A TEXT-BOOK

OF THE

THEORY AND PRACTICE

OF

MEDICINE.

BY

AMERICAN TEACHERS.

EDITED BY

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PREFACE.

In the preparation of this work some of the teachers of Practical Medicine in leading schools of America have associated themselves, in order that each subject should be discussed by an expert of special authority. It may, then, be said to represent truly the best teaching of the science and art of Medicine at the present time in this country. As such it is offered to the medical profession and to the large body of our medical students, with the earnest hope that it will be found to meet their needs and to prove a safe guide. Especial care has been taken to provide a strong article on Hygiene, including the full discussion of disinfection, isolation, and other principles of preventive medicine. Bacteriology is treated at length by an eminent authority, while in connection with each of the infectious diseases full consideration is given to the nature and cause of the morbid process. The important subject of Intestinal Parasites is presented with unusual fulness. Here, as well as in connection with bacteriology, no pains have been spared to provide the best illustrations; so, too, wherever the text has seemed to require it, charts and diagrams have been freely used.

The object of the work is essentially a practical one. The subjects are treated in an authoritative manner. It has been impossible to give space for bibliographical lists or for many references. Any apparent absence of recognition of the views of other writers must be attributed to this cause.

The sections on Symptomatology, Diagnosis, and Treatment are especially full, and many formulæ are admitted.

Important assistance has been rendered in the preparation of this work by Dr. F. A. Packard, who has been associated with the Editor in the revision of MSS. and the correction of the proof-sheets.

WILLIAM PEPPER.

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HYGIENE.

By JOHN S. BILLINGS.

Instruction in hygiene for the medical student has, until recently, been considered as theoretically desirable, but practically unessential. To the student entering on his course of medical study the question may arise, "Why should a physician be compelled to learn how to prevent disease?" From the business point of view he is to support himself and his family by treating the sick: why, then, should he try to prevent the occurrence of sickness and thus lessen the chances for his employment? The answers to this are as follows:

First: From the business point of view the man who has studied modern hygiene is more apt to obtain and retain employment as a practitioner than one who has not. The laws of several States already require a knowledge of hygiene by those to whom license to practise medicine is given; the medical examining boards of the army, navy, and marine-hospital service lay special stress on the knowledge of hygiene possessed by the candidates who come before them; and the same may be said for the civil-service examinations for filling various offices in which medical knowledge is essential. There is, in fact, a rapidly-growing demand on the part of the public that physicians shall receive special instruction as to the causes of disease and the means of preventing or destroying those causes, as well as in the treatment of the diseases produced by them; and this demand exists not only with examining boards, but with the men and women who employ physicians in private life. The people who pay medical bills want to know the cause of their sickness; whether their houses are in good sanitary condition, and if not, why not, and what should be done about it; whether the water is safe to drink; and many other things for which they consult their physicians. Moreover, hygiene is not only the art of preserving health, but of improving it, and is a most important part of the therapeutics of many forms of disease.

Second: It is the duty of the physician to prevent disease whenever and wherever he can, without reference to any considerations as to whether his

doing so will be of any pecuniary benefit to himself or not, and to be fully informed as to the best methods of doing this. It is true that the health interests of the people among whom he lives and works are his own interests, because if they suffer he and his family must also suffer; but, without reference to this, or to his obligations as a husband or father, it is his special duty as an educated medical man to consider and advise upon sanitary problems for the benefit of those who have not this expert knowledge. It is the most direct and certain way in which he can serve God and his fellow-man.

Questions of public hygiene are becoming more and more prominent in the social and political world: all efforts to make the mass of the people more contented and comfortable are connected with health questions, and there is great need of scientifically educated men who will not be induced through ignorant enthusiasm to endorse the numerous quack reform schemes which are being continually proposed and thrust upon the public.

There has been, and still is, a vast amount of charlatanry, humbug, and advertising in so-called sanitary literature, especially in that part of it devoted to attempts to scare people into buying some patent contrivance or article of food or drink; and it is a part of the business of the physician to know when there is real danger and what is best to be done under the circumstances. Half-knowledge in these matters produces much unnecessary anxiety and fear.

Health is a means, not an end. In every-day life many men deliberately choose an occupation and a place of residence which they know involve a certain extra risk to health and life; in fact, the physician does this himself. In insuring health and life, as well as property, the question occasionally comes up, "What is the greatest amount of premium that it is worth while to pay for such insurance?" There is both an upper and a lower limit. Some cannot afford to insure at all.

Compulsory legislation for securing the health of a community must be framed with reference to economic consequences as well as to health.

The public hygiene of to-day dates from about fifty years ago, the time when a really useful system of vital statistics was established for England by Dr. William Farr. Prior to that time the causes and mode of preventing scurvy had been discovered, vaccination had been introduced, and much was known about personal hygiene; but very little was known about the health of communities or particular cities, or whether it was becoming better or worse.

The cholera epidemic of 1849 in England gave a powerful stimulus to investigation, which was made by the Health of Towns Commissions; but the Crimean War, with its positive demonstration of the effects of sanitation elaborated and insisted on by Dr. Parkes, was what finally convinced the governmental authorities of its importance and necessity. Next came the stage of careful study of the causes of various diseases by experiment and observation under the direction of Mr. John Simon as medical officer of the Privy Council, and of his successor, Dr. (now Sir) George Buchanan, until recently the

medical officer of the Local Government Board, and, finally, the work of Pasteur and Koch and their followers in bacteriology and its relations to contagious, infectious, and epidemic disease, which are now leading to practical results of the greatest importance.

Hygiene, as applied to man, includes the study of his relations to those surroundings which influence his health and longevity, and the practical application of the results of this study.

The end of each human life is fixed, in one sense of the word: there is a maximum which it cannot pass. But in another sense it is movable, and may be hastened or postponed by surrounding circumstances. Mere postponement of death is, however, not always desirable, for life itself may be a burden, and to be desirable it should be healthy and productive. A really sound, healthy man is, to a considerable extent, independent of surrounding circumstances: he does not have to regulate his diet minutely, to adjust his clothing to every change of weather, or to be continually guarding against possible causes of disturbance of function in order that he may be able to live and do his work. There was some truth in the old proverb that to live medically is to live miserably.

From the hygienic standpoint the causes of disease may be classified as follows: (A) causes acting from within, or predisposing causes, including (1) heredity, (2) individual peculiarities, connate or acquired after birth, but not inherited; (B) mental and emotional causes; (C) causes acting from without, immediate or exciting causes, including (1) physical, mechanical, and chemical causes, (2) micro-organisms.

