence of the spine causes this region to slope away towards the right loin, with which region it is virtually continuous. The combined areas contain coils of small intestine, the cæcum, ascending colon, lower part of the right

kidney, part of the right ureter, and often the vermiform appendix.

**The Submesenteric Region** is related to the duodeno-jejunal junction, the greater part of the small intestine, the left part of the transverse colon, the lower part of the left kidney, the left ureter, the lower part of the abdominal aorta, and the common iliac arteries. The appendix when it lies behind the termination of the mesentery, is also in relation with it. This region communicates freely with the left lumbar region and with the pelvis.

**The Lesser Sac** may be looked upon as a diverticulum from the subdiaphragmatic area of the greater sac. The viscera in anterior relation to it are the stomach, first part of the duodenum, and Spigelian lobe of the liver. Behind it are the pancreas, the left kidney and suprarenal body, and the transverse colon. This sac varies much in size in different bodies. The variation is particularly marked in the degree to which the cavity extends downwards between the layers of the great omentum. The sac reaches laterally from the hepatic to the splenic flexures of the colon, and is bounded below by the transverse mesocolon. Like the greater sac the cavity is potential rather than real, and the same applies to the foramen of Winslow, the boundaries of which are normally in contact. A diverticulum of the lesser sac clothes the back of the commencing duodenum for about an inch, so that it is possible for duodenal ulcers to open into it. Clinically, fluid collections in the lesser sac are exceptional. It often becomes obliterated by inflammatory lesions before there is any actual perforation. It may be invaded from the stomach, the duodenum, the liver, the pancreas, the left kidney, the suprarenal capsule, the transverse colon, and possibly the spleen. The cystic bile duct has been known to perforate into the sac through the posterior layer of the lesser omentum.

When an effusion occurs a swelling is seen on the left hypochondriac, epigastric and umbilical regions, of somewhat rounded outline, and varying from time to time according to the condition of the overlying stomach. The colon lies below the swelling,



and an area of dulness may be found over the ribs posteriorly, owing to the upraising of the diaphragm in this region.

**The Pelvic Region** is often the site of intraperitoneal abscess. It may be invaded from any of the viscera which it contains. It is also open to invasion from the lumbar regions as already explained, and in cases of sudden perforation of the stomach the great omentum may direct the stomach contents directly to the pelvis. The region is accessible to examination by the rectum or vagina. The proximity of the abscess to the bladder may lead to painful micturition or retention of urine, and even to exfoliation of parts of the mucous membrane of the bladder. Its relation to the rectum may cause tenesmus. The abscess may discharge into bladder or bowel. It is often shut in above by matted coils of intestine and by the great omentum.

#### RETROPERITONEAL SUPPURATION.

Retroperitoneal abscesses may lie in the retroperitoneal connective tissue or beneath the deep fascia of the abdomen. Abscesses in the areolar tissue are usually derived from the appendix, but may originate in the cæcum, lymphatic glands or the tissue around the uterus or rectum. When the appendix is closely bound down to the abdominal parietes by developmental or inflammatory adhesions it is in a favourable position to infect the planes of connective tissue. The pus may spread freely and track in various directions. Upward extension involves the retroperitoneal tissue of the lumbar region, and may give rise to perinephritic abscess. The posterior, non-peritoneal, surface of the liver may be invaded, and even the subpleural tissue giving rise to infection of the pleural sac. In other cases the abscess spreads inwards towards the spine surrounding and eroding the bodies of the vertebræ. Sometimes the pus makes its way along the inguinal canal or even along the femoral sheath. It may invade the connective tissue planes of the pelvis, and has been known to pass through the obturator foramen into the buttock. Perforation of the psoas fascia may

allow appendix suppuration to extend into the psoas sheath. The hip-joint, over which the muscle passes, may be invaded, and the elongated bursal space, which lies between the vastus internus and the inner aspect of the femur, is sometimes infected, giving rise to periostitis and necrosis, which may show itself in the popliteal region. The pus may also be directed into the thigh under the iliac fascia. Psoas abscess dependent upon caries is described elsewhere.

#### ABDOMINAL HERNIA.

An **abdominal hernia** is the protrusion of an abdominal viscus from the abdominal cavity. As a rule such a hernia consists of a sac, composed of peritoneum, and contents, which may be any of the abdominal viscera.

Except in quite rare instances there is no rupture or tearing in the causation of a hernia, and therefore the term "rupture" to designate this condition is not a scientific one.

Certain anatomical peculiarities heighten the likelihood of a protrusion at particular regions of the abdominal wall. These may be termed weak spots. In addition to the area being a weak one, from the want of the thickness of the structures in the region, there is also the possibility of a congenital pouch of peritoneum persisting after birth, into which viscera may descend, and some believe that this finger-like process of serous membrane is the most important factor in the production of a hernia.

The three common weak spots at which herniæ most usually protrude are the inguinal region, the femoral region, and the umbilical region. All other herniæ are comparatively rare, and will only be briefly alluded to.

**Inguinal Hernia.**—In the male the testis, accompanied by its cord, passes from the abdomen into the scrotum. In the female the round ligament of the uterus traverses the abdominal wall and occupies the same position as the upper part of the spermatic cord does in the male. A weak spot is therefore left in this

region. Both of these structures are normally accompanied by a patent process of peritoneum in the fœtus, which should close some few weeks before birth; in many instances, however, it remains as an unclosed tube, ready to receive bowel or omentum when the intra-abdominal pressure is raised sufficiently high.

The serous sac, continuous with the parietal peritoneum lining the anterior abdominal wall, finds its way through the parietes in an oblique manner. It commences at the region known as the deep (or internal) abdominal ring. In the fœtus and young infant this is situated almost immediately above the crest of the os pubis, but as growth proceeds, and particularly when adult life is reached and the pelvis developed, it gradually recedes upwards and outwards, till at last it is found a finger's breadth above the middle of Poupart's ligament. This so-called "ring" is in reality a slit-like depression in the fascia transversalis, readily seen from the abdominal aspect if the parietal peritoneum is stripped off external to the deep epigastric artery.

The neck of the sac then passes along the inguinal canal, which passage is oblique, running downwards and inwards, and of very short length in the young subject, but measuring from one and a half to two inches in the adult. The boundaries of this passage are of great importance, and should be thoroughly understood before undertaking an operation upon an inguinal hernia.

Anteriorly, that is superficially, there are placed the skin; two layers of superficial fascia, one containing fat, superficial epigastric vessels and some cutaneous nerves, and the other, the deeper, being of a more fibrous texture; then the aponeurotic fibres of the external oblique muscle, running in the same direction as the canal itself; and in front of the outer half of the passage, and therefore covering and protecting the deep abdominal ring, the arching muscular fibres of the internal oblique and a few of the transversalis arising from the external half of Poupart's ligament. These fibres are of much moment, seeing that they arch over the cord or the round ligament, and becoming the conjoined tendon, are inserted into the crest of the os pubis, deep to the cord or round ligament. Thus, when they contract, they tend to

straighten and therefore approximate to Poupart's ligament and close the canal, thus having almost a sphincter-like action.

Posteriorly, that is deeply, there is found in the outer half of the canal the fascia transversalis, whilst in the inner half both fascia transversalis and conjoined tendon are seen, and in front of the latter at the most internal part is the triangular fascia.

The roof of the canal is formed by the arching fibres of the internal oblique and transversalis muscles, while the floor is really a groove produced by the fusion of the fascia transversalis with the posterior part of Poupart's ligament.

The congenital pouch of peritoneum having passed the whole length of the inguinal canal, emerges through the superficial (or external) abdominal ring. This again is no true "ring," being a triangular opening bounded by an inner and an outer pillar, derived from the aponeurosis of the external oblique muscle, and below by the crest of the os pubis. Of the two pillars, the inner is flat, and is attached to the front of the symphysis pubis, while the outer is rounded and is inserted into the spine of the os pubis. The size of this slit in the external oblique varies in different individuals.

Except in very stout adults or young infants the superficial ring can be palpated by a finger placed directly over it, immediately above the crest of the os pubis, a fact which is not usually fully recognised. By invaginating the scrotum until the pubic spine is felt, the tip of the finger may be readily made to enter the ring, and if the manœuvre is carried out with care, no pain need be occasioned to the patient. There are two facts which may be gleaned from this method of examination; one, the approximate size of the superficial ring, a knowledge of which is not of much practical moment as it has but little bearing upon hernia; the other, whether there is any protrusion descending the inguinal canal and impinging upon the top of the examining finger, a matter of considerable moment in the diagnosis between an inguinal and a femoral hernia.

In the female, the spine of the os pubis may be defined by

following up the inner border of the adductor longus tendon, made tense by abduction of the slightly flexed thigh.

A hernia which emerges from the inguinal canal is spoken of as a *complete inguinal hernia*. It is usual to give a somewhat elaborate description of the coverings of such a protrusion, but it is difficult, and of little practical importance, to make out these layers separately. In cutting down upon a hernial sac what is really desirable is to be able to distinguish the sac itself, rather than to differentiate the different layers which cover it.

In the older operative procedures, with the incision made through the scrotum, the skin is the first layer, and then follows the dartos tissue, then the layer derived from the deep portion of the superficial fascia, covering the superficial abdominal ring itself and called the intercolumnar fascia, after which the cremasteric fascia and muscle will be exposed, then the infundibuliform fascia derived from the fascia transversalis, beneath which is a somewhat fatty layer composed of extraperitoneal tissue covering the sac itself.

In the present-day method of exposing the hernial sac by an incision parallel with the inner half of Poupart's ligament and a finger's breadth above it, the following structures will be divided before the sac is laid bare:—The skin, two layers of superficial fascia (in which some small blood vessels may be cut), the aponeurosis of the external oblique, and, when this is cut, the interior of the canal will be exposed, and lying within it the ilio-inguinal nerve running down upon the neck of the sac, which itself is covered by the cremasteric and infundibuliform fasciæ, the spermatic cord being placed in the great majority of cases posterior to the sac.

The sac may be recognised chiefly by its colour, which is in most instances a characteristic slaty-blue when strangulation is present, and of a bluish-white opaque look in non-strangulated cases. Sometimes typical arborescent vessels may be observed on its outer aspect, and it is important to recollect that the external surface of the sac is rough, and that it is not until

the interior is exposed that the smooth aspect of the peritoneum is seen.

The somewhat rare form of *direct inguinal hernia* is one in which the protrusion takes place within Hesselbach's triangle, the boundaries of which are, externally, the deep epigastric artery; internally, the outer border of the rectus abdominis; and, below, the inner half of Poupart's ligament. It is difficult or well-nigh impossible to diagnose without dissection the difference between an indirect (or oblique) and a direct inguinal hernia. If the finger can be introduced into the inguinal canal and the pulsation of the deep epigastric artery felt, the protrusion of the hernia, on the patient coughing, internal to this artery proves its direct course. It is important to remember that in a considerable proportion of large, indirect, inguinal herniæ the neck of the sac drags the deep epigastric artery downwards and inwards, till at last the deep and the superficial rings are almost directly the one behind the other, the hernia in such a case simulating a direct one very closely.

**Femoral Hernia.**—Behind the innermost part of Poupart's ligament and a little below and external to the spine of the os pubis is a potential opening, made patent by the scalpel or by the protrusion of a femoral hernia. This is the so-called "femoral ring," and is the upper or abdominal entrance into the femoral canal. It has an oval-shape, with the long axis transversely placed, and in the erect position its plane is nearly horizontal.

Its boundaries are, internally, Gimbernat's ligament, which is in reality the triangular internal attachment of Poupart's ligament; externally, the common femoral vein, separated by the septum of the femoral sheath; in front is Poupart's ligament itself; and behind is the pectineus muscle covered by the pectineus fascia, while deep to the muscle is the horizontal ramus of the os pubis.

The femoral ring opens into the femoral canal, which is the most internal division of the femoral sheath, and is composed anteriorly, of the transversalis fascia, and, posteriorly, of iliac

fascia. The canal itself contains the lymphatic vessels passing from the superficial femoral lymphatic glands to the deep external iliac lymphatic nodes, and sometimes a lymphatic gland itself is actually placed in the canal. The boundaries of the canal are, anteriorly, the fascia transversalis, posteriorly, the fascia iliaca, externally, the septum separating the space from that for the vein, and, internally, the meeting of the fascia transversalis with the fascia iliaca.

Superficial to the femoral canal, and in a sense forming its lower aperture or exit, is found the so-called saphenous opening—a term which is wholly a misnomer, for there is no true opening, and it is not, therefore, an obvious one. The pseudo-opening lies at the upper and inner part of the thigh, a couple of fingers' breadth below and external to the spine of the os pubis. It is formed between two layers of the fascia lata of the thigh, known as the iliac, or anterior, and the pubic, or posterior, portions. It gives passage to the internal saphenous vein. The upper edge of the opening has its upper extremity attached to the anterior aspect of Gimbernat's ligament and the lower border of Poupart's ligament, and is fairly defined and somewhat sharp.

Again, as in the case of an inguinal hernia, the actual coverings of a femoral hernia are not of much moment. They may be mentioned here as the skin, superficial fascia, cribriform fascia (probably the deeper layer of the superficial fascia), fascia transversalis, extraperitoneal tissue, followed by the sac itself.

Whilst the sac of a femoral protrusion is traversing the femoral canal, it necessarily takes a vertical direction, and lies to the inner side of the femoral vein, and its neck is external to the spine of the os pubis. In the male, the spermatic cord is above, that is on a higher level than the femoral ring, the deep epigastric artery is on the outer side and above. The pubic branch of this vessel passes downwards and backwards on the outer side of the femoral ring to anastomose on the upper surface of Gimbernat's ligament with a similar branch derived from the obturator artery. In about two cases out of every

seven this anastomosis is so large as to form the origin of the main portion of the obturator vessel. When this "abnormal" origin of the obturator artery is present, in 10 per cent. of the instances the vessel runs over the anterior or upper margin of the femoral ring, and then down along its inner border to reach the obturator foramen. In another 37 per cent. it passes across the femoral ring, and in 53 per cent. it arrives at the obturator aperture by skirting the external side of the femoral opening. Thus it will be seen that if a femoral hernia becomes strangulated, the artery will be liable to injury, if the knife be used, in at least 10 per cent. of all cases where its abnormal origin is present. Moreover, if the neck of the hernial sac in its descent pushes the artery over, when lying across the ring, to the inner side it will again become placed in a position of danger.

In the strangulation of a femoral hernia the edge of Gimbernat's ligament tends to exercise a sharp cutting pressure upon the sac and its contents; hence the very rapid damage which the intestine is likely to undergo.

When a femoral protrusion has left the femoral canal, it most frequently tends to descend the front of the thigh, rather than to ascend superficial to Poupart's ligament. The upward course is induced probably by the attachment of the deep layer of the superficial fascia to the fascia lata being very firm.

A femoral hernia is not infrequently sacculated by the pressure of fibrous bands derived from the cribriform fascia.

**Umbilical Hernia.**—During the process of development the two lateral halves of the parietes of the abdomen approach one another and ultimately coalesce, save for the aperture through which pass the umbilical vessels. This opening is closed by scar-tissue subsequent to the separation of the cord.

The anatomy of the umbilicus in the first few weeks after birth shows an arrangement of fibres of semi-elastic tissue in the form of a sphincter. These tend to become less elastic and more fibrous, so that they gradually contract, and, as it were, cut through the structures which originally traversed the



opening. At the same time the vessels become obliterated and themselves converted into a mass of scar-tissue, at first weak, but later of considerable denseness and firmness.

In adult life the umbilicus shows two sets of fibres about it, one decussating across the middle line, and the other of circular direction around the aperture or pseudo-aperture itself. These form the so-called ring, which is stronger at its lower than at its upper part. The scar-tissue in the upper margin of the opening is less firm than in the lower, owing to the fact that in the former region there are only the remains of the umbilical vein, while in the latter are found the obliterated urachus and the arteries.

If the two halves of the abdominal wall fail to unite in any marked degree in the region of the umbilicus, a true "congenital" umbilical hernia will result.

Dilatation of the scar-tissue at the umbilicus after the fall of the cord constitutes an acquired umbilical hernia, "infantile" when in a young subject, "adult" after infancy.

The tissues covering an acquired umbilical hernia become rapidly stretched, and there is a tendency for the peritoneum forming the sac to become fenestrated. The contents of such a hernia are often irreducible owing to adhesions, partly within the sac, but partly, also, to the external tissues, if the sac is fenestrated.

#### DISEASES OF THE SALIVARY GLANDS.

**Wounds of the Parotid Region.**—The parotid gland contains within its substance very important structures. The formation of the temporo-maxillary vein in the upper part of the gland by the union of the superficial temporal and internal maxillary veins, and its course somewhat superficially downwards through the gland, renders this venous trunk particularly liable to injury in punctured wounds of this region. Deeper than the vein lies the external carotid artery breaking up into its terminal branches, the superficial temporal and the internal maxillary. It again may be damaged but much more rarely. Deepest of all

in the parotid, entering the gland soon after its exit from the stylo-mastoid foramen, is the facial nerve, which traverses the parotid in a direction towards its anterior margin becoming more and more superficial. It is this structure which is most commonly divided, resulting in partial, or possibly total, temporary or permanent paralysis. In the uppermost part of the gland is found the auriculo-temporal nerve, and incisions in this region may damage it.

A wound of the gland substance, but more especially of its duct, is prone to be followed by a salivary fistula. Stenson's duct emerges from the anterior border of the gland and runs across the cheek, inclining slightly downwards. The middle third of a line drawn from a point half-way between the bottom of the concha and the lobule of the ear, to a point half-way between the ala of the nose and the red margin of the upper lip, represents very accurately the course of the duct. It is well to remember that it first lies on the masseter, and at the anterior margin of the muscle it makes a sharp bend inwards, perforates the buccinator and opens on the mucous membrane of the cheek, opposite to the second upper molar tooth, having a length in the adult of about two inches. Thus any attempt to pass a probe from the mucous opening should be preceded by eversion of the cheek to straighten the duct. An incised wound across the line of the duct may sever it, and thus establish a salivary fistula.

**Parotitis.**—The parotid gland is invested by a complete capsule of deep cervical fascia, and is divided into lobes by processes of the fascia passing into its interior. It is thus that inflammatory products formed within are tightly bound down and cause considerable suffering to the patient. The swelling of the gland interferes with the proper movements of the mandible, and the motion of this bone induces great pain by the pressure of the condyle upon the inflamed gland tissue. Seeing that there is very little tissue intervening between the parotid and the internal jugular vein behind, it is possible that any cerebral symptoms in the course of mumps may be due to cerebral hyperæmia caused by pressure.

There is no anatomical connexion between the parotid and the testis or the ovary, and therefore metastatic inflammations in these organs must be dependent rather upon a general infection.

Parotitis, if not of the specific variety, may be induced by the extension of septic organisms from the mouth along the duct to the parenchyma of the gland. This may happen particularly in enteric fever, and during convalescence from laparotomy, especially where there is neglect of the proper care to keep the teeth, gums, and tongue clean.

Suppuration within the capsule of the gland is not uncommon. The pus formed will track in the line of least resistance. Most usually the line followed is forwards on to the cheek, backwards and upwards through the cartilaginous portion of the external auditory meatus, or inwards towards the pharynx, where there may sometimes result a retropharyngeal abscess. This latter situation is probably gained by the weak spot in the capsule in the space between the anterior aspect of the styloid process and the posterior border of the external pterygoid. For the same reason, a collection of pus primarily originating in the retropharyngeal tissues may find an exit through the substance of the parotid. A parotid abscess may also advance into the temporal fossa, into the tissues of the neck, or into the temporomandibular joint cavity.

In opening a parotid abscess, care must be taken not to wound the structures contained within it. If an incision is made directly into the abscess cavity, it should be so planned as to lie nearly horizontally, and as low down as possible, so as to avoid injury to the facial nerve or the parotid duct. Such an abscess may be conveniently and safely opened by cutting on to the capsule of the gland by an oblique incision, downwards and forwards, and then opening into the abscess cavity by thrusting a director onwards into it, and subsequently enlarging the opening by a pair of sinus forceps.

**Parotid Tumours.**—Certain peculiar innocent growths are found in connexion with the parotid, these tumours being mixed in character, one component frequently being cartilage.

This is possibly explained by the fact that islets of embryonic cartilage from the first or mandibular arch may remain enclosed within the gland. These innocent growths of the parotid merely displace the contained structures, and although they may reach a considerable size, seldom interfere with the functions of the vessels or the nerves. Excision of such growths is fairly easy, many being encapsuled. Temporary facial paralysis may follow.

Malignant tumours of the parotid, particularly sarcoma, quickly infiltrate the tissues and soon implicate the facial nerve, producing paralysis. Such growths are extremely difficult to remove as may be readily understood when the structures contained within the gland are remembered.

The anterior auricular lymphatic glands may lie upon or within the sheath of the parotid. When enlarged they may simulate a parotid tumour. If lying superficial to the capsule they are readily excised. If residing within the capsule, it may even be necessary to divide some gland tissue before they are exposed, and care must be taken while dissecting them out, especially when dealing with the deeper parts of the wound.

**Parotid Calculi.**—The duct of the parotid may be the site of a calculus. This may lead to a fluid swelling occupying the region of the gland, filling up the sulcus between the ramus of the jaw and the internal auditory meatus, and passing forward on to the cheek. Occasionally the concretion may be palpated in the line of the duct on the cheek, but it is much more readily demonstrated by a probe passed into the duct through its buccal aperture. If the cheek is drawn outwards and somewhat everted, a small papilla may be observed opposite the second upper molar tooth, and with a little dexterity a fine eye probe can be guided into the opening on its summit. This, if made to pass along the lumen of the duct, will strike the calculus. Excision of the stone should always be made from the buccal surface to avoid the formation of a salivary fistula.

**Submandibular Gland.**—This salivary gland is much less often the seat of disease than is the parotid. The duct (Wharton's duct)

arises from the deep portion of the gland lying upon the hyoglossus muscle, deep to the mylo-hyoid. The duct runs forwards and somewhat inwards, close beneath the mucous membrane of the floor of the mouth, on which it makes a distinct ridge, to open on a papilla by the side of the frænum linguæ. A calculus in this duct can be easily palpated, or even seen, from within the mouth. The duct is not readily dilated by retained secretion, and therefore its blockage by a concretion is apt to be accompanied by much pain. Consequently a ranula is seldom a dilatation of this duct, but is much more generally a retention cyst of one of the mucous glands of the floor of the mouth. The calculus can be easily excised by an incision made into the duct on the floor of the mouth.

The submandibular gland covers that part of the hyoglossus muscle which overlies the lingual artery, and therefore it will require displacing upwards to expose the lingual triangle formed by the two portions of the intermediate tendon of digastric muscle and the hypoglossal nerve.

#### DISEASES OF THE LIVER.

**Deformities and Displacements.**—The deformity of the liver, which is of most clinical interest, is the presence of a linguiform lobe, known as Riedel's process. This is an elongated projection which springs from the edge of the right lobe, close to the gall bladder. In some instances it is congenital; in others it appears to have been drawn down by the weight of a distended gall bladder, which is attached to its deep surface. The linguiform lobe projects downwards in front of the right kidney, and if its connexion with the liver escapes recognition it may be confused with the kidney, a growth of the colon, or an intussusception. Biliary colic may exist with a linguiform lobe, owing to traction on the bile duct.

Various distortions of the liver are met with, as the result of tight lacing and the use of tight belts, the precise deformity varies with the level at which the compression is applied.

Downward displacement, or ptosis of the liver, may occur alone or be associated with general enteroptosis. The liver is supported by the combined action of its ligaments, its attachment to the diaphragm by means of the inferior vena cava and extensive posterior non-peritoneal surface, and by the action of the muscular portion of the abdominal wall. The liver, being plastic, undergoes considerable modification of shape when displaced. The displacement may cause obstruction of the structures which enter the portal fissure, and also in rare cases the inferior cava may become distorted. The traction of a displaced liver may induce various nervous symptoms, such as pain in the shoulder, dyspnoea, etc.

**Rupture of the Liver.**—The liver is bulky and occupies a considerable portion of the upper part of the abdominal cavity; moreover it is heavy, friable, and subjacent to yielding ribs. Consequently it is more often ruptured as the result of falls and contusions than any other abdominal organ. The liver substance bleeds freely when torn or penetrated, for the portal and hepatic veins are not provided with valves, and the latter in particular are held open by their intimate relation to the liver substance. If the rupture involves the posterior, non-peritoneal surface, the blood will escape into the retroperitoneal tissues, instead of into the peritoneal cavity. In some cases the liver has been extensively damaged without laceration of Glisson's capsule, the blood then remains confined. Damage to the liver is often associated with fracture of the ribs or costal cartilages which overlie it. Posteriorly, in the scapular line the liver is subjacent to the five right lower ribs, the eighth to the twelfth; in the axillary region it is also under cover of five right ribs, the seventh to the eleventh; in front it is under cover of five right costal cartilages, the fifth to the ninth.

Penetrating wounds of the liver may be in such a position that they also traverse the pleural sac, the lung, the diaphragm, and the peritoneal cavity. Wounds of this nature may be situated in the sixth or seventh intercostal spaces anterolaterally, or in the eighth, ninth, or tenth spaces posteriorly.

**Catarrhal Jaundice** is usually considered to be an infective inflammation of the common bile duct, extending from the duodenum. Occlusion of the common duct by inflammatory swelling of its lining membrane is favoured by the following anatomical conditions—the terminal portion of the duct traverses the duodenum very obliquely; the walls of the duct are thick and do not easily stretch; its lumen is narrow, and in addition, the pressure under which bile is secreted is very low.

In a very large percentage of cases the common bile duct is completely embraced in the head of the pancreas, and in the remainder it lies in a deep groove in the gland. It is therefore possible that inflammatory swelling of the head of the pancreas may be the cause of some of the cases of so-called catarrhal jaundice.

Slight jaundice is easily recognised in the ocular conjunctiva. When looking for it in this situation the presence of yellow sub-conjunctival fat in the parts remote from the cornea should be borne in mind, and attention particularly directed to the conjunctiva immediately surrounding the cornea where the fat is absent.

**Jaundice in the Newly-born** is usually transitory and in all probability due to inspissation of the bile and slight catarrh of the duct. There are, however, graver forms. Obliteration of some part of the bile passages may be present. There appears to be no special site for this, for the stenosis may be found anywhere in the course of the bile ducts from their commencement in the liver to their termination in the duodenum; at the same time the gall bladder may present inflammatory changes and multilobular cirrhosis be present. In other instances the jaundice is due to obstruction of the ducts by perihepatitis, for Glisson's capsule becomes invaginated into the liver at the portal fissure, and so comes into close relation with the ducts, and when inflamed may compress them. Another variety is that in which the umbilical vein, becoming infected at the umbilicus, gives rise to a portal pyæmia. General pyæmia may also result, since, at this age, the ductus venosus connects the portal and systemic venous systems.

**Passive Congestion of the Liver.**—The liver is peculiarly liable to suffer in all conditions of venous back pressure because the hepatic veins are large, open into the inferior vena cava quite close to the right auricle, and are quite destitute of valves. Hence the passively congested, or nutmeg, liver occurs as the result of valvular disease of the heart, obstruction of the pulmonary circulation, adhesion of the pericardium, constriction of the inferior vena cava by mediastinitis, displacements of the heart which bend the inferior vena cava, or pressure on that vessel by enlarged glands or aneurysm. A moderate degree of passive congestion is also seen in the livers of persons who have died slowly with heart failure.

In a nutmeg liver the injected areas correspond to the intra-lobular and sublobular branches of the hepatic veins, the first-named branches occupying the centres of the lobules. The pale areas consist of cells which have degenerated in consequence of the stasis of the circulation. General pressure on the bile capillaries accounts for the moderate degree of bile staining often present.

The jaundice which results from passive congestion of the liver is frequently associated with dropsy of cardiac origin, which indeed often precedes the jaundice. It is often noticeable that the jaundice is much more intense in the upper parts of the body than in the lower œdematous parts, the explanation being that both the fluid in œdema and the pigment of jaundice tend to accumulate in the lymphatic interspaces, and when these are already occupied by stagnant dropsical fluid, it is difficult for the bile pigment to exude into them.

The venous congestion of the liver, coupled with a certain amount of resistance offered by its capsule, causes the organ to become tender to the touch.

The congested liver is enlarged. This is detected by determining the position of the lower edge, after ascertaining that the upper level of percussion dulness is not below its normal situation. The upper limit of dulness to light percussion corresponds to the line indicating the lower limit of the lung. It is found at the



sixth rib in the nipple line, the eighth rib in the axilla, and the tenth dorsal spine posteriorly. In the recumbent position the liver only comes from under cover of the ribs in the epigastrium, its edge passing from the ninth right to the eighth left costal cartilage. The edge can rarely be felt unless the organ is enlarged, but when felt can readily be recognised by its sharpness and the notches corresponding to the fundus of the gall bladder, and the position of the round ligament. A tendinous intersection in the rectus is apt to be mistaken for the liver, so it is well to keep to the outer side of this muscle when seeking for the liver edge. The position of the liver in the dome of the diaphragm causes it to move freely with the respiratory movements.

**Infarction of the liver** is rare. The organ is practically supplied with blood from two sources, the hepatic artery and the portal vein, and the capillaries of both these vessels communicate freely with the hepatic veins. When infarcts do occur they are small and present a nævoid appearance. They have been met with in thrombosis and embolism of the portal vein, embolism of the hepatic artery and retrograde embolism of the hepatic veins.

**Cirrhosis** of the liver is believed to be due to the action of some toxic or infective agent. Most of the blood which traverses the liver is brought from the digestive tract by the portal vein, and this vessel is supposed to be the common track of invasion. The hepatic arteries afford another means of access to the liver and may be responsible for the production of interstitial cirrhosis in certain of the infectious fevers. The pigmentary cirrhosis of bronzed diabetes is often associated with endarteritis of the hepatic arteries, which has led to the belief that infection by this route may cause the disease. The bile ducts afford a third means of invasion, and by some authors the form of cirrhosis designated monolobular or biliary is held to arise in consequence of an infection which ascends these from the duodenum. In multilobular cirrhosis of the liver, which is the common form of the disease, the new fibrous tissue spreads from the portal canals. Branches of the hepatic artery, portal vein and bile duct lie in company in these spaces, but the vein chiefly suffers compression

the artery and duct mostly escaping, in consequence the cirrhotic process is believed to be more intimately associated with the vein than with the artery or duct. The new fibrous tissue is permeated by branches of the hepatic artery, and that vessel becomes enlarged. The portal vein is also enlarged as the result of the obstruction. The portal congestion may be considerably relieved by the opening up of communications which exist between the portal and general venous systems (Fig. 47, p. 338) or by operative measures designed to the same end. Portal radicles anastomose freely with the retroperitoneal venous plexus, and this in turn communicates with the lumbar and renal tributaries to the inferior vena cava. The anastomosis between the two systems takes place behind the pancreas, duodenum, and colon, where these are directly applied to the posterior abdominal wall without the intervention of the peritoneum. Communications also occur between the same systems in the roots of the various mesenteries. A plexus of tortuous veins may often be found between the layers of the coronary ligaments of the liver, communicating on the one hand with the veins of the liver and on the other with the phrenic and intercostal tributaries to the azygos veins. Some small veins which are constantly present descend from the left division of the portal vein along the round ligament to the umbilicus and there form connexions with the epigastric veins and the superficial veins of the abdominal wall. When these veins are much enlarged the venous circle of the umbilicus becomes engorged, giving rise to the snake-like appearance known as the *caput Medusæ*. A venous hum may at times be heard between the umbilicus and the liver, and is attributed to enlargement of the veins descending from the liver. At the cardiac end of the stomach the gastric tributaries to the portal system anastomose with the œsophageal tributaries to the azygos veins, and these communications may become enlarged and varicose. At the lower end of the intestinal tract the portal system effects another communication with the systemic veins, for the superior hæmorrhoidal tributary to the inferior mesenteric vein joins the middle and inferior hæmorrhoidal tributaries to the internal iliac veins.

Engorgement of the various tributaries to the portal vein has been supposed to produce the various signs and symptoms of cirrhosis of the liver in a purely mechanical way. On this assumption, congestion of the gastric tributaries is responsible for dyspepsia, nausea, and hæmatemesis; congestion of the splenic vein for splenic enlargement and anæmia; congestion of the superior mesenteric vein for ascites; and congestion of the hæmorrhoidal vein for hæmorrhoids. The matter, however, is not so simple as this, although it is certain that the splenic enlargement may very much diminish when the portal system is depleted by the result of hæmorrhage from the œsophageal, gastric or hæmorrhoidal veins, or by profuse diarrhœa. Thrombosis of the portal vein, on the other hand, may be followed by great increase in the size of the spleen and ascites.

The ascites in some cases has been supposed to be due to thrombosis of small radicles of the portal vein or of its compensatory communications. This, however, remains to be proved. The effusion is possibly due to a further catarrhal swelling of the liver cells, which strangles the small portal radicles owing to the resistance of the fibrous tissue in which the lobules are enmeshed.

The accumulation of fluid in ascites exercises pressure on the large renal and iliac trunks, causing albuminuria and œdema of the legs, which sometimes extends to the lower part of the abdominal wall, but similar results may arise from the cardiac degeneration which so often accompanies cirrhosis.