Those causes which are more or less under our control, and which therefore may be modified, prevented, or avoided, are of special practical interest; but the so-called unavoidable causes must also be studied, partly because the disease or source of injury or nuisance against which we can to-day devise no protection may, in the rapid advance of science, be to-morrow within our control; and partly because we must know what and how much disease is due to unpreventable causes in order to understand the real scope and results of those influences which we can hope to modify.

For example, the effects of weather, including especially excessive cold, dampness, or heat, upon the health and life of the poorer classes in large cities are often very marked; hence these must be carefully considered in comparing the death-rates of two different years in order to determine whether a water-supply is becoming worse or better, or whether the system of sewage-disposal is producing satisfactory results.

Our knowledge of the causes of disease is derived from observation and experiment, the latter being in the main limited to those diseases which can be induced or produced in animals. Observation may be applied to individual cases or to communities. By the first we compare individual with individual and minutely; by the second we compare the vital phenomena of communities, but only on broad lines and with regard to circumstances easily noted.

To ascertain whether a block of houses or a town is unhealthy, whether it is getting worse or better, and what is the matter, we must obtain certain information with regard to the amount, character, and fatality of the diseases prevailing in the place; and the most important means of doing this is by examining the mortality statistics. Mortality does not mean the number of deaths, but the death-rate, or liability to death, as shown by comparing the number of deaths in a given time with the quantity of life in which they have occurred. It refers to a definite unit of time—viz. one year's life of one person—and the quantity of life is the sum of the time lived by each of the population expressed in years; thus, five men living one year, or ten men living six months each, or sixty persons living one month each, give five years of life. The number of years of life is usually stated as being the mean population for the year, and the annual death-rate is given for each thousand of mean population. It is computed by adding three ciphers to the number of deaths occurring during the year and dividing by the number of mean population for that year. Thus, if there were 350 deaths in a year in a city of 20,000 mean population, the death-rate would be 350,000 divided by 20,000, equal to 17.5 per 1000. If the time for which the mortality is calculated is less than a year, the result must be reduced to an annual ratio; thus, if the number of deaths in one week be given, this number must be multiplied by 52.177, the number of weeks in a year, and the product by 1000, and divided by the mean population, to give the annual death-rate. A more convenient, and sufficiently accurate, method is to divide the mean population by 52, and use the quotient throughout the year as the constant divisor for the weekly number of deaths multiplied by 1000. The mean population is ascertained either by an actual count or census taken in the middle of the year, or (and usually) by computation from the data given by two successive counts, from which is ascertained the annual rate of increase.

It is usually assumed that a population increases in geometrical progression, and the calculation is made by the use of logarithms according to the following formulæ, in which $r =$ annual ratio of increase, $p =$ population at last census, $p' =$ population at present census, $n =$ number of years between these two censuses, $x =$ mean population sought, and $n' =$ number of years between last census and the time for which the population is sought:

$$\log r = \frac{\log p - \log p'}{n}, \text{ and } \log x = \log p' + \log r \times n'.$$

The mean population for a period, as found by this geometrical-progression formula, is greater than the population actually living in the middle of the period, and less than the arithmetical mean of the population living at the beginning and end of the period; but if the period be only a year or two, the differences are small and either figure may be used.

The assumption that the population is increasing regularly in geometrical progression is rarely correct, and when the census is taken only once in ten

years it may lead to very erroneous results. The best method of correcting the result thus obtained is by comparing it with the result obtained by multiplying the number of inhabited houses, as shown by tax records or personal count, with the average number of inhabitants per house for that particular city, as shown by the previous census. Estimates based on the number of voters or of school-children or on city directories are almost invariably in excess of the true figures.

The number of deaths is ascertained from the public record in which each death is registered as it occurs. No system of registration of deaths is complete and accurate unless a permit for burial, granted by some public official, is required in each case of death. To obtain such a permit a certificate must be presented stating the name, age, sex, color, residence, etc. of the dead person and the cause of death. The certificate as to the cause of death must be signed by some one who is competent to tell whether the death was due to so-called natural causes or to crime. Hence it must be signed by an educated physician, and hence the matter of registration of deaths is one of great importance to medical men, for it is the foundation of state legislation to determine who are "educated physicians" and competent to sign such certificates.

The shorter the period for which a death-rate is given, and the smaller the population to which it refers, the less probable it is that it is to be relied on for purposes of comparison. This is due to the law of probable error as connected with the use of large or small numbers.

For rough calculations it may be assumed that the possible variation in the number of deaths is equal to the square root of that number. Thus, if 16 deaths occur in a year in a village of 1000 inhabitants, the possible error is 4, so that the death-rate might vary between 12 and 20 per 1000 without giving any certain indications of corresponding variation in the sanitary condition of the place; while in a city of 100,000 inhabitants, with 1600 deaths yearly, the possible variation in the number of deaths would be 40, so that the death-rate could only vary between 15.6 and 16.4 per 1000, without indicating the action of some special cause.

For localities for which the population cannot be determined, or for which the number of deaths occurring in a given time is unknown, death-rates cannot be calculated, and the essential foundation for a useful public health organization is therefore wanting.

Fair average annual death-rates are from 9 to 16 per 1000 in rural districts and small villages, from 14 to 18 per 1000 in towns of from 5000 to 20,000 inhabitants, from 17 to 20 per 1000 in cities of from 25,000 to 100,000 inhabitants, and from 18 to 21 per 1000 in cities of over 100,000 inhabitants. When death-rates below the lowest of the above rates are given, it is probable that the population has been over-estimated or that all the deaths have not been counted, or both; when they are higher than the highest of the above rates, it is probable that some special cause exists for the high mortality. In comparatively new and rapidly-growing cities, however, the large proportion of adults gives a lower general death-rate than those above stated.

Gross death-rates, obtained by comparing the total number of deaths with the total mean population, although useful, are not nearly so useful as the death-rates of particular classes of the population, and especially death-rates for different groups of ages with distinction of sex. This is due to the fact that the natural tendency to death varies greatly at different ages; thus, the average annual death-rate of children under five years of age is from 50 to 100 per 1000; of persons between five and twenty years of age, from 2 to 7; between twenty and forty, from 6 to 11; between forty and sixty, from 12 to 25; and over sixty, from 55 to 95 per 1000. Hence the proportion of young children and of old persons present in a given population has a great influence on its death-rate, and, unless these proportions are nearly the same, the comparison of the gross death-rates of two different populations may give very erroneous results. This also applies to the death-rates of different occupations. The death-rate of judges and major-generals is greater than that of students or lieutenants, because of the average difference in ages.