The effusion also interferes with the action of the diaphragm, leading to dyspnœa, collapse of the lung bases and irregularity in cardiac action. The shifting dulness of ascites may be simulated by a loaded colon if that be provided with a long mesentery, but may be distinguished by the fact that the gain of resonance in the uppermost flank is not accompanied by a corresponding loss of resonance in the flank which is lowermost.

The inferior vena cava, although embedded in a depression at the back of the liver, is rarely exposed to much compression.

The indications of enlargement of the liver have been considered already. The normal vertical extent of liver dulness

may be given as four inches in the right nipple line and four and a half to five inches in the axilla.

**Abscesses of the Liver.**—The routes by which pyogenic infection may reach the liver are the portal vein, the bile ducts, the hepatic artery and the lymphatics. Ulceration of any part of the intestinal tract from the cardiac end of the stomach to the anus, or inflammation of the umbilical vein in infancy may give rise to portal infection and multiple abscesses. Radicles of the portal vein may also be infected by abscesses of the spleen or mesenteric glands, by inflammation of the gall bladder or large bile ducts, or by retroperitoneal suppuration through their communications with the subperitoneal venous plexus.

Infections of the gall bladder and large bile ducts may extend directly to the liver along the biliary passages, the retrograde extension being much facilitated when biliary stasis is present. Multiple small abscesses like those of portal pyæmia result.

Liver abscesses may also be part of a general pyæmia. The infection in such cases is carried by the hepatic artery. To reach this vessel the infective emboli must first traverse the lung, but do not necessarily lodge here on account of the large size of the pulmonary capillaries compared with those of other parts of the body.

When abscesses in the neighbourhood of the liver invade it from without, they probably follow the course of the lymphatic spaces.

**Tropical abscess** is usually solitary and situated in the upper part of the right lobe. The direction in which these abscesses tend to rupture is determined by their anatomical relations. Owing to the common situation of the abscess, it usually bursts upwards into the lung or pleural sac, for it is in close contact with the dome of the diaphragm, and in addition many of the lymphatics of the liver pass towards the diaphragm between the layers of the coronary ligament. A considerable number of abscesses burst into the peritoneum, and some, extending from the under surface of the liver in the region of the right kidney pouch, may open into the stomach, duodenum, colon, or even the

inferior vena cava or bile ducts. The pericardium is perforated in some instances, or pus, burrowing between the layers of the coronary ligament, may travel down behind the peritoneum and reach the pelvis of the right kidney.

Pain on the top of the shoulder may occur in liver abscess. The pain is localised in the angle between the clavicle and acromion process. The phrenic nerve which is distributed to the diaphragm and in part to the liver, arises chiefly from the fourth cervical root, and filaments from the same root help to form the supraclavicular branches of the cervical plexus. This anatomical fact may account for the peculiar localisation of the referred pain.

The base of the right lung is hollowed out and fits on the convex surface of the liver like a cap, the diaphragm and pleura intervening. A large abscess, projecting from the upper part of the right lobe of the liver posteriorly, may give rise to a dome-shaped area of dulness which encroaches on the pulmonary resonance. If fluid is effused in the intervening pleural sac the characteristic outline of the dulness is obscured. A dull area of similar outline may be found in the axilla when the abscess is situated more laterally. The normal lower border of pulmonary resonance is a horizontal line which corresponds to the sixth costal cartilage in front, the sixth rib in the nipple line, the eighth rib in the axilla, the tenth rib in the scapular line, and the tenth dorsal spine posteriorly. When the liver is enlarged in the upward direction the base of the lung is pushed upwards, but the level of the lower border of the pleural sac remains practically unchanged. The lower limit of the pleura extends from the mid-line in front across the seventh costal cartilage; it reaches the eighth rib in the nipple line, the lower border of the tenth rib in the axilla, and the level of the twelfth dorsal spine behind. The fixed position of the lower limit of the pleura under all circumstances is accounted for by the presence of the strong phrenico-pleural ligament which passes between the point of attachment of the diaphragm to the adjacent costal cartilages and the lower limit of the pleural sac.

Abscesses in the usual position in the right lobe are best reached by the transpleural route.

**Tuberculosis of the Liver.**—The routes by which tubercle bacilli may reach the liver are the same as those already mentioned as giving access to pyogenic infections. Miliary tuberculosis is due to infection which is carried to the liver by the hepatic artery. Tuberculous pericholangitis, curiously enough, is probably a portal infection, the bile ducts being invaded from the portal radicles which lie alongside the ducts in the portal canals. Tuberculous infection of the liver along the lymphatics is rare, for the liver is not interposed in the lymph stream from the intestines to the thoracic duct.

**Syphilis of the Liver.**—The liver is more likely than any other viscus to be the site of gummata in acquired syphilis. They mostly lie on the anterior surface, this being exposed to slight injuries; and in the right lobe, which, being the larger, offers a wider field for infection. Gummata in the region of the portal fissure may compress the bile ducts and portal vein, giving rise to jaundice, ascites, and splenic enlargement, but these symptoms are uncommon. Fever may be present. Gummata are usually accompanied by perihepatitis, and if this is extensive, the action of the diaphragm may be interfered with, and partial collapse of the base of the right lung occur. At the same time pain may be reflected along the phrenic nerve to the branches of the cervical plexus, which supply the point of the shoulder. In such cases the condition is apt to be confounded with subdiaphragmatic suppuration or basal pneumonia.

**Carcinoma of the liver** is usually secondary to some part of the alimentary track which is drained by the portal vein. Venous transfer is unusual in carcinoma, but growths in the intestine are prone to ulcerate, and may open up the portal radicles in this way. Moreover, as already pointed out, the liver lies off the track of the main lymphatic stream from the intestines. Carcinoma may reach the liver by other routes, for it is sometimes secondary to growths in the breast, or may extend from the gall bladder or pylorus by direct continuity.

Carcinoma of the liver can extend along the lymphatics of the round ligament, and so may give rise to secondary nodules at the umbilicus. It may also infect the glands in the portal fissure, and so produce jaundice and ascites.

**Sarcoma**, which is of rare occurrence primarily within the portal area, is equally rare in the liver as a secondary growth.

#### DISEASES OF THE GALL BLADDER AND BILE DUCTS.

The bile duct opens into the duodenum on the tip of a small nipple-like projection, which is sometimes known as the papilla of Vater. Within the papilla, whilst traversing the duodenal wall, the duct presents a terminal dilatation which usually, but not always, receives the termination of the main duct of the pancreas. The dilated portion of the duct is known as the ampulla of Vater. The papilla is situate on the inner and posterior aspect of the descending portion of the duodenum, about four inches from the pylorus. Infection from the duodenum can gain access to the common duct through the papilla, and ascend through the cystic duct to the gall bladder, or through the hepatic ducts to the liver. The pancreatic duct may be infected at the same time on account of its relation to the ampulla.

The cystic duct near its junction with the gall bladder describes an S-shaped curve, and this, together with the duplicatures of mucous membrane which form Heister's valve, accounts for the ease with which it is obliterated by inflammatory changes in its mucous membrane. When the cystic duct is obstructed in this way, the gall bladder becomes distended with the mucous secretion of its own glands.

The fundus of the gall bladder usually projects against the abdominal wall at the upper part of the right linea semilunaris, and when the viscus is distended, may be felt as a rounded and superficial tumour at the outer border of the right rectus muscle, just internal to the ninth costal cartilage (Fig. 49, p. 362). Unless it is firmly fixed by inflammatory lesions, the gall bladder follows

the respiratory excursions of the liver. When inflamed, even though its outline cannot be distinctly felt, its position may often be discovered by the local tenderness elicited by its descent

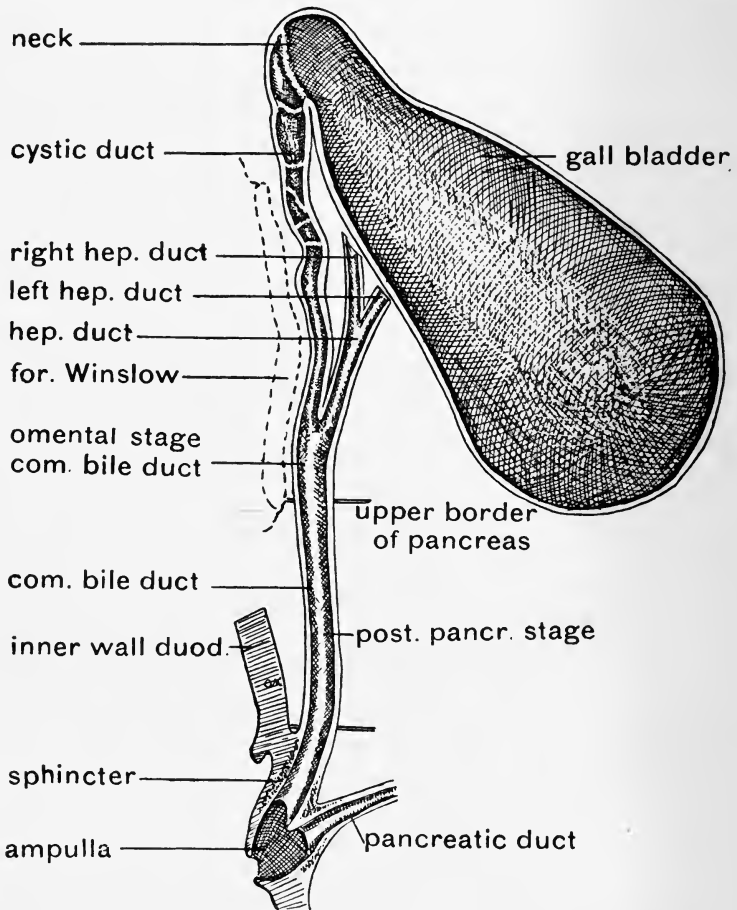


FIG. 51—THE GALL BLADDER AND BILE DUCTS VIEWED FROM THE RIGHT. (*Hughes and Keith.*)

against the fingers if a full inspiration be taken. A greatly distended gall bladder forms a pyriform tumour which can be moved laterally and also antero-posteriorly, but cannot be separated from the liver above, or restrained from following its up



and down movements. This is owing to the fact that the stalk of the gall bladder is closely connected to the liver by its duct, and also that, except in rare cases where a mesentery to the gall bladder exists, the upper surface of the gall bladder is closely attached by connective tissue to the fossa of the liver in which it lies. Owing to the restraint exercised by its attachments to the liver a greatly distended gall bladder may assume a forward curve, and then its shape has been likened to a cucumber. The transverse colon or even coils of small intestine may ride up over the fundus of the gall bladder and lie between it and the liver. In such cases, a resonant area may separate the fundus from the liver dulness. Inflation of the stomach tends to carry the enlarged gall bladder upwards and to the right, owing to the pressure exerted on it by the pylorus and first part of the duodenum, whilst inflation of the colon usually displaces it upwards.

The nerves of the gall bladder are derived from the eighth and ninth dorsal segments of the cord, through the great splanchnic nerve and the cœliac plexus. When the gall bladder is inflamed, superficial tenderness may be present over an area corresponding closely to the fundus, and another in the right subscapular region at the level of the eleventh dorsal spine; the former is the site of maximum tenderness of the ninth segmental area, the latter is situated in the eighth dorsal segmental field. These tender areas are often met with in cases of gall stones. (Figs. 20-23, p. 226.)

Large gall stones may pass direct from the gall bladder into the bowel by a process of adhesion and ulceration. Since the under surface of the gall bladder lies in direct contact with the duodenum and the transverse colon, it is into one of these that the stone is likely to be discharged. Occasionally it makes its way into the neighbouring pylorus. The fundus of the gall bladder being directly applied to the anterior abdominal wall may become adherent to it, and ulcerate into its deepest layer. The purulent contents may then be extravasated into the connective tissue which ensheaths the round ligament of the liver and point in

the neighbourhood of the umbilicus. Gall stones have been discharged this way.

The resistance to the passage of gall stones along the ducts is greatest where the cystic duct unites with the hepatic to form the common duct, and where the latter opens into the duodenum. The common duct, where it lies in the edge of the lesser omentum, is normally about the size of a crow quill. When obstructed at its duodenal end it has become so dilated as to be mistaken for the gall bladder itself. The cystic and common ducts, being comparatively narrow tubes, may become the sites of stricture. The stricture is more commonly found in the cystic duct owing to the anatomical characters which have already been described as favouring its occlusion. In cases of obstruction, the liver may become honeycombed with dilated ducts and much increased in size.

The lymphatic glands which receive the lymphatics of the gall bladder and bile ducts share in their inflammation, and may become so enlarged and hard that they have been mistaken for gall stones or malignant growths. A gland which lies on the cystic duct near the neck of the gall bladder is nearly always enlarged. This is the cystic gland, and it receives the lymphatics from the gall bladder. Another fair sized gland is usually found near the junction of the cystic and hepatic ducts, and it is succeeded by a chain of glands which lie along the common duct in the edge of the lesser omentum. These compose the "satellite chain of the bile duct," and the lymph which they transmit finally reaches the glands at the upper border of the pancreas.

Pylephlebitis may result from gall stones. The portal vein is in least intimate relation with the common bile duct in the middle of its course. Above and below it is in close relation and in some cases is actually applied in spiral fashion to the upper part of the common bile duct on the right side of the latter. The portal vein may be infected by ulceration into it of a gall stone, or by infection of the small radicles it derives from the gall bladder.

The peritoneal adhesions set up by inflammations of the gall bladder may involve the duodenum, transverse colon or pylorus, all of which are adjacent. Gastric pain and dilatation, or pain in the region of the colon, may result.

The part of the peritoneum first infected as the result of inflammation or rupture of the gall bladder is the right kidney pouch, which lies above the transverse meso-colon. The omental stage of the common bile duct has been known to rupture into the lesser sac, which is rendered possible by the position of the duct in front of the foramen of Winslow.

A stone lodged in the lower part of the ampulla of Vater may ulcerate through the duodenal wall into the bowel, or cause a retroperitoneal perforation. Rarely it infects the peritoneum in this region. Stones in this neighbourhood are in a position to obstruct also the pancreatic duct or cause regurgitation of bile into it.

#### DISEASES OF THE PANCREAS.

**Inflammation of the pancreas**, whether acute or chronic, is usually a consequence of infection of the duct. The inflammation may spread to it from the duodenum or from the bile duct in common with which the pancreatic duct usually opens. A gall stone impacted at the lower end of the bile duct may impede the outflow from the pancreatic duct and lead to infection, or if near the orifice of the biliary papilla, may actually cause bile under pressure to regurgitate into the pancreas and set up acute inflammation in this way. On the other hand the swelling of this gland when acutely inflamed may cause compression of the common bile duct. Drainage of the bile ducts is sometimes adopted as a means of indirectly draining the ducts of the pancreas, but can only be effectual when both open into the ampulla of Vater.

It is probable that in some cases the pancreas is infected by the blood stream. The only direct connexion between the parotid gland, the pancreas and the testicle, is vascular, and all these structures may be inflamed in mumps. It is possible that the virus

of other infective diseases, such as typhoid fever, may really gain access to the gland in this manner.

Extension of inflammation from closely related structures sometimes produces a localised pancreatitis. Gastric and duodenal ulcers, tumours of the pylorus and other organs, disease of the lumbar vertebræ and aneurysms of the aorta or cœliac axis may in this way set up an inflammation.

The depth at which the pancreas lies in the abdomen and the

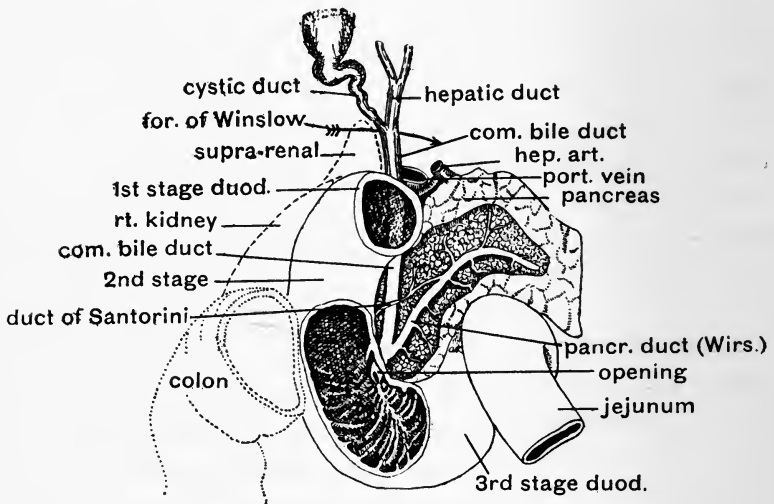


FIG. 52.—THE HEAD OF THE PANCREAS, DUODENUM, AND COMMON BILE DUCT. (*Hughes and Keith.*)

presence in front of it of the stomach, render it difficult or impossible to recognise moderate degrees of enlargement of the gland. Indeed, it is usually impossible to feel the gland at all.

Acute pancreatitis usually shows itself as an acute peritonitis in the upper part of the abdomen, involving the lesser sac, which covers the front of the gland, or that part of the greater sac which is in contact with its lower border. The pancreas is in close relation with the great nerve plexuses of the abdomen, which may explain the extreme pain and collapse which is so characteristic of acute inflammation.

The splenic vein lies along the back of the gland and receives tributaries from it, so it is surprising that portal thrombosis is not more commonly associated. The fat necrosis which accompanies acute pancreatitis is attributed to the escape of the fat-splitting ferment into the tissues of the immediate neighbourhood, but sometimes is observed in remote situations, in which cases the ferment may have been carried by blood vessels or lymphatics.

In hæmorrhagic pancreatitis the bloody infiltration is liable to extend beyond the pancreas in the retroperitoneal tissues, involving in this way the roots of the mesentery and transverse mesocolon, the colic retroperitoneal tissue and the perinephritic tissue also.

Sclerosing inflammation of the pancreas leads to compression of the termination of the common bile duct and also of the pancreatic duct. It is rare for the portal vein to be implicated since it is not in the substance of the gland. In the interacinous form of the disease the gland is much more closely permeated with connective tissue than in the interlobular form, and consequently the interacinous cell islets are more likely to be destroyed. It is possible that the destruction of these cell islets in the tail of the organ has some influence in inducing diabetes.

**Calculi in the pancreas** lead to dilatation of the duct of Wirsung, so that it can easily be traced through the gland. They may be associated with pancreatitis. When dilated, the pancreatic duct has been known to form fistulous communications with the lesser sac, or the stomach, or duodenum. When an accessory duct of the pancreas is present, it usually opens into the duodenum three-quarters of an inch above and somewhat ventral to the main pancreatic duct. The accessory duct is very variable, and so not of great clinical importance. When present it may compensate for a blockage of the main duct.

**Abscess of the pancreas** may form as the result of inflammation or injury. Such an abscess may bulge into the lesser sac and, pushing forwards the lesser omentum, present between

the liver and the stomach; or it may push the stomach forwards or present below it, above the transverse colon. Following the connective tissue planes it may insinuate itself between the layers of the transverse mesocolon and, reaching the lumbar region, surround the kidneys. The duodenum, colon and stomach are the parts of the intestinal tract into which a pancreatic abscess is most likely to perforate, particularly the two latter on account of their close relationship to the head of the gland.

**Pancreatic cysts** may arise when the main duct is obstructed by a pancreatic calculus, or by a gall stone in the ampulla of Vater, or by external pressure. The relations of the pancreas to the peritoneum have considerable influence in determining the directions in which cysts may extend and present themselves. The greater part of the head and body of the gland is in immediate relation with the lesser sac. Consequently pancreatic cysts usually project into the lesser sac, either pushing the stomach in front of them or presenting above, between it and the liver, or below, between it and the transverse colon. But pancreatic cysts may project into the greater sac; thus a cyst arising to the right of the foramen of Winslow may bulge in the right hypochondrium, simulating a cyst of the kidney or suprarenal body or a distended gall bladder. A cyst springing from the head of the gland below the transverse mesocolon may present below the hepatic flexure of the colon and be mistaken for a renal or colic tumour; the root of the mesentery will prevent such a cyst from encroaching on the left half of the abdomen. It is possible for a cyst arising from the uncinat process of the gland to appear to the left of the root of the mesentery, below the transverse colon, where it may simulate a tumour of the left kidney, or one arising from the colon in this situation or from the small intestine. If large it may even simulate an ovarian cyst. Cysts in some cases burrow between the layers of the mesentery or of the transverse colon when they spring from the gland opposite the point of reflection of the peritoneum to form these folds. When cysts arise from the posterior



TOPOGRAPHY OF ABDOMEN.

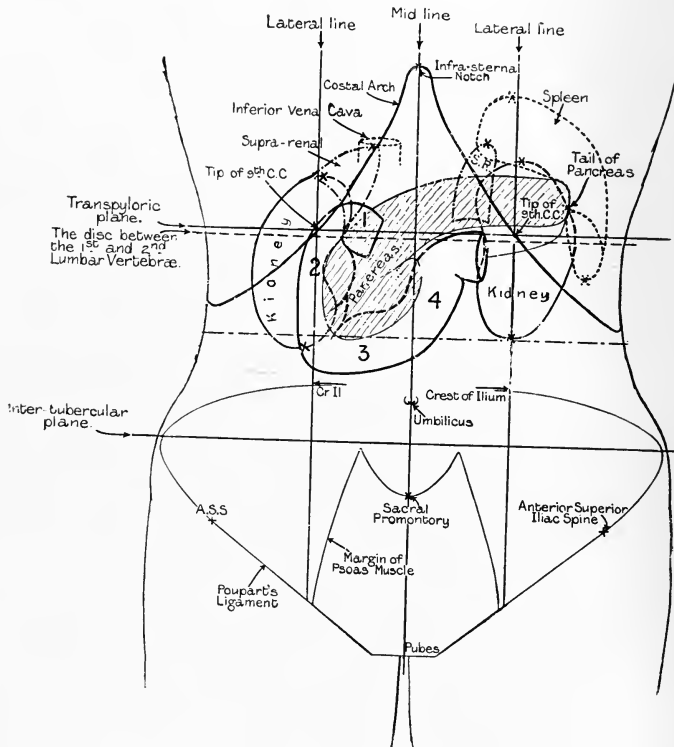


FIG. 53.—Diagram showing the disposition of the deep organs in the regions of the abdomen. (From Ellis's Anatomy, after Christopher Addison.)



part of the gland, retroperitoneal extension may occur, the cyst invading the lumbar region.

Pancreatic cysts, from their position, may transmit the pulsation of the aorta, or compress the inferior vena cava or the portal vein. They have been known to cause obstruction of the small bowel. They must be distinguished from encysted fluid in the lesser sac of the peritoneum, such as may result from crushing of the pancreas against the vertebral column.

**Carcinoma of the Pancreas.**—Since the head of the pancreas is the usual seat of cancer in this organ, pressure symptoms are pronounced. The intimate relation of the common bile duct to the head of the gland accounts for its early obstruction with the production of jaundice, distension of the gall bladder, swelling of the liver from retained bile, and rarely, attacks of colic indistinguishable from those due to gall stones. The infiltrated pancreas is not easily felt, and is usually concealed by the distended gall bladder. Occasionally, however, it can be recognised as a deep-seated tumour above the umbilicus, for it lies in front of the second lumbar vertebra, whilst the umbilicus corresponds to the body of the fourth. The tumour may transmit the pulsations of the aorta, which lies behind it, and may ultimately give rise to compression of the portal vein, the splenic vein, or the inferior vena cava, all of which are also in posterior relation with the gland. Ascites, swelling of the spleen, or dropsy of the legs and lower part of the abdomen, may thus be produced. The intimate relation of the duodenum to the head of the pancreas accounts for the occasional invasion of this part of the bowel. The transverse colon which crosses the head of the pancreas, is sometimes infiltrated. The pylorus also is not far distant from the head of the gland, but it is more common for cancer of the pylorus to become adherent to the pancreas than for cancer of the pancreas to invade the pylorus. The cancer being in portal territory secondary growths may be expected in the liver, but the patient often dies before metastasis occurs. The chain of lymphatic glands along the pancreas also becomes involved. When malignant disease invades the tail of the pancreas, which is uncommon, it is not

favourably situated to produce pressure symptoms, but left hydronephrosis may occur from compression of the upper end of the ureter. A growth in the tail of the pancreas can sometimes be excised, but a growth in the head has such intimate relation to important structures that excision is practically out of the question.

## CHAPTER XVII.

### DISEASES OF THE URINARY SYSTEM.

#### DISEASES OF THE KIDNEY.

**Congenital Abnormalities.**—The renal bud, arising as it does from the dorsal aspect of the Wolffian duct near the cloaca, may be said to have a pelvic origin. In the process of growth it assumes a position behind the peritoneum of the lumbar region. The kidney sometimes remains in the pelvis between the rectum and bladder, in other instances it lies on the sacral promontory below the bifurcation of the aorta, or may be found on the sacro-iliac synchondrosis, on the venter of the ilium, or abnormally low in the lumbar region. In all such cases the kidney is firmly fixed and the vessels are short, being derived from the arterial and venous trunks in the immediate neighbourhood, the vessels wandering into the organ during development. Fusion of the renal buds may occur, giving rise to a solitary kidney, which is usually of abnormal shape. The horse-shoe form is commonest, the kidneys being united across the spinal column by their lower poles. Occasionally the fused renal mass lies on one or other side of the spine.

The suprarenal bodies do not participate in displacements of the kidneys, but are usually found in their normal positions.

**Congenital cystic kidneys** consist of a congeries of retention cysts, which largely replace the renal substance. The cysts vary up to three-quarters of an inch in diameter, and are supposed to be cystic dilatations of Wolffian remanants included in the kidney substance proper. Minute masses of tissue having the structure of the suprarenal body are also occasionally found embedded in the kidney. These are known as "adrenal rests."

**Injuries to the Kidney.**—The kidney being retroperitoneal, it is possible for it to be extensively lacerated as the result of blows, falls and other injuries, without the peritoneum being involved. Being supported by the bodies and transverse processes of vertebræ, and also by the last rib, it may be crushed against these bony points by violence applied in front. The impressions left in a hardened kidney by the twelfth rib and transverse processes of the first and second lumbar vertebræ show how intimate the relation is. If the proper capsule of the kidney is torn, the extravasated blood is mainly checked by the renal fascia which forms the outer boundary of the fatty capsule; if the blood escapes through this, there is but little obstacle to its wide dissemination in the connective tissue planes of the abdominal wall. Extravasated urine is usually present, as well as blood, and may be in large quantity if the renal pelvis is torn. Hæmaturia often occurs. Rents of the peritoneal covering when present add much to the gravity of the condition.

In acute flexion of the spine the kidney may be squeezed between the ilium and the lower ribs.

The kidney may be injured from behind by a penetrating wound without implication of the peritoneum, but this is very unlikely when the wound penetrates from in front.

**Perinephritic abscess** is situated in the retroperitoneal tissue in which the kidney lies, and must be distinguished from localised intraperitoneal suppuration in front of the kidney. Perinephritic abscesses may be primary, such as occur from injury or during the course of certain infectious diseases, or may arise by extension of suppuration in the neighbourhood. The infection may extend from a lesion of the kidney, such as calculus, tuberculosis, or abscess, or may originate in the bones of the spine, or arise in connexion with the vermiform appendix, colon, pancreas, or duodenum. In the case of the vermiform appendix, the peritoneum will have been perforated in some manner; but with extension from the colon, pancreas and duodenum, this is not essential, since all these have areas free from the serous membrane. Sometimes perinephritic infection spreads

from the pelvic connective tissue, or from above the diaphragm, and conversely the pus of a perinephritic abscess may extend into the pelvic connective tissue, or invade the subpleural tissue, and so the pleura and lung. It rarely bursts into the peritoneum, but may open into the colon, or present in the lumbar region.

**Movable kidney** may, or may not, be associated with general prolapse of the abdominal viscera. The presence of a mesonephros as a cause of mobility is rare. More commonly the kidney slips up and down behind the parietal peritoneum. Normally the kidneys, being deeply situated in the loins, cannot be easily palpated, save in very spare persons, but with care that the abdominal muscles are well relaxed and bimanual palpation, the difficulty may be got over. In the adult the right is three-quarters of an inch lower than the left, but its lower pole is above the level of the umbilicus, and an interval of an inch and a quarter usually separates it from the iliac crest. (Fig. 53, p. 419.)

The factors which keep the kidney in position are its fascial capsule, the attachments of the renal vessels, the pressure which the abdominal muscles exert on it through the medium of the abdominal viscera, and possibly also the reflections of peritoneum on its anterior surface. The fatty capsule of the kidney is an infiltration of the retroperitoneal tissue with fat, and the outer layers of this capsule are condensed into a firm fibrous layer known as the renal fascia. These layers fuse along the outer border of the kidney and above, but do not closely unite below it. The renal fascia is firmly attached to the crura of the diaphragm, to the aorta, and the posterior abdominal wall. The same connective tissue surrounds the renal vessels, forming with them a strong pedicle. The pressure exerted by the muscles of the abdominal walls has an important influence in keeping the kidney in place, and the anterior surface of the organ when hardened *in situ* shows two inclined planes which slope from the middle towards each pole. These planes indicate the direction of the pressure exerted by the superjacent viscera, the upper plane corresponding to pressure exerted from above, and the lower to supporting

pressure from below. The bed on which the kidney lies is an inclined plane which, in the erect position, slopes downwards and forwards towards the iliac crest. The bed is narrower and less vertical in women. The kidney normally slides a short distance down the inclined plane of its bed in inspiration.

Absorption of the retroperitoneal fat, yielding of the muscles of the abdominal wall or pelvic floor, and disarrangement of the planes in which pressure is exerted in consequence of tight lacing, all conduce to the occurrence of movable kidney. The greater frequency with which the right kidney is involved is no doubt due to the superincumbent mass of the liver. The frequent occurrence of movable kidney in the female sex is accounted for by the shallow and more vertical kidney bed, coupled with the influence of tight lacing and pregnancy.

The excursion of which a movable kidney is capable is limited by the length of its vascular pedicle. When displaced from its proper position, the corresponding loin may feel less full and less resistant.

The symptoms which may accompany nephroptosis are determined by the relations and connexions of the kidneys. The displaced organ may exercise either pressure or traction on neighbouring parts of the intestinal tract. The descending portion of the duodenum may become sharply bent by the agency of peritoneal bands which pass from the anterior surface of the kidney to the duodenum opposite the point of entrance of the bile duct. The duodenum may be carried downwards with the kidney, and become kinked by the traction exerted on it by the hepato-duodenal ligament. The kidney may assume such a position as to exercise direct pressure on the duodenum or pylorus. By any of these agencies dilatation of the stomach may be produced. Transitory jaundice may be induced by traction on the bile duct or by pressure. The ureter may become twisted, flexed, or otherwise obstructed, and the renal veins may suffer in the same way. Thus hydronephrosis, renal congestion, renal atrophy, and even renal thrombosis, may arise.

Pain may occur in various forms. In some instances it

occurs as renal colic; in others it is referred to the spine and termed "rachialgia;" sometimes it is reflected along the nerves of the corresponding abdominal segments, producing modifications of tactile sense, and in others it radiates down the outer side of the thigh, or appears in the knee, the heel, or the sole of the foot. The pains are in part due to a stretching of the renal plexus, which is an offset of the solar plexus and accompanies the renal vessels. The nerve supply of the kidney is derived from the tenth, eleventh and twelfth dorsal and first lumbar segments of the cord, through the splanchnic nerves. Renal pain may be referred along any of the sensory nerves derived from these segments. In some instances, the displaced kidney exercises direct pressure on branches of the lumbar plexus, *i.e.*, on the ilio-hypogastric, ilio-inguinal, genito-crural, external cutaneous or anterior crural nerves.

**Nephritis.**—Infective or toxic matter may reach the kidney by the blood stream, the ureter, or the lymphatics. Blood infection is probably the cause of most cases of nephritis. The toxic material may have been ingested or be the product of the activity of bacteria in some other part of the system. The cortex of the kidney contains the active secretory epithelium, and the blood is chiefly distributed to this part. Consequently the stress of inflammation falls chiefly on the cortex, involving the glomeruli and the convoluted tubes. The blood supply of the convoluted tubes is derived from the afferent vessels of the glomerular tufts.

The tough renal capsule resists the swelling of an acutely inflamed kidney, and thus may produce further damage. The favourable results of nephrotomy in acute nephritis may be attributed to diminution of renal tension. The tension may induce acute renal colic simulating that produced by calculus. The practice of wet cupping the loin in acute nephritis is justified by the free connexion which exists between the superficial vessels of the kidney, the subperitoneal vascular plexus of Turner and the lumbar vessels.

Infection which ascends the ureter usually gives rise to a pyelitis before nephritis supervenes, and cystitis may precede

both. The urinary tubules become directly infected from the calyces into which they open. Ascending infections are generally suppurative, and occur in connexion with urethral strictures, prostatic enlargements, and infections of the lower urinary passages.

The kidney is occasionally infected by the lymphatic route. The lymphatics of the ureter communicate directly with the lymphatics which lie between the urinary tubules. Hence infection can travel from the urinary bladder along the lymphatics of the ureter to the kidney. Extension by this track explains those cases in which the renal cortex is alone involved, the pyramids and pelvis remaining free.