To obtain satisfactory and reliable mortality statistics we must know not only the population, but the population of each sex at different groups of ages, and the number of deaths in a given time, with corresponding distinctions of sex and age.

For a large part of the United States the data necessary for calculating death-rates cannot be obtained: only the New England States, New York, and New Jersey have a system of registration of deaths which gives fairly complete results, and in some of these it has been in operation but a short time. Even where there is a fairly good registration of deaths, its results are often partially or entirely useless for calculating death-rates because of the want of reliable information as to the number of population at different ages.

The best means of eliminating the influences of sex and age on death-rates is by the preparation of a life table, from which can be determined the expectation of life at each age in each sex, such as that shown in the following table:

Table showing Expectation of Life.

		Ages.						
		0	5	10	15	25	40	60
American Life Ins. Co. . .	Males . . .	. .	. .	49.9	46.6	39.5	28.5	14.5
Massachusetts, 1883–87 . .	Males . . .	39.7	52.7	49.3	45.1	38.2	28.2	15.3
Boston, 1883–87	Persons . .	33.3	47.2	47.5	44.7	37.1	28.5	16.7
Providence, 1883–87 . . .	Persons . .	40.8	51.1	48.3	44.2	37.5	27.7	15.0
New York, 1879–81	Males . . .	33.3	48.0	44.9	40.6	33.2	23.9	13.0
Baltimore, 1880, colored . .	Males . . .	21.0	41.8	40.0	36.8	31.0	21.7	11.3
Society of Friends, Phila. .	Persons . .	43.7	51.8	48.8	44.6	37.5	28.5	15.6
Society of Friends, England	Males . . .	45.3	53.8	50.5	46.4	39.9	29.2	14.4
Benedictine Monks, Paris .	. . . . .	. .	. .	. .	. .	34.6	24.0	11.7
English Life Insurance . .	. . . . .	. .	50.6	45.6	40.6	34.0	24.5	12.6
Dublin, 1841	Males . . .	23.8	38.5	37.2	33.4	27.0	19.8	11.0
Berlin	Males . . .	17.2	44.9	43.1	39.5	32.0	22.0	10.0

From this table it will be seen that at the age of twenty-five the mean expectation of life of that class of American males who insure their lives is

thirty-nine and a half years, while for colored males in Baltimore it is only thirty-one years.

The expectation of life is the mean after-lifetime; the probable duration of life is the age at which the population at a given age will be reduced one-half. If of 100 children born, 30 live just one year, 20 just five years, 30 live 40 years, and 20 live 60 years, then the probable duration of life of any one of these children at birth is five years, because at the end of that time one-half of them will be dead, but the expectation of life of any one of these children is 25.3 years, because the 100 altogether live 25,300 years of life.

If we have the results of a registration of deaths, but no information about the population, the best we can do is to compare the number of deaths under one or under five years of age with the whole number of deaths, or the number of deaths from one particular cause with the number of deaths from all causes; but the results are unsatisfactory and may be very misleading. Suppose, for instance, that in a city 1000 deaths occur in a year, and that 250 of them are due to consumption, and that in another city there are 2000 deaths in a year, of which 500 deaths are due to consumption, then the proportion of the number of deaths from phthisis to total number of deaths would be the same in the two cities. But if the two cities were of the same size, the liability to death from phthisis would really be twice as great in the second city as it was in the first.

In investigating the healthfulness of a place it would be very desirable to know not only the number of deaths, but the amount and kind of sickness which has prevailed. The usual estimate is, that for every case of death there is an average of two years' sickness in a community, so that if the annual death-rate is 18 per 1000, the average number constantly sick is 36. It is impossible, however, to obtain complete and reliable information on this point from any city, since it is, as a rule, only collected for the army and navy and for certain societies. For certain contagious diseases, however, physicians in cities are often required to report all cases which come under their observation, these diseases being Asiatic cholera, yellow fever, typhus fever, small-pox, scarlatina, and diphtheria, and sometimes typhoid fever and measles. The results are useful as far as they go, but the returns are generally incomplete.

Predisposing Causes of Disease.

The most important predisposing causes of disease are those connected with the structure of the body, being individual peculiarities which may be inherited, or may be congenital but not inherited, or may be acquired after birth. In most of the so-called hereditary diseases that which is transmitted from parent to child is not the disease itself nor its direct specific cause, but some peculiarity of structure of tissues or organs which, in the course of development, either makes the person peculiarly susceptible to causes of disease acting from without, or produces disorder itself by excess or defect of structure or function of some particular part. Syphilis, small-pox, and a few other diseases may be directly transmitted by transference of the specific cause to the child *in utero*. In scrof-

ula, consumption, and other forms of tuberculosis the specific germ is very rarely if ever transmitted, inheritance giving only a special susceptibility to its action. In gout, rheumatism, or insanity due to heredity there is abnormal structure of some particular part which ultimately leads to disease. Heredity transmits immunity against certain forms of disease. Its effects are seen not only in certain families, but on the large scale in certain races; thus, the number of cases of cancer in the white residents of the Southern States is more than twice as great as it is in the negroes of the same region in proportion to the number of each class, while, on the other hand, the tendency to tuberculosis is decidedly greater in the negro than in the white. Jews are specially liable to diabetes and to various forms of degeneration in the spinal cord in advancing years, but are somewhat less liable to cancer and consumption than other whites. In many cases it is very difficult to distinguish the effects of inherited bodily peculiarities from those due to modes of life peculiar to certain families or races, but the general practitioner soon learns to expect certain special symptoms in the members of a particular family, as, for instance, delirium in the course of fever. In the examination of applicants for life insurance the family tendency to death from certain forms of disease, such as consumption, apoplexy, or insanity, is carefully inquired into, and is held to be of great practical importance. Persons having the same hereditary tendency to disease should not intermarry, for the tendency will be markedly increased in their offspring. Persons affected with hereditary or well-marked constitutional syphilis should never marry. Every one has certain individual peculiarities which may or may not be manifestly inherited, and it is in the detection of these peculiarities, and in the estimation of their relations to each other, to disease, and to results of certain remedies, that the skill of the physician largely consists. A normally well-developed, healthy young man has more lung surface, more kidney, or liver, or lymphatic gland, than is actually necessary to preserve life under ordinary circumstances, and therefore if a part of one of these organs becomes, through injury or disease, unable to do its proper work, or if an extra amount of work is thrown on the organ for a short time, it does not necessarily disable him. But if the amount of absorbing, secreting, or excreting surface in any department of his economy is barely sufficient to supply the needs of the organism, either because of originally insufficient development or because of loss of a portion of it through want of exercise, disease, or injury, then disturbances or excessive demands, which in a healthy man would produce no noticeable effects, may give rise to the most serious results, and hence require special care in diet, clothing, and mode of life to maintain comfort, if not existence.