**Tuberculosis of the kidney** may be the consequence of infection carried to the organ by the blood stream or reaching it along the ureter, the ureteric lymphatics, or from structures with which the organ lies in direct contact.

In miliary tuberculosis the tubercles are widespread and originate in the small vessels. But tubercle bacilli brought to the kidney may be excreted and may find a nidus in the glomeruli or tubules during this process; a chronic renal excretion tuberculosis may originate in this way. Presumably the number of organisms carried to the kidney in such cases is not very great. In the earlier stages of excretion tuberculosis the cortex is mainly involved.

When infection ascends the ureter from below it naturally tends to attack and erode the lower parts of the pelvis first, spreading to other parts of the kidney later. Occasionally infection appears to ascend the lymphatics which accompany the ureter and in such cases the pelvis may at first escape.

The kidney has been known to be directly infected by a sub-jacent psoas abscess. The direct anatomical connexion of the kidneys, ureters, bladder, prostate, vesiculæ seminales and epididymis has much influence in determining the distribution of tuberculous lesions, and particular attention should be paid to all these structures when investigating a case of renal tuberculosis.

The tuberculous thickening of the ureter can sometimes be



detected by palpation. In old standing tuberculous disease the kidneys become pyonephrotic and are matted to surrounding structures. Their adhesion to the vena cava inferior on the right side and to the aorta on the left may give rise to considerable difficulty when excision is attempted.

**Renal calculi** usually lie in the pelvis of the kidney. The calyces, which are diverticula of the pelvis, have narrow lumina, so a stone lying in a calyx may be well concealed, and may actually become embedded in the renal substance. The contraction of the pelvis, where it joins the ureter, offers a great obstacle to the passage of calculi, which becoming wedged in the narrow outlet give rise to partial or complete obstruction of the passage. If the obstruction is complete and permanent the kidney becomes atrophic; if incomplete or recurrent hydro-nephrosis will result. Irritation of the renal pelvis by the stone accounts for the presence of albumen, pus and blood in the urine.

The kidney lies on the psoas and quadratus lumborum muscles, and movements which necessitate the action of these muscles will aggravate the pain, or sometimes elicit it if absent. Pain may also be induced by making firm pressure on the front of the pelvis of the kidney. A point on the anterior abdominal wall, a finger's breadth internal to the tip of the ninth costal cartilage fairly indicates the position of the pelvis. This point lies on the transpyloric line. (Fig. 53, p. 419.) Another method of seeking for the presence of calculus is to make forcible percussion of the loin, upwards and inwards, just below the tips of the last two ribs; this procedure sometimes produces a sharp, darting pain.

Two varieties of pain may occur with renal calculus, one fixed and localised, and the other intermittent and radiating. The latter is renal colic. The fixed pain is usually in the position of the kidney, but may be referred to some distant spot supplied by a branch of the lumbar plexus. Thus it may be localised in the testicle or the groin, being referred along the genito-crural nerve, in the leg or inner side of the foot being referred to a branch of the anterior crural nerve, or in the domain of the great sciatic nerve posteriorly. The pain of renal colic, on the other hand, shoots

round the lower part of the loin to the front of the abdomen, and thence to the testicle, groin or inner side of the knee, following the direction of the branches of the lumbar plexus. The pelvis of the kidney and the ureter, where the pain originates, are particularly associated with the eleventh and twelfth dorsal and first lumbar segmental areas. (Fig. 20-23, p. 226.) Cutaneous hyperæsthesia may be expected to occur in these areas during and after a paroxysm of colic. The skin of the scrotum, which is supplied from sacral segments, does not become tender, but the testicle does; the implication of the latter may be explained by the presence of filaments of the genito-crural nerve in the tunica vaginalis, which is said to be the only serous membrane supplied by a spinal sensory nerve.

The connexion of the vagus nerve with the renal plexus may account for the nausea and vomiting which may be associated with renal calculi.

The anterior and posterior arterial vascular areas of the kidney are separated by the pelvis and do not anastomose. As a rule three-quarters of the kidney belong to the anterior vascular area and the remaining quarter to the posterior. The direction of the axes of the posterior row of calyces indicates the plane of division between the two territories. The veins are not arranged in the same way, but all ultimately pass to the front of the pelvis. A longitudinal column of renal substance, consisting of fused columns of Bertini, and bearing large vessels, intervenes between the anterior and posterior sets of pyramids. In incising the kidney to search for stone it is well to keep behind this column. The least damage to vessels is done by an incision parallel to the posterior surface of the kidney a little behind its convex border; this will pass between the two vascular territories and open the posterior calyces. It should be carried into them in front of the posterior row of papillæ to avoid section of the collecting tubules of the pyramids.

**Ureteric Calculus.**—There are three narrow points in the ureter at which a stone is liable to become arrested. Of these one is just below the renal pelvis; the second is at the pelvic brim



STRUCTURES WHICH LIE BENEATH THE SPINO-UMBILICAL LINE.

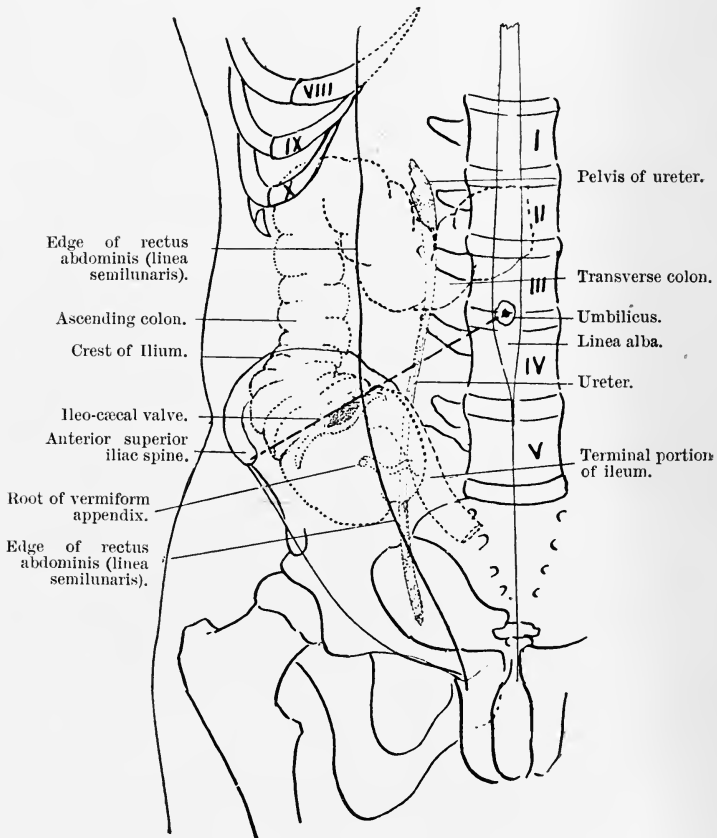


FIG. 54.—Diagram of the average position of the ileo-cæcal valve, cæcum, appendix, and right ureter with relation to the line from the right anterior superior spine to the umbilicus. (From Sir Frederick Treves's Cavendish Lecture, 1902. After Keith.) The spino-umbilical line runs from the anterior superior iliac spine to the umbilicus. The point where it crosses the outer edge of the right rectus is called "Monro's point," and corresponds in position to the ileo-cæcal valve. The base of the vermiform appendix lies rather more than an inch below this. The ureter crosses the spino-umbilical line at the point of junction of its middle and inner thirds. The eleventh dorsal nerve enters the sheath of the rectus near Monro's point. The genito-crural nerve lies close to the ureter.

where the ureter describes a curve known as the flexura marginalis; the third is where the tube traverses the muscular wall of the bladder. It is not as a rule possible to palpate the abdominal part of the ureter. This part descends vertically from the kidney in the umbilical region about an inch and a half from the mid-line. The junction of the outer and middle thirds of a line joining the anterior superior iliac spines corresponds to the flexura marginalis,

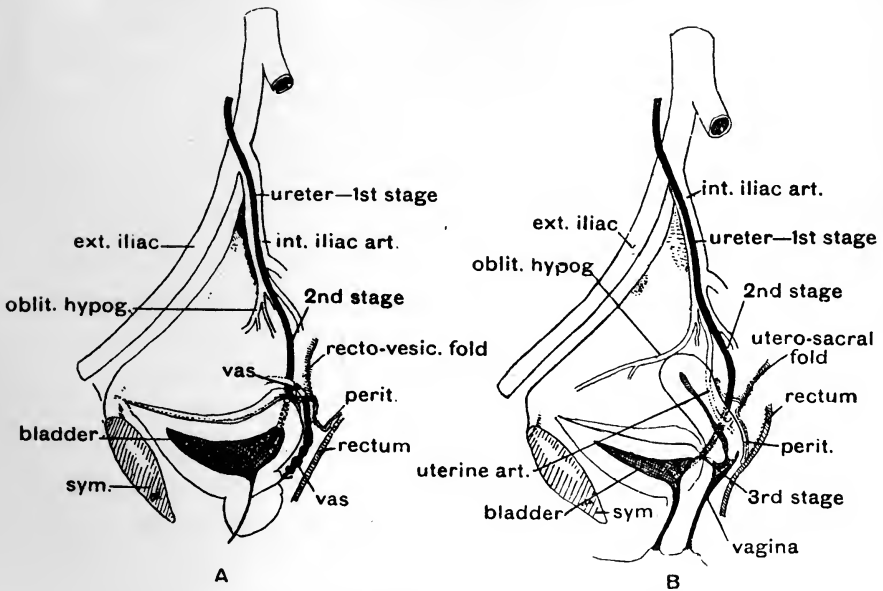


FIG. 55—DIAGRAMS OF THE PELVIC COURSE OF THE URETER IN THE MALE (A) AND FEMALE (B). (*Hughes and Keith.*)

or portion of the ureter which crosses the brim of the pelvis in front of the bifurcation of the common iliac artery. It is said that the first part of the pelvic portion of the ureter can be palpated from the rectum in both sexes in the knee-elbow position. In the female the terminal part of the tube passes above the lateral fornix of the vagina, then crosses the side of the latter to enter the bladder at a point corresponding to the middle of the anterior vaginal wall. It can easily be palpated from the vagina. The ureter is retroperitoneal in the whole of its course, and can

be reached, without opening the peritoneum, from the lumbar region, the iliac region, or the vagina. Calculi have also been extracted from its lower end through the urinary bladder.

In **hydronephrosis** and **pyonephrosis** the pelvis of the kidney is dilated. This is due to some obstruction to the exit of urine from it. Whether one or both kidneys suffer will depend upon the site of the obstruction. If the impediment is in the urethra or at the neck of the bladder both kidneys will be involved. Pelvic lesions outside the bladder, near the trigone, may also involve both ureters. The ureters run in the pelvic connective tissue, towards the base of the bladder, coming into fairly close contact with the cervix uteri. They may both be obstructed by pelvic cellulitis, carcinoma of the cervix uteri, or uterine prolapse.

Unilateral hydronephrosis may be due to obstruction of the renal pelvis or of the ureter below this. It is particularly associated with mobility of the kidney and is often intermittent in such cases. An abnormal renal artery or vein may pass across the upper part of the ureter and obstruct it, or the obstruction may be due to an impacted calculus or pressure from without. The relation of the first part of the pelvic colon to the left ureter explains how the latter may become obstructed by a carcinoma in this situation. The ureter as it passes into the pelvis follows the course of the internal iliac artery and forms a posterior boundary of the ovarian fossa; consequently it is at times compressed by malignant tumours of the ovary.

**Enlargements of the Kidney.**—The distinguishing characteristics of renal enlargements are all based upon the anatomical features and relations of the kidneys.

The outline of a renal swelling is rounded and has no sharp edges, conforming to the normal contour of a kidney; it may thus be distinguished from enlargement of the spleen.

The position of the colon with regard to the kidney is usually maintained when the latter is enlarged. The presence of the bowel in front of the swelling can sometimes be determined by palpation and at others by percussion if the bowel happens to contain gas, or has been artificially inflated from the rectum.

The hepatic flexure of the colon lies in front and to the inner side of the lower end of the right kidney, whilst the splenic flexure lies in front and to the outer side of the left, coming into relation with it nearer the level of the hilus so that a considerable length of descending colon passes down in front of it. This difference in the relation of the colon to the kidney on the two sides accounts for the fact that colic resonance is more often demonstrable over a left than a right renal swelling. Bowel never passes between the spleen and the parietes, the colon lying on the inner aspect of this viscus, and only under exceptional circumstances does large or small intestine override the liver.

The kidneys lie closely applied to the spinal column on each side and no area of resonance can be demonstrated between a renal tumour and the spine, whereas theoretically it should be possible to elicit a triangular area of pulmonary resonance, with the apex downwards, between the vertebral end of a large spleen and the spinal column. The thick muscular mass at the side of the spinal column renders this sign, however, of but little use.

The close proximity of the kidneys to the spine also explains the fact that there is no appreciable interval between a renal swelling and the vertebræ of the lumbar region.

The directions in which an enlarged kidney is seen to project are also determined by its surroundings. It cannot project backwards because the bed on which the kidney rests consists of the psoas, the quadratus lumborum and the diaphragm, and to the resistance offered by these muscles is added the support of the lumbar transverse processes, the last rib and the arcuate ligaments. Extension of the tumour directly inwards is resisted by the prominence of the vertebral bodies, whilst extension upwards is prevented by the action of the diaphragm. The kidney as a result tends to enlarge forwards and outwards, projecting forwards at or near the level of the umbilicus below the position of the liver or stomach, and also extending towards the flank. Although a renal enlargement, except in certain cases of infiltrating malignant disease or of perinephritic suppuration, does not bulge backwards, yet it can be felt to "fill the loin"

and so contrasts with swellings of the gall bladder or spleen which are more superficially placed. A word of warning, however, is necessary with regard to massive splenic tumours, for these too tend to fill the loin and can be felt through the lumbar muscles if of sufficient size.

The kidneys normally move slightly in respiration, following the movement of the diaphragm. Renal enlargements also show the respiratory excursion unless fixed by adhesion. Owing to the fact that the kidneys are not so closely related to the dome of the diaphragm as the liver and the spleen are, the respiratory excursion of the latter viscera and of the fundus of the gall bladder, is more marked.

As a renal tumour increases it may obstruct the passage of blood along the left spermatic vein in the male by stretching the vessel in front of it. A gradually increasing varicocele may be produced in this way. The right spermatic vein does not open into the renal vein like the left, but joins the inferior vena cava at a lower level, consequently it is not liable to be compressed by an enlargement of the right kidney.

#### DISEASES OF THE BLADDER.

**Cystitis.**—Inflammation of the mucous membrane of the bladder is most commonly the outcome of infection. Micro-organisms usually reach the bladder in one of three ways:—First, ascending along the urethra, the distance being comparatively long in the male and short in the female—though the same anatomical fact may serve to explain the reason of the more complete and prolonged retention of organisms within the bladder in the male as compared with the female; secondly, descending from a focus within the kidney, as, for instance, in the secondary infection of the bladder from a tuberculous pyelitis; and, thirdly, although rarely, through the blood stream.

The rugose condition of the mucous membrane makes it difficult to dislodge the bacteria even by repeated and thorough washings out.



The pain in acute cystitis is felt in the supra-pubic region from the position of the bladder behind the symphysis in the pelvis, but it is also referred to the end of the penis and to the perineum, owing to the common nerve supply from the fourth sacral.

**Hypertrophy of the Bladder.**—Hypertrophy of the muscular coat of the bladder is, as a rule, the outcome of obstruction to the passage of urine through the urethra. When it occurs the detrusor urinæ exhibits considerable enlargement of its fibres, so as to produce distinct fasciculation. The firm, projecting ridges of hypertrophied muscular fibres may often be felt by a sound introduced into the bladder, and may even be mistaken for calculi or growths.

The mucous membrane between these fasciculi of muscular tissue is apt to bulge, from the pressure caused by the retained urine, which bulging amounts in many cases to an actual hernia of the membrane. Such protrusions are known as *sacculi*, and they may occur at those parts of the bladder uncovered or covered by peritoneum. If they attain a fair size, and particularly if the entrance into them becomes narrowed, urine is apt to stagnate within them, and calculi may be formed. In other cases a *sacculus* may give way under pressure, and urinary extravasation occur, possibly into the peritoneal cavity, or into the cellular tissue of the pelvis or into that lying behind the anterior abdominal wall, the so-called cavity of Retzius.

**Distension of the Bladder.**—The empty bladder lies entirely below the brim of the true pelvis, but when it becomes distended it rises up into the abdomen, and may then reach as high as the umbilicus. During this distension the reflection of the peritoneum from its anterior surface by the urachus and the obliterated hypogastric arteries is carried somewhat upwards, and an interval is left between the level of the symphysis pubis and the peritoneum, through which the organ can be tapped or suprapubic cystotomy performed, without the peritoneal cavity being traversed. In childhood the bladder is almost entirely out of the pelvis proper.

**Rupture of the Bladder.**—Laceration or rupture of the bladder may be caused by traumatism or over-distension. Gun-shot injuries, fractures of the pelvis, rough usage of instruments, particularly sounds or lithotrites, may all cause perforation of the undistended or only slightly filled organ. External violence such as a kick or blow, coming upon the distended viscus, without the intervention of the protection of the contracted recti abdominis, may induce a rupture of that part of the bladder wall which is covered by peritoneum. Further, it is possible, though not common, for over-distension to spontaneously cause a giving way of the vesical wall, either where covered or uncovered by peritoneum, and this is particularly liable to happen in those cases where the distension occurs in a hypertrophied and sacculated bladder.

When the rupture is into the peritoneal cavity and sterile water is injected into the bladder, this will find its way through the rent into the larger space beyond. Hence it follows that if a measured quantity is introduced, only a portion of this will be withdrawn, thus establishing the fact of the perforation of the vesical wall.

**Retention of Urine.**—Retention of urine within the bladder is caused by complete obstruction to its outflow along the urethra, and is therefore due to pressure from without, disease in the walls of the tube itself, or blockage from within.

One of the commonest causes of complete retention is organic stricture of the urethra, the anatomical connections of which are discussed later. Enlargement of the prostate gland is also a not uncommon factor in producing retention, and is also subsequently dealt with. Here the obstruction of the urethral passage by a calculus may be alluded to.

Particularly in children, a calculus which is either formed in the bladder or passed down the ureter from the kidney, is flushed out by the flow of urine from the bladder into the urethra, and, as a rule, is arrested in the narrowest part of that tube, that is to say, just within the lips of the external meatus. In order to extract the impacted stone it is well to remember that since

the meatus is the narrowest part, the opening should be enlarged in a direction upwards into the glans, and that the penis should be seized on the proximal side of the stone, so that the calculus may not slip into the higher and wider part of the urethra during its removal.

If the urine cannot be withdrawn per urethram, the bladder may be aspirated or tapped immediately above the symphysis pubis, seeing that the retention of urine has distended the bladder and elevated the peritoneal reflection; or it is possible to withdraw the urine by tapping through the rectum, above the prostate and through the trigone at the base of the bladder, which is also uncovered by peritoneum.

**Specific Urethritis (Gonorrhœa).**—Inoculation of the mucous membrane of the urethra with the gonococcus occurs at the lips of the meatus externus or just within the fossa navicularis. From this starting place the whole length of the tube may become involved by direct continuity of infection, and beyond it the bladder and even the ureters and the kidneys may suffer from the invasion. The openings of the glands in the mucous membrane form convenient nesting places for the cocci.

The lymphatics of the urethra absorb the septic material, and convey the organisms, both gonococci and staphylococci, to the upper set of inguinal lymphatic glands, and sometimes also to the lower set, thus inducing the formation of a bubo. The gonococci reaching the prostatic portion of the urethra may enter one or other of the common ejaculatory ducts, and may travel along this tube, pass the entrance of the duct from the vesicle and through the whole length of the vas, to eventually reach the globus minor of the epididymis, there to cause inflammation or epididymitis. The vas itself is usually involved in the inflammatory process and is thus thickened, and often the surrounding tissues of the cord become œdematus. In some instances, before the actual inflammation of the epididymus occurs, pain may be experienced on defæcation, owing to the hardened fæces pressing upon the lower end of the vas and the seminal vesicle, both of which structures may have been already

infected. Pain also in the inguinal canal may indicate an inflamed condition of that part of the vas which lies therein.

The sub-mucosa of the urethral wall is abundant, hence the marked swelling which occurs in urethritis, leading, not infrequently, to an obstruction of the channel and acute retention of urine. In relieving such retention by the use of a catheter, one of a large size should be employed, so as to press aside the swollen mucous membrane, and not to pierce the soft tissue.

**Stricture of the Male Urethra.**—The urethra in the male commences at the internal meatus at the neck of the bladder, the lowest part of the trigone, and ends at the external meatus at the termination of the glans penis. Between these two points in the adult the prostatic portion measures a little more than one inch, the membranous a little less than one inch, and the penile about five inches. It is therefore obvious that a catheter to reach the bladder by way of the urethra, and to project into it, must be at least eight inches in length for the fully-developed male.

The channel is narrowest at the external meatus, this being a vertical slit. An instrument which will pass through this aperture will traverse any portion of the rest of the normal urethra, even the membranous part which comes second in narrowness. Just within the external meatus is a wide area of the urethra, owing to the fossa navicularis found in its floor, and in the roof the lacuna magna, in which the tip of a small catheter may be caught.

Numerous mucous glands open, in depressions or lacunæ, into the distal portion of the penile urethra. It is these pits which are so liable to be hiding places for micro-organisms, particularly the gonococcus, and casts of these sulci often appear in urethritis, being washed out by the flow of urine.

In the bulbous portion of the urethra just below the anterior layer of the triangular ligament may be seen the openings of the ducts of Cowper's glands, the glands themselves being situated between the two layers of the ligament, on either side

of the membranous part of the tube. Only mucous glands open into this latter part of the urethra.

The prostatic portion is wide, and on its floor is found an elevation, on the summit of which there opens the sinus pocularis, and on each of its sides the common ejaculatory duct. The urethra terminates in the bladder at a spot which, in the adult, is one inch behind the upper border of the symphysis pubis, and three inches from the skin surface of the perineum. The urethra is partially covered with voluntary muscles, the bulbo-cavernosus around the bulbous part, and the constrictor urethræ (deep transversus perinei) about the membranous portion.

The passage of a catheter or other instrument along the healthy urethral channel may be obstructed first by the narrowness of the external meatus, and then by the actual curvature of the tube. Thus it becomes desirable to draw up the penis over the symphysis in the early stage of passing a catheter, so as to convert the double curve of the passage into a single long one. During its passage the tip of a catheter may lodge in the lacuna magna, or against a possible fold of mucous membrane, caught up as it were by the instrument. Its progress also may be impeded by the spasmodic contraction of the peri-urethral muscles. Frequently the point of the catheter may be run against the superficial layer of the triangular ligament, not properly engaging the entrance into the membranous portion of the urethra. Beyond this spot there is little to interfere with the onward passage of the instrument in the normal tube. When, however, there is an enlargement of the prostate, particularly when a so-called middle lobe is hypertrophied, much obstruction at the entrance into the bladder may be met with.

Organic stricture of the urethra is most common at the junction of the bulbous with the membranous portion of the urethra, and this is accounted for by the fact that the urethra passes through the anterior layer of the triangular ligament at this spot, that annular ulcers of gonorrhœal origin are most frequently

found here, and that the urethra is usually torn by injury in this region. Stricture may also occur in the penile part, but it never occurs in the prostatic portion.

**Extravasation of Urine.**—Extravasation of urine is most usually caused by a giving way of the wall of the urethra. This may be induced by, (1) a peri-urethral abscess, generally the result of gonorrhœa, and usually on the proximal side of a stricture, opening into the urethra, thus allowing urine to be forced into the emptied cavity of the abscess during micturition, and if the pressure is great the extravasation may be widespread: (2) laceration of the urethra as the outcome of falls astride, or blows upon the perineum. Here blood will pass from the meatus independently of micturition, and should the patient attempt to pass urine, some of it will invariably be extravasated through the rupture; (3) perforation of the wall of the urethra by the forcible use of instruments; (4) the giving way of the dilated and perhaps even ulcerated wall of that portion of the urethra immediately proximal to the site of a stricture; (5) lacerations of the urethra associated with fracture of the bones of the pelvis.

If it is the membranous part of the urethra which has given way, urine will pass into the substance of the compressor urethræ muscle between the two layers of the triangular ligament, and theoretically, should be limited to this space, but usually transgresses it in a superficial direction. Extravasation, however, is far more common in front of, that is superficial to, the anterior layer of the triangular ligament. Here there is a space which is securely limited behind and at the sides, but open in front. It is sometimes spoken of as the superficial compartment of the urogenital triangle, and has the following boundaries, which readily serve to limit the progress of extravasated urine, and direct its advance in one direction only. Superficially are found the skin and two layers of superficial fascia. The superficial layer of this fascia is continuous with the subcutaneous tissue of the thighs, scrotum, penis and the lower part of the anterior abdominal wall. The deep layer of this fascia is distinctly

membranous in character. It is definitely attached laterally to the periosteum of the rami of the ossa pubis and the ischia, thus preventing extravasated urine from finding its way into the tissues of the thighs. Posteriorly it winds round the hinder border of the transversus perinei muscles, to become blended with the margin of the base of the triangular ligament, thereby effectually hindering the escaped urine from passing back into the perineum proper. In front, however, it is as it were unattached, and is continuous with the same layer covering the scrotum, the penis, and the lower part of the abdominal wall. From this anatomical arrangement it will be readily seen that urine, extravasated by rupture of the bulbous portion of the urethra, will find its way directly into the tissue beneath this deep layer of the superficial fascia. Further, the urine will be forced to extend forwards, being prevented from going backwards or outwards by the attachment of the deep layer of the superficial fascia, and upwards towards the pelvic cavity by the anterior layer of the triangular ligament. It will therefore creep forwards into the scrotal tissue, on to the penis, rising up on to the anterior abdominal wall, and reaching, if allowed, as high even as the level of the axilla. It will not descend from the hypogastric region into the thighs more than to just below Poupart's ligament, on account of the attachment of this deep layer of the superficial fascia to the fascia lata two fingers' breadth below the ligament. The urine readily induces an intense inflammation with great swelling, and unless quickly drained out by free incisions will cause sloughing. The incisions may be, and should be, made deeply into the infiltrated tissue, without fear of doing harm.

## CHAPTER XVIII.

### DISEASES OF THE GENITAL SYSTEM.

#### MALE.

##### THE TESTIS AND EPIDIDYMS.

**Orchitis.**—Inflammation of the testis may be due to the invasion of the organ by bacteria, or to its injury by external violence.

The body of the testis is most commonly the site of inflammation due to the syphilitic virus. The investment of the body of the testis by the peritoneum forming the tunica vaginalis indicates the reason why a collection of fluid around the organ, secreted by the serous membrane, is often seen in gummatous orchitis. The presence of hydrocele in such cases also serves to prevent the adhesion of the parietal and visceral layers of the tunica vaginalis. Thus the process of inflammation but rarely extends to the skin of the front of the scrotum, and hernia testis in this condition is rare. The dense, firm, unyielding nature of the tunica albuginea explains the severity of the pain felt when acute orchitis is present.

**Epididymitis**, when occasioned by infection, is due to the invasion by gonococci from the urethra along the vas to the globus minor, or the deposit of tubercle bacilli, probably brought by the blood stream.

Seeing that the epididymis is only partially covered with serous membrane, its posterior aspect being destitute of it, it is easy to understand why an inflammation of the epididymis may quickly involve the overlying tissues, and lead to hernia testis.

**New Growths of the Testicle.**—In malignant growths of the testicle it has to be remembered that the lymphatic glands which are secondarily infected are those found in the lumbar



region near the kidney, because the testicle was originally developed within the abdomen in this region. Hence it follows that it is not easy to determine whether or not these glands have become involved in the early stages of malignant deposit in the testis.

**Imperfect Descent of the Testis.**—The testis may be arrested at any stage in its “descent” into the scrotum, or it may descend but pass elsewhere than into the scrotum. The first abnormality is termed arrest, whilst the second is known as ectopia.

There are several anatomical peculiarities which may lead to arrest of the testis. Its blood-vessels may be too short, the vas may not be long enough, the mesorchium, or fold of peritoneum attaching the testis to the dorsal wall, may be so broad as to give the testis so much play that it may never engage the deep abdominal ring, or the testis may be tethered by intra-abdominal adhesions.

Again, the testis or the epididymis or both may be too large to permit of the passage of the gland along the inguinal canal or through the superficial ring.

And, lastly, the pull of the gubernacular fibres may be too feeble owing to their lack of development.

In ectopia testis the organ is most commonly found at the side of the anus in the perineum. It is probably drawn there by a band of gubernacular fibres, which can be felt fixed to the junction of the rami of the os pubis and ischium. In other instances it is drawn by gubernacular fibres into Scarpa's triangle, when the spermatic cord can be felt passing through the superficial ring.

**Hydrocele.**—The processus vaginalis may remain patent in its whole length, and peritoneal fluid draining into the pouch constitutes a typical *congenital hydrocele*. In many of such cases the actual communication with the general peritoneal cavity is but small, and consequently it may be difficult to reduce the fluid and impossible for abdominal viscera to descend—hence the presence of a hydrocele and the absence of a hernia.

The processus vaginalis may be obliterated at any spot between its commencement in the parietal peritoneum and the summit of

the testicle. Normally it becomes closed throughout the whole length between these two points, and then a collection of fluid in the tunica vaginalis, surrounding the testis, between its parietal and visceral layers constitutes the common *vaginal hydrocele*.

Failure in the complete obliteration in any portion of the length between the site of the deep ring and the commencement of the tunica vaginalis leaves a potential cavity which may become distended with fluid. This will constitute the so-called *encysted hydrocele of the cord*.

Supposing the processus vaginalis becomes closed at the site of the deep abdominal ring but remains patent the whole way below this, and its cavity becomes distended with fluid, there is present what is termed an *infantile hydrocele*.

On the other hand, given that the processus vaginalis becomes obliterated about the region of the superficial abdominal ring or slightly below it, remaining patent and in communication with the abdomen on the proximal side, fluid trickling into this portion will constitute a *funicular hydrocele*.

In tapping a common vaginal hydrocele it is important to remember the relationship of the body of the testis to the fluid. In by far the larger number of instances the testis lies below and behind, and the trochar and cannula introduced from the front to a depth not greater than half an inch should not touch the anterior surface of the organ. Occasionally, however, the testis is placed in front of the tunica vaginalis, in a position liable to injury, but its presence at this spot may be determined by the position of the shadow cast when testing for translucency.

#### THE SPERMATIC CORD.

**Inflammation of the Cord.**—In considering the question of inflammation of the spermatic cord it may be well to note the various structures which enter into its composition.

The first is the vas, together with its artery, usually derived

from the inferior vesical, and its veins, which empty themselves into the vesical plexus, these vessels being derived from and terminating in an altogether different region to those belonging to the testicle, although there is a definite anastomosis between them. Then follow in order of importance the spermatic vessels, the artery being derived from the abdominal aorta, immediately distal to the renal arteries, while the veins empty themselves on the right side into the inferior vena cava and on the left into the left renal vein. Next there are the spermatic nerves, derived from the renal and aortic plexuses; hence it is that severe pressure upon the testicle is apt to induce serious collapse, and that a calculus in the kidney may be associated with pain referred to the testis. The spermatic lymphatics found in the cord pass up to the lumbar lymphatic glands. In addition to the above there are also in the cord the remains of the gubernaculum testis in the form of unstriped muscle fibres, and the obliterated portion of the processus vaginalis.

Inflammation of the cord may occur either through the vas or the veins.

A vas which is transmitting bacteria is liable to have its own walls infected, and the inflammation may spread beyond into the tissues surrounding it. Therefore in certain cases, particularly those of tuberculosis, the vas itself is thickened from the actual tuberculous deposit in its walls, and not infrequently becomes nodular to the touch; whilst in cases of gonococccic invasion the parts external to the vas itself sometimes become markedly infiltrated and indurated, and in some of these instances a true phlebitis of the spermatic veins may result.

On the other hand a blow upon, or stretching of, the spermatic cord may give rise to some extravasation of blood and subsequent inflammation. Occasionally the vas itself may be ruptured, either in the inguinal region or in the extra-peritoneal tissue of the iliac fossa, and this may give rise to a dribbling of blood from the urethra altogether apart from micturition, owing to the direct connection of the vas with the prostatic portion of the urethra by way of the common ejaculatory duct.

## THE PROSTATE GLAND.

**Prostatitis.**—The prostate is situated at the meeting place of the genital and the urinary tracts; hence it follows that there are three sources through which it may become infected and inflammation result. First, by way of the urethra, and particularly by the gonococcus; secondly, along the excretory tube of the testicle and through the common ejaculatory duct, and especially by the tubercle bacillus: lastly, along the ureter through the bladder and the first part of the urethra, either by septic or tuberculous organisms. The orifices of the numerous glands of the prostate and the wider aperture of the sinus *pocularis* afford convenient nesting-places for the bacteria derived from any one of these channels.

Inflammation of the prostate leads to severe pain, on account of the denseness of its capsule, and frequently to retention of urine, because of the obstruction to the urethral passage from the bulging forwards of the floor of the tube.

Pus within the substance of the prostate most commonly finds its way into the urethra or into the bladder, through the lower part of the trigone; occasionally into the rectum, in front of which the prostate lies, only separated by its capsule and some loose cellular tissue; still more rarely, through the skin of the perineum.