Mental Causes of Disease.

Certain forms of nervous disease may be produced by expectant attention or suggestion, or, which is much the same, by involuntary imitation of the symptoms presented by a person affected with it. This occurs in various forms of epidemic chorea and hysteria, especially those occurring under religious excitement, in some cases of stammering, etc. Expectant attention directed to

some particular part or organ of the body, especially if accompanied by strong belief in or fear of some result, may produce marked changes in function, excess or deficiency of blood-supply, abnormal reflexes, and even permanent change in structure. Excessive worry or anxiety is often a cause of disease, either by producing loss of sleep or by interfering with the nervous mechanism regulating the appetites and the action of the digestive and secreting organs. Simple mental exercise in the form of study or writing, even when carried to excess, rarely produces marked ill effects; it is only when it is accompanied by anxiety that its results are likely to become serious. The disorders produced or aggravated in children by school attendance are more usually due to impure air, to defective or improper lighting, and to badly-shaped seats and desks than to excess of study; nevertheless, under the stimulus of prizes, final pass-examinations, etc. the health of some sensitive and ambitious children may be seriously impaired, and such means of inducing them to work should be used with great caution. As a rule, in our public schools too much is demanded of the children, and the most of them have to do too much studying at home in order to keep good standing in their classes. In adult life the effects of mental strain are often complicated with, or marked by, those of such stimulants as coffee, alcohol, opium, etc.; and this should be borne in mind in the investigation of such cases.

Micro-organisms.

Of the diseases due to more or less preventable causes and liable to occur in epidemic form—and which are therefore of special interest from the hygienic point of view—the most important are those which are known or supposed to be caused by very minute living organisms. These micro-organisms include the minute animal forms, or the microzoa; the minute vegetable forms, or the microphytes; and also minute living particles of protoplasm which may be doubtfully classed as distinct organisms.

With regard to the microzoa, or those doubtful forms known as the protozoa, considered as causes of disease, our knowledge is as yet scanty. One form, the *Amœba dysenteriæ*, is the cause of a peculiar and dangerous form of dysentery; some of the sporozoa appear to be connected with certain skin diseases, and it is probable that the cause of malaria belongs to this class. Here also, although belonging to higher orders of the animal kingdom, may be mentioned such parasites as the *Anchylostomum duodenale*, the *Trichina spiralis*, *Bilharzia hæmatobia*, and worms of various kinds, including hydatids.

The microphytes of most known importance in the causation of disease are certain kinds of the schizomycetes, commonly known as bacteria, and especially those known as cocci or micrococci, being minute spherical forms, and those named bacilli, which are rod-shaped or oval. There are many kinds of these bacteria, differing slightly in shape and size, and greatly in the appearances they present when growing in masses in or upon various media, such as beef-broth, gelatin, agar, etc. They differ also in the ease with which they may be stained with different substances, in the tenacity with which they retain these

stains, in the decompositions and decomposition-products which they produce, and in the effects which follow their entrance into the living human body. These differences are constant and each kind breeds true, so that we may say that there are many distinct species. The great majority of the species are not only harmless so far as man is concerned, but beneficial. They feed upon dead, insoluble organic matters, the products of higher animal and vegetable life, and convert them into soluble forms of simpler composition which may be utilized by living plants. They are present in the lower layers of air over the land except in the Polar regions, in the upper layers of soil, and in almost all water, and there is very little dead organic matter which escapes them. Almost all forms of putrefaction and fermentation are produced by them. They multiply by simple division with great rapidity under favorable circumstances, some of them dividing once every half hour, so that a single cell may produce ten millions or more in twenty-four hours. They are about one-twenty-five-thousandth of an inch in diameter, and from one-fifteen-thousandth to one-five-thousandth of an inch in length. It would require about twelve million micrococci placed side by side to cover an ordinary pin's head. All of them require the presence of moisture, nitrogen compounds, usually in the form of dead organic matter, and of a suitable temperature to enable them to grow and develop. A few of them produce disease in man, either directly or through their products. The proof that a particular form of disease is due to a particular micro-organism is as follows:

1. The disease must be one that can be identified—that is, that presents a tolerably distinct series of symptoms or of pathological results—so that it can be distinguished from other diseases either in the living or the dead subject, or in both.

2. In all cases of the disease the specific form of the micro-organism must be found in the fluids or tissues of the body.

3. This micro-organism must be separated from the fluids or tissues of the body, and from other micro-organisms, and cultivated in suitable media outside the animal body until a series of pure cultures is thus obtained.

4. The pure culture thus obtained must produce the specific disease in a healthy animal when introduced into its body by inoculation or through the alimentary canal or air-passages.

5. In the animal in which the disease has thus been produced the same micro-organism must be found.

Since many different micro-organisms may be found at different times in the human body, including all the varieties found in water and air, the mere occasional coincidence of the presence of some one form in a particular disease is not sufficient to prove a causal connection. It must also be remembered that the specific micro-organisms may be present in or on the skin or mucous membranes of the body without producing disease, for in many cases they require special conditions of injury or lowered vitality of the tissues with which they come in contact to enable them to develop, and in some persons they produce no effects, as will be explained hereafter in speaking of immunity. As inoculations

of disease-producing organisms, or of those supposed to be such, cannot, as a rule, be tried on man, the chain of positive experimental proof can usually only be completed for those diseases which can be produced in other animals; nevertheless, a high degree of probability may be obtained when a particular form of micro-organism is always found present in a person affected with a disease having well-marked characteristics, and is seldom or never found in the living body when such disease is not, or has not recently been, present.

No satisfactory classification of the bacteria has yet been made. For the purposes of this article it is sufficient to say that the spherical forms, or micrococci, include Streptococcus, in which the individual cocci after subdivision remain united together in little chains or strings; Staphylococcus, in which they are clustered together like a bunch of grapes; and Micrococcus, in which the granules are usually seen singly or in pairs or in short chains. When they are usually in pairs they are called diplococci. Some authors use the term micrococcus as the generic name of all forms; thus, the *Micrococcus pyogenes aureus* is the same as the *Staphylococcus pyogenes aureus*. Of the rod-shaped forms, some authors refer to a separate genus, Bacterium, all those in which spore-formation is absent or unknown, but most writers include them all under the term Bacillus; thus the *Bacterium prodigiosum* is the same as the *Bacillus prodigiosus*.

The student should bear in mind that there is no sharp dividing-line between the coccus forms and the rod-shaped forms; that very short rods with rounded ends or shaped like an ellipse are called micrococcus by one observer and bacillus or bacterium by another; and that the same organisms in different stages of growth and development may show single cocci, chains, and rods. The spirally-twisted forms of bacteria are classed as Spirillum, but the spirillum of Asiatic cholera is commonly called the cholera bacillus. The following is a list of the principal diseases of man which are due to bacteria, with the usual names of the species of bacteria which cause them:

1. Inflammation of tissues, producing suppuration and its consequences, as in abscesses, boils, pyæmia, osteomyelitis, puerperal fever, etc. These are produced by the *Staphylococcus pyogenes aureus*, the *Staphylococcus pyogenes citreus*, the *Staphylococcus pyogenes albus*, the *Streptococcus pyogenes*, and by a few other forms, the whole forming a group known as the pyogenic micrococci. Two or more kinds of these are often found together in pus. The specific cause of erysipelas also belongs to this group.
2. Gonorrhœa, produced by the *Merismopedia gonorrhœæ* or gonococcus.
3. Anthrax, caused by the *Bacillus anthracis*.
4. Tuberculosis, caused by the *Bacillus tuberculosis*.
5. Leprosy, caused by the *Bacillus leprœ*.
6. Glanders, caused by the *Bacillus mallei*.
7. Typhoid fever, caused by the *Bacillus typhosus*.
8. Diphtheria, caused by the *Bacillus diphtheriæ*.
9. Tetanus, caused by the bacillus of tetanus.
10. Specific croupous pneumonia, caused by the *Diplococcus pneumoniæ*.

11. Asiatic cholera, caused by the cholera bacillus.

12. Relapsing fever, caused by the *Spirochæte Obermeierii.*

In addition to these there are various forms of dysentery, of so-called cholera morbus, cholera infantum and summer diarrhœa of infants, and of chronic forms of diarrhœa which are probably produced by one or more species of bacteria, as are also various forms of endemic tropical ulcer, such as the Delhi boil, and of skin disease, such as rhinoscleroma. There are also a number that produce specific diseases in certain animals, but which have thus far been very rarely or never observed in man, such as the bacillus of hog cholera, of swine erysipelas, of malignant œdema, of black leg of cattle, the vibrio of Metschnikoff, etc. It is also probable that small-pox, measles, scarlatina, yellow fever, typhus fever, influenza, Oriental plague, and syphilis are due to specific micro-organisms, but this is not yet demonstrated. Some of the specific pathogenic micro-organisms, such as those of diphtheria, typhoid fever, and cholera, may grow and multiply in dead organic matter of animal origin at the ordinary temperatures of the air, and hence collections of such organic matter may become dangerous centres of infection. Others, like those of tuberculosis, of glanders, and probably also of small-pox, measles, scarlatina, etc., do not grow and multiply outside the living animal body under ordinary circumstances, so far as we now know, being what are termed obligatory parasites. Many different kinds of bacteria are found on the surface of the human body, in the mouth and air-passages, and in the alimentary canal. A few exist in the hair-follicles beneath the epidermis, and among these are some of those which cause suppuration under favorable circumstances. They gain admission to the body through the air and in articles of food and drink. The bacteria of the air come from the upper layers of the soil, from hay, straw, clothing; in short, from whatever produces dust, to the particles of which they are usually found adherent. They are not detached into the air by simple evaporation from moist surfaces. The air expired in respiration does not contain them unless there is coughing or sneezing, in which case they may be in the spray. An open tubful of diphtheritic membranes or of typhoid stools will give off no specific germs to the air so long as its contents are kept thoroughly moist and no bubbles arise to break into spray and throw particles of liquid into the air. Sewer air contains fewer bacteria than outside air, and those found in sewers do not, as a rule, come from the sewage, but are brought in in air-currents from the outside. Whatever produces dust increases the number of bacteria in the air, as, for example, dry sweeping, bed-making, stamping of feet in assembly halls, etc. The fact that disease-producing organisms cannot escape into the air from the surface of still fluids or from thoroughly moist surfaces is one of great practical importance in hygiene, and will be referred to hereafter in speaking of disinfection, of ventilation, and of house-drainage. The number of bacteria in the upper layer of the soil is very great, the most important disease-producing forms found there being the bacilli of malignant œdema and of tetanus, and in cities those which produce summer diarrhœa. The soil-organisms which are of the greatest importance in decomposing organic matters are those which produce nitrites and nitrates, and are