In opening a prostatic abscess from the perineum it is important to warn the patient that urine will almost certainly flow through the wound, seeing that if the abscess has already partially evacuated itself into the urethra, the wound in the perineum will constitute a direct tract from the urethral passage to the skin.

**Enlargement of the Prostate.**—It would be out of place to discuss the various theories as to the exact etiology and histology of enlargement of the prostate. It will be sufficient to show how this enlargement anatomically alters the outflow of urine.

The two lateral masses of the prostate—often called lobes—are united together by a thin commissure anterior to the urethra,

but more firmly behind that channel, the urethra itself traversing the organ. Hence it follows that any increase in size of the prostate must necessarily lead to alterations in the direction and size of the urethra.

But while this is true, it is the projection of the enlarged prostate into the vesical cavity which, as a rule, causes most inconvenience, and the subsequent serious results. In enlarging, the tissue increases in the line of least resistance, and not infrequently this is towards the interior of the bladder. Hence there projects upwards a so-called middle lobe which is in reality an asymmetrical outgrowth of a lateral lobe. This tends to block the exit from the bladder, and particularly so when the patient strains during the act of micturition. It may very easily lead to complete retention of urine, necessitating speedy relief.

The passage of a catheter along the urethra into the bladder may be rendered difficult by the point of the instrument impinging against this projecting mass from a lateral lobe. Hence it is advantageous to use a *coudé* or elbowed catheter, so that it may mount up over the elevation and pass more readily into the bladder beyond.

Removal of an enlarged prostate, either by the suprapubic or perineal route, is in many cases probably the enucleation of adenomata which have compressed the prostatic tissue itself into a very thin layer. It is, however, theoretically possible by inserting the finger between the prostatic sheath proper and the visceral layer of the pelvic, recto-vesical, fascia, to enucleate the whole of the gland, but in such cases there is a great tendency for considerable hæmorrhage, on account of the damage done to the plexus of veins lying between the two layers of fascia. In either case the contained urethra is usually torn away.

#### FEMALE.

##### THE OVARY.

**Ovarian Cysts.**—The ovary is placed in the peritoneum forming the postero-internal layer of the fold constituting the

broad ligament. Hence it follows that cysts of the ovary proper during their enlargement do not separate the layers of the broad ligament, but increase in an upward direction, lifting the intestines and omentum above them, and having the broad ligament as a pedicle.

The ovarian artery, derived from the abdominal aorta, runs between the layers of the broad ligament just below the site of the ovary itself, and ends by anastomosing with the termination of the uterine artery near the junction of the Fallopian tube with the body of the uterus.

In securing the pedicle of an ovarian cyst the interlocked ligature is tied round the ovarian artery in two places, one between the artery and the pelvic brim, and the other between the ovary and the uterus. Hence it follows that a piece of the vessel itself is excised with the tumour.

Cysts derived from the parovarium, or remains of the Wolffian tubules, lie between the two layers of the broad ligament, and during their increase in size separate them. There is, therefore, no true pedicle to such cysts, and a layer of peritoneum has to be divided in order to remove them.

#### THE FALLOPIAN TUBE.

**Salpingitis.**—The direct communication of the lumen of the Fallopian tube with the exterior through the vagina, the cervix and body of the uterus, explains the fact that micro-organisms not infrequently find their way into it.

Inflammation caused by the invasion of these bacteria may lead to occlusion of portions of the tube and to the formation of pus within the unobliterated parts. If the bacteria extend along the whole length of the tube and through its ostium abdominale, pelvic peritonitis may ensue, and this may lead to a sealing of the fimbriated extremity, thereby preventing the passage of ova through the canal.

On the right side the close proximity which the appendix vermiformis, lying over the brim of the pelvis, has to the ovary

and tube explains why in certain cases of appendicitis the pain and other symptoms are mistaken for disease of the uterine appendages. It is of course possible on the other hand for septic inflammation of the right tube to extend to the appendix lying near it, but the reverse—that is to say, inflammation passing from the vermiform appendix to the broad ligament—is more common.

**Tubal Pregnancy.**—The ovum arrested in the Fallopian tube may become impregnated, and a tubal pregnancy result. The increase in the size of the ovum and the growth of its membrane may erode the blood vessels supplying the part. The consequent hæmorrhage ploughs up the tissue of the broad ligament, producing a hæmatoma, or ruptures the tube or the broad ligament, and allows of free hæmorrhage into the peritoneal cavity.

#### THE UTERUS.

**Prolapse.**—Prolapse of the uterus is practically a hernia of the organ together with a portion of the pelvic floor.

Procidentia is normally prevented by the support which the uterus receives both from above and below.

From above, the round ligament, passing from the upper part of the lateral aspect of the body of the uterus, extends across the anterior part of the broad ligament into the inguinal canal, and serves as a very adequate support.

The broad ligaments, directed outwards and then backwards, reach the pelvic wall, to which they become attached. Their lower borders are separated from the muscles constituting the pelvic floor by a layer of connective tissue supporting the ureter and a number of large veins passing away from the uterus and the vagina.

Lastly, the utero-sacral ligaments extend backwards from the level of the supravaginal portion of the cervix, along the side of the pelvis, to the front of the rectum. These form the upper limit of the pouch of Douglas.

From below the uterus is supported by the perineal body and

the visceral layer of the pelvic fascia, together with the muscles forming the floor of the pelvis, particularly the levator ani, on each side.

Coincidentally with the descent of the uterus in prolapse, the bladder is drawn downwards, and the vaginal wall may protrude at the vulva, constituting a cystocele. Similarly, but later, the rectum may be displaced together with the posterior vaginal wall, forming a rectocele.

When the prolapse is great, the ureters may be so stretched as to interfere with the passage of urine through them, and double hydronephrosis result.

**Metritis.**—The serious consequences which result from septic inflammation of the body of the uterus are readily explained anatomically.

The blood from the uterus is returned through a large plexus of veins in the broad ligament, which eventually empties its contents into the internal iliac vein. In septic metritis following upon parturition, the venous spaces in the uterine wall become filled with blood clot teeming with micro-organisms, and gradually an infective thrombosis extends proximally to the internal iliac, and even the common iliac veins on one or both sides. Should the common iliac veins become blocked with a septic clot, phlegmasia will occur.

Inflammation of the wall of the uterus may extend laterally into the tissue between the layers of the broad ligament, constituting pelvic cellulitis or parametritis. Or it may progress so as to involve the peritoneal covering, thereby inducing pelvic peritonitis or perimetritis.

Pus originating from inflammation of the uterus may, if left to itself, find its way out through the vagina, or may track upwards between the layers of the broad ligament, so as to reach the iliac fossa and point above the middle of Poupart's ligament, or may evacuate itself through the rectum.

**Myomata.**—Myomata of the uterus may be said to exist in three varieties, according to the position in which they are found—interstitial, subperitoneal, and submucous.



With the enlargement of an interstitial myoma, the mucous layer of the wall of the uterus will be separated from the serous layer. The entire uterus increases in size, and the cavity of the body is lengthened. The uterine and ovarian arteries become considerably enlarged to supply the new growth. The veins in the broad ligament are correspondingly dilated, and large venous sinuses are formed in the capsule of the tumour.

When the myoma develops in that part of the uterine wall close to the broad ligament, particularly the region of the supravaginal cervix, it may extend outwards and become intraligamentous.

The subperitoneal variety tends to become pedunculated, and as a result of traction or violence the pedicle may be twisted or actually torn through. When the subserous myoma is formed from the anterior aspect of the uterus, it not infrequently carries the vesico-uterine fold of peritoneum upwards, and now and again the bladder will be stretched out on the surface of the tumour, and the ureter on one or both sides considerably displaced.

When the myoma commences beneath the uterine mucosa, it tends to become extruded into the cavity of the organ, and may present as a pedunculated growth. The uterus will endeavour to extrude what is really a foreign body, and consequently the pedicle will become more and more lengthened, and the vascular supply considerably diminished; hence it is that gangrene of the myoma frequently occurs.

The enlargement of the whole uterus tends to produce pressure upon and displacement of the surrounding structures. Interference with micturition and defæcation are by no means uncommon. Hæmorrhoids may also be induced by the interference with the return of blood from the mucous membrane of the rectum. Pressure upon nerve trunks, where the growth occupies and is somewhat impacted in the pelvic cavity, will often induce very severe pain. Pressure upon the ureters may result in obstruction and dilatation of the tubes, and subsequent hydronephrosis and even uræmia.

**Carcinoma.**—There are four varieties of epithelium from which carcinoma may arise—the stratified epithelium of the vaginal portion of the cervix, the columnar epithelium of the cervical canal, the epithelium of the cervical glands, and the epithelium of the glands imbedded in the body of the uterus.

Whilst it is true that an epithelioma commencing in the cervix may involve the vagina below and the uterus above, the important fact concerning the spread of the disease is that the carcinoma extends in a lateral direction into the parametrium, or the tissue at the base of the broad ligament. It is this extension which renders futile any operation unless it is undertaken in quite the early stages of the disease. The close relation of the ureter to the supravaginal portion of the cervix, as it passes forwards and inwards, explains why the tube is liable to be injured during operations in this region.

It is in this situation that the growth tends to rapidly involve the ureters, and produce obstruction of these tubes.

As it progresses anteriorly it will invade the bladder, which may eventually be ulcerated into, a vesico-uterine or vesico-vaginal fistula being thereby caused.

By the growth of the neoplasm into the uterine veins, particles may become detached and carried by the blood stream to the heart and lungs, inducing rapid dissemination and growth of secondary deposits. The lymphatics may also convey the cells, carrying them so as to infect the iliac group of glands, and later those placed along the aorta, distal to the origin of the renal arteries.

#### THE VAGINA AND VULVA.

**Vulvitis and Vulvo-vaginitis.**—Invasion of the vulva, and subsequently of the vagina, by micro-organisms, particularly the staphylococcus and gonococcus, is very frequent, and on account of certain anatomical peculiarities it is difficult to entirely eradicate them.

Bacteria may lie latent in the various folds and depressions of the labia, remnants of the hymen, and rugæ of the vagina, as

well as within the ducts and cavities of the vulvo-vaginal glands and even the cervical canal or uterus.

Absorption of septic products by the lymphatics of the vulva induces lymphadenitis in the inguinal region; absorption by the lymphatics of the lowest part of the vagina also infects the inguinal glands, but absorption by those of the upper part of the vagina involves the iliac lymphatic glands.

#### THE MAMMARY GLANDS.

**Mastitis** (inflammation of the nipple).—Inflammation of the nipple is common during lactation. The skin surmounting the nipple, although covered by stratified epithelium, is delicate and becomes easily cracked. Fissures infected by bacteria thus readily arise and may be extremely painful, sensations being conveyed by the third and fourth thoracic nerves. In the areola around the nipple are numerous large sebaceous cysts, which are sometimes the seat of inflammation which may terminate in suppuration giving rise to one form of supra-mammary abscess.

**Mammitis** (inflammation of the mamma or breast proper).—The usual variety of mammitis is the acute, caused by septic absorption from the nipple. The summit of the nipple is slightly fissured, and opening upon it are the orifices of the radiating milk ducts which, in the adult, and particularly during lactation, are sufficiently large to admit a bristle. Just beneath the surface the ducts are somewhat dilated. Each duct represents the excretory tube of a lobe of the gland, there being some twelve to fifteen lobes and ducts. Each lobe is separated from its neighbour by an intervening septum of fibrous tissue, often called a suspensory ligament. It will thus be seen that there is a direct and open channel along which organisms may pass from the surface to the depth of the gland, through the orifices of the milk ducts, and along the ducts themselves to the acini of the lobes. There is also another track along which infection may occur, namely the lymphatic, setting up an acute lymphangitis of the mammary tissue.

In whichever way the infection proceeds, it is usual for more than one lobe to be involved. Should suppuration occur, two or more foci of pus will be formed, which are, at any rate in the earlier stages, quite distinct from one another, being separated by the interlobular trabeculæ. This fact is of the greatest importance in the effectual treatment of intramammary abscesses, for it indicates the necessity there is of breaking down the intervening wall between the two cavities, each containing pus, in order to ensure proper drainage. It is for this reason that most of these abscesses require a general anæsthetic to allow of the necessary manipulation being satisfactorily carried out. All incisions should be made radiating from the nipple to avoid severance of the ducts, and should be placed so as to secure the most thorough drainage, the exact site being dependent on the position which the patient will for the most part assume subsequent to the operation. Owing to the mobility of the gland, and of the pectoral muscle beneath the gland, it is often necessary, in order to obtain more rapid healing, that the breast be bandaged back against the thorax, and the arm be kept bound to the side.

**Carcinoma.**—The terrible frequency with which carcinoma affects the mammary gland in the female, and the extensive operations undertaken for its removal, make a knowledge of the anatomy of the organ most essential. There are three varieties of epithelium in connection with the mamma from any of which carcinoma may arise. First there is the stratified epithelium covering the nipple itself. Epithelioma of the nipple starts from this. Secondly, there is the cubical or columnar epithelium of the ducts, from which so-called “duct carcinoma” appears to have its origin. Thirdly, the spheroidal or glandular epithelium of the acini is the origin of the varieties of spheroidal-celled carcinoma, scirrhus, medullary, and atrophic.

The limits of the mammary tissue proper are very ill-defined, and by no means consist only of the projection of the breast from the thoracic wall, extending often as high as the level of the second rib, so low as to lie on the upper part of the rectus

and external oblique muscles of the abdomen, as far inwards as the margin of the sternum, and outwards well over the serratus magnus to practically the mid-axillary line. This extensive area must be remembered when it is requisite, as in excision for carcinoma, to remove the whole of the glandular tissue.

Malignant disease of the mamma infiltrates the peri-glandular connective tissue, and disseminates by the lymphatic vessels so abundant in it. Hence it is that three of the most characteristic signs of mammary carcinoma are not infrequently in evidence, although too much stress cannot be laid on the fact that cancer of the breast ought to be diagnosed long before the signs are present, should the patient come at this early stage. Nothing is more reprehensible than to wait for their appearance before warning the sufferer of her serious complaint and advising adequate treatment. The three signs are, (1) The skin becomes adherent, and creases or puckers when moved or pinched over the site of the disease. This is due to the contraction of the fibrous bands, surrounding and invaded by the growth, and passing from the gland to the over-lying skin. (2) The nipple becomes retracted. Again this is due to contraction of the connective tissue around the affected acini, which tissue is continuous superficially with that about the ducts passing through the nipple. (3) Lymphatic glands become palpably enlarged. The gland is everywhere freely supplied with lymphatics which form peri-acinous and peri-ductal networks. Afterwards these are gathered up in the interlobar trunks, and there is a free anastomosis between these latter and the vessels found in the subcutaneous tissue covering the gland, and in the pectoral fascia upon which it lies. The efferent lymphatic vessels from the gland pass to at least three sets of lymphatic nodes. The majority go directly into the anterior axillary nodes, placed beneath the outer margin of the pectoralis major. Through these the lymph may carry the carcinomatous material to the central or deep nodes arranged along the axillary artery and vein, and so to the nodes below and above the clavicle. Other lymphatics, chiefly from the upper and inner quadrant of the mamma, pass directly through the

third, fourth, and fifth intercostal spaces to the nodes lying in the anterior mediastinum. Some from the uppermost part of the gland may pass directly to the glands situated above the clavicle.

From this diffusion of lymph from the mamma, it is easy to understand how impossible it is to be sure that the whole of the disease has been eradicated, when even every lymph node in the axilla and subclavicular region has been extirpated. Apparently also in certain cases the lymphatic vessels of one gland communicate with those of the opposite side across the middle line. "Cancer en cuirasse," or the hide bound infiltration of the skin, is due to the passage of carcinomatous cells along the connective tissue lymphatics from the gland to the skin, and the spread of the growth through the subcutaneous lymph network. This infiltration may possibly be precipitated by the nearest chain of lymphatic glands becoming saturated as it were by malignant deposit or even by the blockage of lymphatic pathways by the removal of the axillary glands.

The extreme swelling of the upper limb often seen in the later stages of carcinoma of the mamma is likewise due to the blockage of lymphatics by malignant deposit.

Death may sometimes be brought about by direct extension of the growth through the thoracic wall to the pleura, a malignant pleurisy supervening which speedily exhausts the sufferer.

Secondary malignant deposits in bones during the course of mammary carcinoma are explained by blood transmission.

## CHAPTER XIX.

### DISEASES OF THE EYE.

#### EYELIDS AND LACHRYMAL APPARATUS.

**Hæmatoma.**—The areolar tissue of the eyelid is so loose that it readily becomes infiltrated with blood after a blow, leading to a characteristic “black eye”; or with inflammatory products in septic conditions in the neighbourhood; or with air in fracture of the nasal bones or of the frontal bone over the sinus.