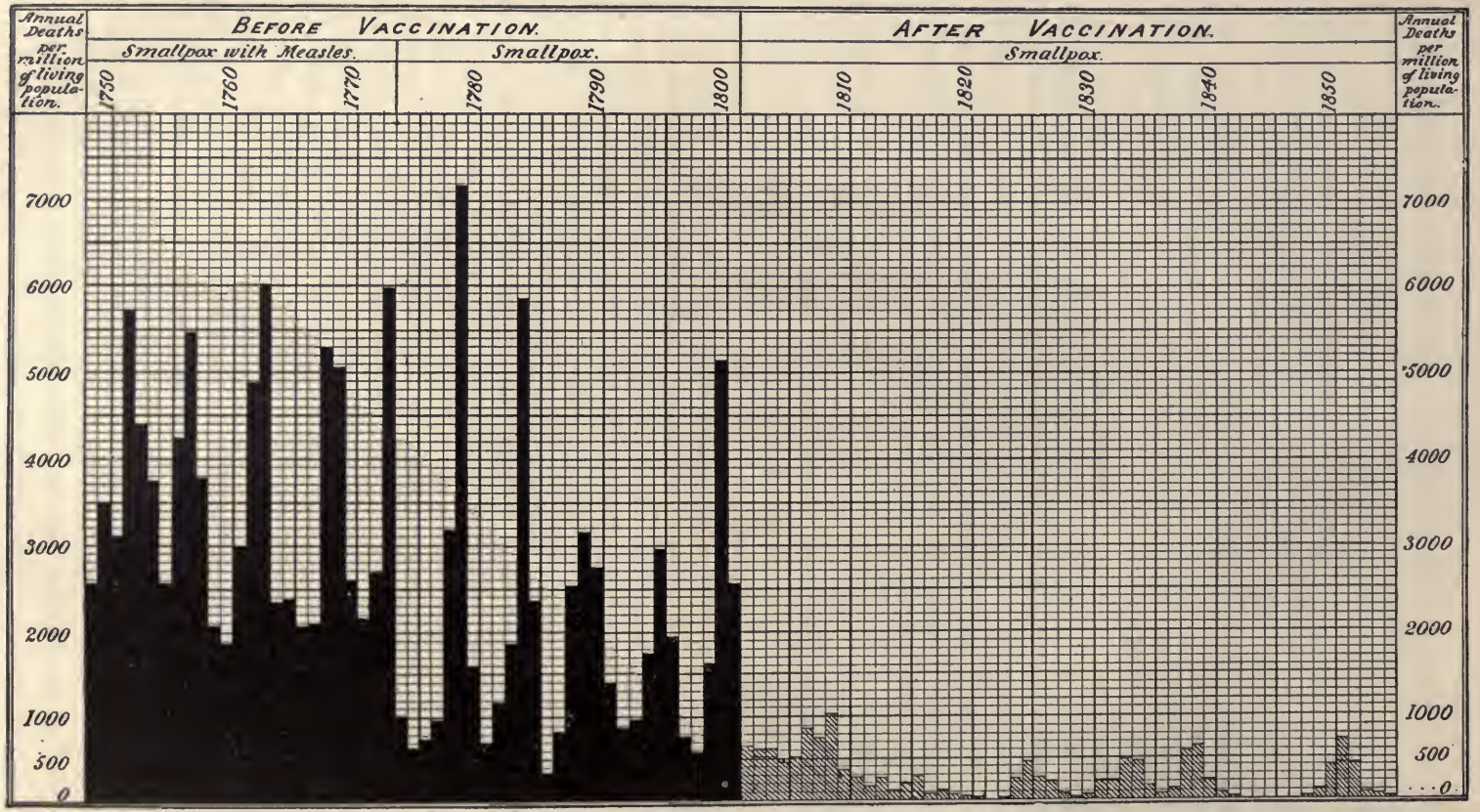
known as the nitrifying bacilli, and the action of these in the purification of waters contaminated by organic matters is of great practical interest. The chemical compounds produced or excreted by many bacteria check the growth, or even destroy the vitality, of other forms, and especially of the disease-producing forms; while, on the other hand, the power of certain bacteria to produce disease is greatly increased by the presence of other forms which by themselves appear to have no harmful influence. The means at our disposal for preventing the diseases caused by micro-organisms consist of the production of immunity in individuals, of disinfection, and of isolation.

Immunity.

A person is said to possess immunity as regards a certain disease when he is but slightly or not at all affected by the causes of that disease when brought into contact with them. Immunity may be natural or artificial, partial or complete, relative or absolute. Natural immunity may be hereditary; as, for example, in the comparative insusceptibility of the negro to malarial and yellow fevers. Artificial or acquired immunity may be produced by the action of the disease itself. Thus, a person who has recovered from an attack of small-pox, scarlet fever, measles, whooping cough, typhoid fever, or yellow fever is more or less immune against a second attack of that disease, and one who has been properly vaccinated is immune against small-pox. Precisely how this immunity is produced we do not know, and it probably differs somewhat in different diseases, but in general it may be said to be due to the presence of certain albuminoid substances which have the power of killing or weakening pathogenic micro-organisms or of neutralizing their toxic products; and this presence is probably connected with peculiarities in the mode of life and chemical products of large masses of cells in the body. In some cases these masses may perhaps be definitely localized, forming a sort of new organ with specific powers, as, for example, in the locality in which vaccination has been performed. Cases are known in which amputation of the vaccinated limb has seemed to destroy the immunity against small-pox.

Immunity is rarely absolute and complete; second and even third attacks of all the diseases named above may occur, but from experiments on animals it is probable that it requires the concurrent action of a much larger number of the specific organisms to produce a second attack than it did for the first. The natural fluids and living tissues of the body, when healthy, have the power of destroying a certain limited number of micro-organisms; thus, almost every one at some time or other inhales the bacilli of tubercle, yet in only a certain number do they develop and multiply. At present, vaccination for small-pox is the only operation for producing immunity in man which is advised by physicians, but it is probable that this method of prophylaxis may be extended in the future. It is important to bear in mind the effects of immunity in investigating the conditions of outbreaks of disease in certain localities; for instance, yellow fever is not likely to become epidemic in certain old parts of cities specially liable to be affected by it, simply because almost every

FIG. 1.



Showing Small-pox Death-rates for the Kingdom of Sweden for the one hundred and seven years 1749–1855. (From the first *Report of the Royal Commission on Vaccination, 1889.*)

inhabitant of those quarters has had the disease; and in like manner after an epidemic of typhoid has swept through a village a large number of the survivors will be immune against that disease. Immunity thus produced is the cause of the appearance of certain contagious diseases in epidemics at tolerably regular intervals, as small-pox before vaccination was introduced used to be epidemic at intervals of five or six years, and scarlet fever now appears in somewhat similar waves. It requires about that length of time for a new generation of children who are epinosic—that is, susceptible to the specific germ—to be developed in order to furnish sufficient material for an epidemic. This is shown diagrammatically in Figs. 1 and 2.

FIG. 2.

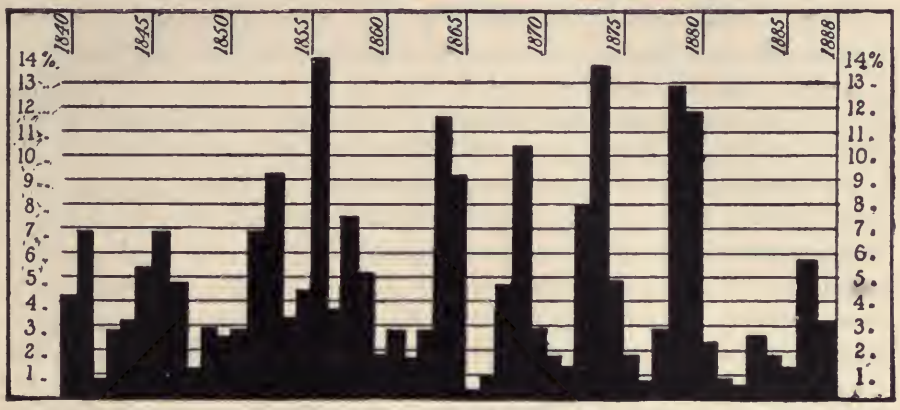


Diagram showing percentage of deaths from Scarlet Fever in total deaths in Providence, R. I., for forty-nine years.

DISINFECTION.

Sterilization of a substance, or of a flask or other vessel, consists in the killing of all living organisms contained in it. It may be partial or total, relative or absolute, permanent or temporary. In experiments in cultivating bacteria the culture media and the vessels which contain them are usually totally and absolutely sterilized, but it is not desired that this sterilization shall be permanent under all conditions. It is to be temporary, and to endure only until we inoculate the media with some particular form of organism which we wish to grow there. Total permanent sterilization is rarely used unless we wish to entirely decompose and destroy the substance, as by fire. The application of sterilization to the destruction of those micro-organisms which cause specific infectious or contagious diseases is called disinfection, and the agents used for this purpose are called disinfectants or germicides. In popular language the word "disinfection" is also applied to processes intended to destroy infection if it be present. Thus we speak of the disinfection of a privy-vault, of sewage, etc., and there is no special objection to this use of the word, for in the majority of cases in which we use disinfectants we do not know positively that specific disease-producing germs are present, but merely think it probable that they

may be there. Disinfection may often be obtained without complete sterilization. This is due to the fact that most micro-organisms which are rapidly growing and multiplying in moist media, including most of the micrococci and the bacilli of cholera, typhoid, and dysentery, are much more easy to kill than are the spores of certain forms, especially of the hay bacillus and of the bacillus of anthrax. In comparing the effects and efficiency of various disinfectants time is a most important element, for a degree of heat or a chemical solution which will have no effect in five minutes and very little in half an hour may effectually destroy the vitality of the organisms in twenty-four hours.

Antiseptics are substances which prevent the growth and development of micro-organisms, and especially of those which cause fermentation or putrefaction or which produce suppuration. They may or may not be disinfectants or germicides. A universal germicide—that is, an agent which effects complete sterilization—is necessarily an antiseptic for the time being, but if meat broth be heated until it is sterilized and is then left in an open vessel, it is not thereby made antiseptic. An antiseptic is something which remains, and prevents the development not only of the bacteria present, but of those which may be added afterward, although it does not necessarily kill them. A deodorant is an agent which destroys or mitigates foul and unpleasant odors, but many of these agents have little or no disinfectant powers. There is no definite relation between foul odors and specific disease-producing organisms: either may be present without the other, and it is improper to speak of the process of masking or destroying the odor produced by a uterine cancer in the last stages as being a process of disinfection.

The principal agents now used for disinfection are heat, carbolic acid, bichloride of mercury, chloride of lime, quicklime, alcohol, and sulphurous acid. These are the cheapest, the most generally applicable, and the least likely to damage clothing, furniture, etc. The strong mineral acids, chloride of zinc, chlorine, hypochlorite of soda, and certain coal-tar products are also good disinfectants, but are only used in special cases. What may be called the natural process of disinfection is accomplished in the course of time by light, fresh air, and the action of the common bacteria. All of these are of the greatest practical importance in preventing the undue increase of pathogenic organisms and in aiding in their destruction in water, soil, and air, and should be constantly employed as auxiliaries; but for prompt and certain disinfection we must resort to other agents. The practical utility of these depends not only on their germicidal powers, but on the ease with which they can be applied, their cost, and the danger of injury to persons or property from their use.

The most important of these special disinfecting agents is heat, and the simplest method of applying it in many cases is to burn the infected article. Sometimes it is best to do this for the moral effect, to reassure the community, as, for example, to burn up an old small-pox hospital instead of tearing it down. The cremation of garbage, of dead animals, or of human bodies is a disinfecting process, though not usually performed for that purpose. Dry heat—that is, a sort of baking in a closed chamber or oven—has been used to

a considerable extent in many places for the disinfection of clothing, bedding, and small movable articles, but is now being abandoned except for laboratory purposes and for the sterilization of some surgical instruments. It penetrates very slowly into non-conducting articles, such as bedding, mattresses, and pillows; it is very difficult to regulate so as to secure a proper temperature in all parts of the oven; it fixes stains of blood, excreta, etc. in clothing and bedding so that they cannot be washed out; and it is very liable to injure the texture of woven stuffs, scorching woollen at about 250° F. Exposure to hot air at 220° F. for one hour will kill micrococci and bacilli, but not spores, which, however, may be killed by five hours' exposure to this temperature. One hour's exposure to a dry heat of 245° F. will kill the spores.

Heat combined with moisture destroys the life of micro-organisms at lower temperatures with the same time of exposure, or with much less time of exposure at the same temperatures, than dry heat, and is therefore less liable to injure the articles subjected to it. The simplest form of application is by boiling in water, and this is the best method of disinfecting all articles of clothing, bedding, towels, etc. which can be washed without injury. The experience of large public laundries, and especially of laundries connected with hospitals for infectious diseases, such as that in Glasgow, shows that all germs of infectious disease are thus destroyed, and that clothing of small-pox, typhus, and other patients may be mingled and go through the boiling-vats without risk to the subsequent wearers. It should be borne in mind that infected clothing and bedding is chiefly dangerous when it is dry. When it is soaked with water it does not give off germs to the air. It would often be best, in collecting clothing and bedding supposed to be infected, to place the articles at once in a cask or tub or other vessel containing cold water, partly to soak out any stains and partly to prevent the giving off of any dangerous dust. It is usually advised that the clothing of the sick, and especially of those in hospitals for infectious diseases, should be washed in a place entirely separated from that in which other clothing, such as that of nurses and attendants, is washed; but while this may be desirable as a matter of sentiment and imagination, it is not necessary, for half an hour's boiling makes all the stuffs harmless. Boiling is also an effectual means of destroying choleraic, typhoid, or dysentery germs in water which must be used for drinking, and it is a good method of sterilizing surgical instruments that are properly constructed with reference to this mode of treatment. Moist heat may also be applied by means of steam in boxes or chambers specially constructed for the purpose. To obtain satisfactory results, all air should be driven out, and the steam should be moist or saturated at a temperature of about 220° F. If the pressure is less than that pertaining to the temperatures as given by Regnault's law, the steam is superheated, and is little better than hot air for disinfecting purposes, while if the pressure is greater than that pertaining to the temperature, there is admixture of air, and the clothing, etc. are not properly penetrated. The pressure should only be great enough to secure that there shall be no condensation of moisture in the chamber. Many forms of steam disinfecting apparatus, both