**Ciliary Blepharitis.**—The large hair follicles, and the associated sebaceous or Meibomian glands, are frequently the site of invasion by bacteria, with resulting acute or chronic inflammation. In consequence of this there may follow a certain amount of ectropion, together with scarring of the edge of the lid. The duct of one of the glands may become blocked, and a chalazion or Meibomian cyst result. This is placed between the conjunctiva and the tarsal cartilage, but it may cause some thinning of the latter, and so present as a swelling which can be seen through the skin of the lid.

A **stye**, or **hordeolum**, is a localised septic inflammation occurring in a hair follicle or Meibomian gland, and the suppurating contents can often be readily evacuated by pulling out the lash from the affected follicle.

**Ptosis** may be due to paralysis of the third nerve, the levator palpebræ superioris becoming inactive, or to paralysis of the seventh nerve producing a relaxation of the palpebral portion of the orbicular muscle.

**Entropion.**—Granular conjunctivitis and spasm of the orbicularis palpebrarum are the two most common causes of inversion of the eyelids. The usual result of entropion is trichiasis, or turning in of the cilia. This will lead to the surface of the

cornea being irritated by the lashes, with consequent ulceration, or pannus.

**Ectropion.**—Ectropion may be due to atrophy of the palpebral portion of the orbicularis or to cicatricial contraction. Eversion of the lower eyelid leads to a faulty position of the lower punctum lachrymale, and therefore to epiphora.

**Epiphora.**—The overflowing of the lachrymal secretion from the conjunctival sac down the cheeks, which is termed epiphora, may be physiological or pathological.

The lachrymal apparatus consists, firstly, of the secreting organ, the lachrymal gland. This is situated in the upper, outer and anterior part of the orbit, resting up against a hollow in the orbital surface of the frontal bone. It is invested in a capsule of fascia which separates it from the rest of the orbital space. The ducts of the gland are from twelve to fifteen in number, and open at the fornix, or reflection, of the conjunctiva beneath the upper lid. The gland is supplied with blood by the lachrymal artery, a branch of the ophthalmic, and with secretory stimulation by the lachrymal nerve, a branch of the first division of the fifth cranial nerve. It is over-stimulation of the gland through this nerve, usually reflexly, which induces physiological epiphora, the drainage apparatus being unable to cope with the superabundance of tears. It is possible to remove the gland for tumour, over-action, incurable epiphora, or other reason without invading the general cavity of the orbit.

The secondary part of the lachrymal system consists of the drainage apparatus for conveying the secretion of the gland out of the conjunctival sac into the nose. Each eyelid near the inner canthus is provided with a minute aperture, the punctum, that in the upper lid, however, being almost functionless. Each punctum rests normally against the ocular conjunctiva, and receiving the lachrymal secretion which has flowed across the front of the eyeball, passes it on into two small channels, the canaliculi, one in each lid. At first these are almost vertical in direction, then, making a sudden bend, they run almost horizontally inwards, above and below the caruncle, to open



either separately or after joining, into the lachrymal sac, situated at the side of the nose in a groove on the lachrymal bone, behind the tendo-oculi and in front of the tensor tarsi muscle. The lachrymal sac opens into the nasal duct, which passes downwards, somewhat backwards and outwards, to terminate in the nose under cover of the anterior part of the inferior turbinate bone. This duct contains as a rule two small valves of mucous membrane. Generally, however, air can be blown from the nose through the lachrymal channels and out through the puncta. Obstruction to any part of the lachrymal drainage system may induce epiphora.

#### THE CONJUNCTIVA.

The exposed position of the conjunctiva readily explains the frequency with which it becomes infected, **conjunctivitis** resulting. Reflex irritation of the orbicularis palpebrarum muscle in conjunctivitis, is a cause of the blepharospasm. The loose cellular tissue of the conjuncture may become greatly infiltrated by inflammatory products leading to chemosis. Blood effused beneath the conjunctiva is not infrequently seen in cases of fracture of the anterior fossa of the base of the skull.

#### THE CORNEA.

Normally the cornea is partially covered by the upper eyelid even when the eyes are open, and wholly covered by the approximation of the lids during their closure. For this reason, as well as owing to the lachrymal secretion, it is well protected and cleansed, and therefore guarded from injury and infection. Its want of blood vessels, however, may be a predisposing cause of keratitis, and it is interesting to see how the surrounding conjunctival blood vessels frequently invade the corneal area for the purpose of absorbing inflammatory products. Thus a leash of vessels can often be seen extending from the conjunctiva to vascularise an ulcer of the cornea.

In the case of **interstitial keratitis** the vessels are derived

from the deeply situated arteries of the adjacent sclerotic, and hence they appear to come out from beneath the limbus. They are often quite indistinct, and of a dirty red or greyish-red colour, because they are covered by the clouded superficial layers of the cornea. They are easily distinguished from the superficial vessels, not only by their position and appearance, but also by the fact that their branches are given off in tufts, like a brush, whilst in superficial keratitis they branch in arborescent fashion. Traces of these vessels can often be detected as thin parallel fibres after all sign of interstitial keratitis has passed off—a point of some importance in determining the presence of congenital syphilis.

The opacities following upon corneal inflammation may be placed in its most superficial layers, when the keratitis has only damaged the surface epithelium. Nebulæ deeper in its substance are most commonly the outcome of interstitial inflammation.

**Perforating wounds** of the cornea, when occurring near the periphery, allow of the escape of aqueous humour from the anterior chamber, and possible prolapse of the iris, particularly when the pupil is dilated; hence the value of eserin for the prevention of the extrusion of the iris. In the same way it may happen that the iris, though not extruded, becomes attached to the posterior surface of the cornea at the site of the perforation, and a permanent anterior synechia is formed. In some instances, after perforation of the cornea, the lens itself is pushed forward during the escape of aqueous, so that its anterior capsule becomes applied to the posterior surface of the cornea. This sets up an irritation which produces a permanent opacity in the anterior capsule, known as an anterior polar cataract.

During the progress of an ulcer of the cornea, its tissue may become so thinned that the normal intra-ocular pressure may induce a bulging forward of the posterior lamellæ, known as keratectasia.

**Wounds of the sclero-corneal junction** are most dangerous when septic, owing to the great risk of so-called “sympathetic ophthalmitis.” It is probable that in the majority of such cases

the inflammation of the sympathising eye is due to invasion by bacteria, and that the tract along which they travel may be as follows:—from the lymphatic spaces of the sclero-corneal junction to the lymphatic tracts in the optic nerve, and so to the optic chiasma: across this structure, and along the lymph vessels of the opposite optic nerve to the opposite eye.

The cornea is supplied by the long ciliary nerves, derived from the nasal branch of the ophthalmic division of the fifth, and associated with inflammation of these nerves, herpes of the cornea may occur, whilst, if the function of the nerves is in abeyance, ulceration of the cornea frequently follows.

#### THE IRIS.

**Iritis.**—The pain in iritis is felt over the distribution of the ophthalmic division of the fifth nerve, hence the supra-orbital and temporal neuralgia so frequently accompanying this disease. The inflammatory œdema of the iris causes it to lose its lustre and acquire a different colour. For the same reason, and because of the increased amount of blood present in the dilated vessels, the pupil becomes somewhat contracted, and at the same time sluggish in its movements. The pink circumcorneal zone, due to the injection of the epi-scleral vessels, is another indication of the congested state of the part.

In plastic iritis the exudation on the posterior surface of the iris brings it in contact with the anterior capsule of the lens, inducing adhesion of the two. Such adhesion produces an irregular pupil, and if the connection becomes organised and thereby permanent, a posterior synechia will be present.

#### THE CILIARY BODY.

**Cyclitis.**—Owing to the extraordinary vascularity of the ciliary processes, inflammation occurring in this region is usually accompanied by a considerable amount of œdema and exudation. The exudate first passes into the posterior chamber, where it

may form a considerable mass, which, in some cases, leads to the adhesion of the whole posterior surface of the iris to the anterior capsule of the lens. Part of the exudate also commonly passes into the vitreous, there to form vitreous opacities; while another part may be carried through the pupil into the anterior chamber. This last portion of the exudate consists of conglomerations of round cells, many of which contain pigment granules, and as the movements of the eye take place they are thrown against the posterior surface of the cornea and become deposited there. The larger cells usually fall to the bottom, and the deposit has a triangular shape with its apex upwards. Owing to the turbidity of the aqueous or the exudation in front of the lens or in the vitreous, vision is necessarily interfered with during an attack of cyclitis. Great pain is also experienced when the ciliary region is palpated. The elevation of the tension during the early stages of cyclitis is due to œdema of the ciliary body blocking up the iridic angle, whilst the lowering of tension in the later stages is to be accounted for by the shrinking of the exudate.

An iridectomy in cases of glaucoma opens up the lymphatic spaces in the ciliary region and allows the absorption of the fluid which is causing the increased tension of the eye.

## CHAPTER XX.

### DISEASES OF THE EAR.

**Foreign body in external auditory meatus.**—The difficulty of extraction of foreign bodies from the external auditory meatus depends partly upon the character of the body introduced, but chiefly upon the anatomical peculiarities of the passage.

The external auditory canal extends from the bottom of the concha to the membrana tympani. It passes in a direction inwards and somewhat forwards; its anterior wall is slightly convex, whilst its floor presents a distinct elevation, which again renders this portion of the passage convex. Owing to the obliquity of the membrana tympani, the lower and anterior walls of the canal are longer than the upper and posterior.

The passage consists of two parts, an external shorter portion composed of cartilage and an internal slightly longer portion bounded by bone. By drawing the pinna upwards and backwards the meatal canal can be made nearly straight—a factor of some importance in connection with the extraction of a foreign body and with obtaining a clear view of the membrana tympani. The length of the canal in the adult is about one and a quarter inches, but it is proportionately shorter in the child. The diameter of the canal naturally varies with age, but is also not quite uniform in its whole length. Two constrictions can usually be demonstrated, one in the cartilaginous portion near its junction with the bony part, and the other a little external to the membrane. Impaction of a foreign body beyond one of these constrictions constitutes a real difficulty in extraction. This impaction may often be aggravated by the swelling caused by the onset of inflammation. If a stream of water can be forced between the foreign body and the wall of the meatus, the object can usually be driven out.

Probably the most frequent seat of arrest is at the junction of the cartilaginous and bony parts of the canal. If syringing fail to remove the intruding substance, instrumental extraction will have to be attempted, and the presence of the *membrana tympani* at the end of the canal must be borne in mind, otherwise severe damage may be done to it.

**Exostosis of the external auditory meatus** usually occurs at the junction of the cartilaginous and osseous portions of the canal. Such a bony outgrowth will have a great tendency to block the meatus, causing retention of secretion and inflammation with deafness.

**Otitis media.**—The middle ear or tympanum communicates with the outer air through the Eustachian tube. Its mucous membrane is thus brought into direct continuity with that of the naso-pharynx, and a ready path for the passage of micro-organisms provided. Seeing the frequency with which infective conditions are met with about the upper portion of the pharynx, it is remarkable how comparatively seldom the tympanic cavity becomes affected. The action of the cilia of the epithelium of the tube in a direction towards the naso-pharynx, tends to thwart the ascent of bacteria. When infection of the mucous membrane of the Eustachian tube occurs, its lumen is quickly obliterated; therefore any muco-purulent fluid poured out into the middle ear, when this becomes involved in the ascending inflammation, has no exit. Consequently the *membrana tympani* is of necessity pressed upon from within, and thereby loses its natural outward concavity, and may even become convex. The fluid in many cases will find its way out through the tympanic membrane by rapid ulceration, seeing that the membrane itself is not more than  $\frac{1}{8}$  of an inch in thickness. As a rule, the perforation takes place in the upper and anterior quadrant, an area which is generally thinner than even the rest of the membrane. It is, therefore, in this region that it is best to perform paracentesis of the cavity. Occasionally the *membrana tympani* has a natural aperture through it in its upper part, which would then readily give exit to purulent fluid.



TEMPORAL BONE.

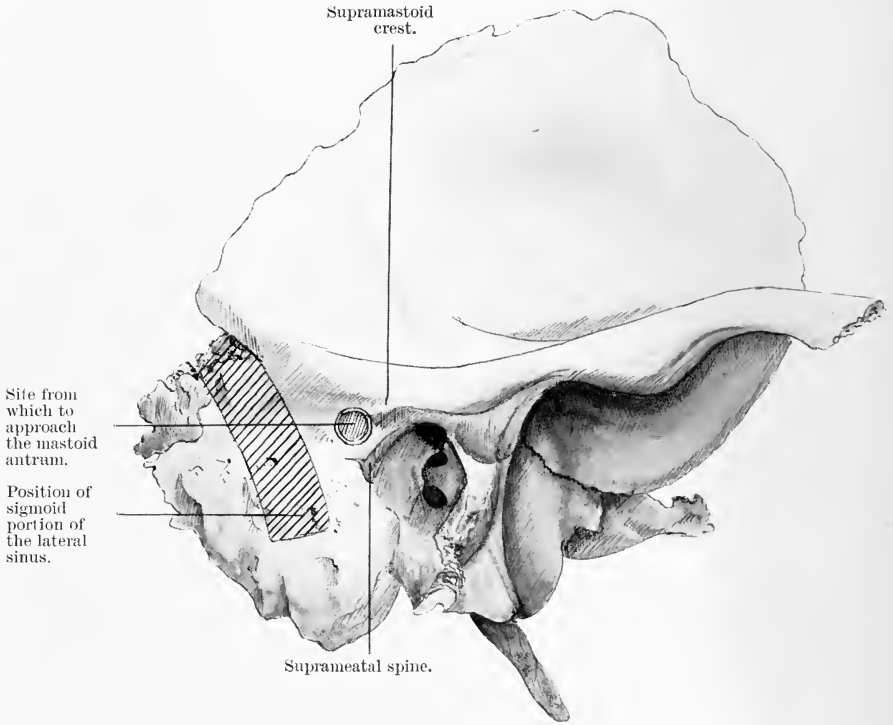


FIG. 56.—A right temporal bone to show the relations of the mastoid antrum.



It must also be remembered that purulent fluid may find its way into the mastoid antrum, through its foramen of communication with the tympanic cavity. Further, the mastoid cells when developed may also become infected through the antrum, and mastoiditis result.

The roof of the tympanic cavity is composed of very thin bone, covered above by fairly firmly attached dura mater. The interior of the cranium may be reached by pathogenic micro-organisms from the middle ear in various ways. Carious erosion of bone is probably the commonest method of extension, giving rise to an extra-dural collection of pus, and later on to meningitis. Another route is along lymphatic channels passing between the membranes and the tympanum; or along fibrous trabeculae, stretching between the mucous membrane of the tympanic cavity and the dura mater covering the petrous portion of the temporal bone. Possibly also the bacteria may pass through the petro-squamosal suture in quite young subjects. Also infection may track along the sheaths of the facial and auditory nerves. It is, however, very difficult to explain the exact method by which micro-organisms may cause inflammation within the substance of the cerebellum or the temporo-sphenoidal lobe, without any meningitis being present intervening between the site of the abscess and the source of infection.

The proximity of the sigmoid portion of the lateral sinus to the mastoid antrum and cells, explains the occurrence of septic thrombosis and subsequent pyæmia.

Occasionally pus from the middle ear may track downwards along the side of the Eustachian tube, and give rise to a lateral retro-pharyngeal abscess.

The course of the facial nerve from the internal auditory meatus through the aqueductus Fallopii to the stylo-mastoid foramen, lying in close relationship with the inner angle of the roof of the tympanum, and subsequently along the posterior wall of this cavity, indicates the reason why otitis media may be accompanied by facial paralysis.

Pus in the mastoid antrum is placed in a cavity which can

only be reached with safety by an intimate knowledge of the anatomy of the part. The supra-meatal triangle is perhaps the best guide in evacuating it. This little triangle is formed by the horizontal part of the posterior portion of the temporal ridge and the posterior curving margin of the auditory process, upon which there is frequently a little prominence of bone called the supra-meatal spine. Important structures lie in relationship with this small cavity: above is the dura mater, only separated by the thin tegmen antri, anteriorly is the facial nerve, internally the external semi-circular canal, and posteriorly the lateral sinus. The cranial cavity can be avoided by directing the chisel or drill horizontally inwards, the level of the floor of the cranial cavity being slightly above the temporal ridge. The facial nerve will be free from danger except when the chisel is directed forwards, particularly when the opening between the antrum and the tympanum is being enlarged. The external semi-circular canal will not be injured unless the inner wall of the antrum, which consists of a fairly thick layer of white compact bone, is perforated. The lateral sinus will be in danger, more particularly when the mastoid cells are being attacked, after the antrum itself has been exposed.

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