fixed and movable and of various sizes, are now constructed by English, French, and German manufacturers. In judging of the merits of any particular form or in devising a new one the following points should be borne in mind: A constant and uniform temperature should be secured throughout the chamber and in the interior of the mattresses, rolls of bedding, etc. to be disinfected. To ensure this, a metallic thermometer with electric connections with a small gong should be placed free in the chamber, so arranged that when the temperature reaches 221° F. (105° C.) the gong will sound; and a similar thermometer, similarly connected and adjusted, should be placed in the centre of the most bulky article to be disinfected, such as a mattress or pillow. Mercurial thermometers are not as serviceable for this purpose as those made of a coiled strip of metal which make the electric connection by expansion and contact. The steam should flow through the chamber freely at first, until the greater part of the air is expelled; and this seems to be best effected in those forms of apparatus in which the steam is admitted to the top of the chamber, the outlet being at the bottom and so controlled by a valve as to secure the amount of pressure required. After the greater part of the air of the chamber has been expelled, the valve may be closed and the pressure and temperature allowed to rise until the gong indicates that the temperature of 105° C. has been reached in the chamber. The valve should then be opened again, and the pressure be made to vary, for the purpose of expelling the air from the interior of the mattresses, etc., until the enclosed thermometer rises to 105° C., as indicated by its gong. When this has been secured the valve should be so set as to maintain this temperature and pressure for about forty minutes, which will be sufficient to secure complete disinfection. The chamber itself is usually made of boiler iron with double walls, and, if it is a fixture in a central disinfecting establishment, it has a door at each end, and is set in a partition wall in such a way that the articles to be disinfected are introduced at one door and removed from the other, so that they do not come out into a room which has contained infected articles. The central disinfecting stations of Berlin and of Paris may be taken as types of the arrangement which is desirable. Where the apparatus is in constant daily use it may have its own boiler; where it is only used occasionally it will be better to obtain the steam from some plant which is in daily use, for the articles to be disinfected should not be allowed to accumulate, but should be promptly treated. It must be borne in mind in operating a public disinfecting station that the results will be judged by housekeepers with reference to the effects upon color, size, and texture of the articles submitted to the process. As Dr. Russell remarks, it is of no use to quote scientific authorities to the woman who finds that her blankets come back a shade yellower than they were when she sent them to the station. "Dynamometric experiments on the breaking-point of hair-fibres will be of little use against a claim for damages which is supported by the fact that the upholsterer has charged so much for the wool or hair necessary to make up the original weight of the mattress or pillow. The result is, that there is constant friction in carrying out disinfection on a large scale. There is a temptation to the

officials to scamp the work to avoid censure, and there are constant private efforts to escape interference by concealment or appeal to domestic processes."

The chemical disinfectants may be divided into those which are used in gaseous and those which are used in liquid forms. Of the gaseous disinfectants, sulphurous acid is the one chiefly employed, and next to this comes chlorine. Nitrous-acid fumes and hydrochloric-acid fumes have also considerable disinfecting powers, but are very rarely used. Attempts to disinfect the air surrounding a patient are useless. It may to a certain extent be deodorized or be given some special odor, as by the use of saucers containing chloride of lime placed about the room, or of strips of cloth soaked in carbolic acid, or by burning pastilles of various kinds; but all these things, so far as disinfection is concerned, are what Simon calls "a futile ceremony of vague chemical libations or powderings, savoring rather of superstitious observance than of science." Theoretically, it is possible to disinfect air by passing it over highly-heated surfaces or by drawing it through cotton filters, but these processes are only used on a small scale in the laboratory.

We cannot conveniently apply heat to the walls, floors, and surfaces of rooms, to certain kinds of furniture, etc., and for this purpose it has been usual to employ the fumes of burning sulphur—a very old process, for Homer tells in the *Odyssey* that Ulysses purified his house in this way. The advantages of sulphurous acid are that it is extremely diffusible, so that it will readily penetrate into the interior of a mattress or pillow or the upholstery of a chair; it has little or no injurious effects on the ordinary furniture of apartments; it is easy to use and is cheap. It will not destroy spores, and is therefore useless in disinfecting for anthrax and tuberculosis, and it escapes so rapidly from ordinary rooms that it is very difficult to keep a sufficient strength of it in the air for a sufficient length of time to produce certain germicidal results. It should not be relied upon as the exclusive means of disinfecting an apartment, but may be applied after the application of liquid and cleansing disinfectants to all surfaces as far as possible. In most cases it is applied by burning sulphur in an iron vessel placed on sand in the room which it is desired to disinfect. The quantity necessary is about sixty grammes per cubic metre, and it is difficult to secure complete combustion of this amount if the room be sufficiently air-tight to secure useful results. It is used in France and in New York, but has been abandoned in Germany. Its efficacy is increased by the presence of moisture in the air. For cleansing walls, woodwork, floors, etc. in a room presumed to be infected rubbing with bread-crumb, as recommended by Esmarch, is a good method. The crumbs should be kept moist, carefully collected, and promptly burned. Rubbing with old cloths wet with an acid solution of corrosive sublimate is also a good method. All mere rubbing and scrubbing methods, however, can effect only partial disinfection in the habitations of the poor, because of the number of fissures and cracks in the walls, ceilings, and floors, the interior of which cannot be reached in this way.

Of liquid disinfectants one of the most useful is a solution of corrosive sublimate acidified with hydrochloric or tartaric acid. That used in the Paris dis-

infection service is composed of corrosive sublimate 2 grammes, tartaric acid 24 grammes, water 1 litre, with 5 drops of a 5 per cent. solution of carminate of indigo. That most used in England is corrosive sublimate ½ ounce, hydrochloric acid 1 ounce, water 3 gallons, tinted with 5 grains of aniline blue. That recommended by the Committee on Disinfectants of the American Public Health Association consists of 2 drachms each of corrosive sublimate and permanganate of potash to the gallon of water. Of these the first is the least likely to stain or injure the articles to which it is applied, which should be those that cannot be subjected to dry or moist heat, including articles made wholly or in part of leather, rubber, fur, or pasteboard. It is also used for moistening cloths for wiping floors or woodwork of rooms. Corrosive sublimate is not a good disinfectant for sputa or fæces, as it forms an insoluble compound with albumins. For these matters a solution of chloride of lime, 4 ounces to the gallon of water, is the best, provided the chloride of lime contains at least 25 per cent. of available chlorine. An infectious stool from a typhoid, cholera, or dysentery patient cannot be disinfected by pouring a little strong disinfecting solution on it, shaking it around a little, and then emptying the vessel. About a quart of the solution employed should be placed in the vessel in which the stool is discharged, and the mixture should remain in the vessel at least three hours before it is emptied. Equal parts of pure sulphuric or hydrochloric acid and water will disinfect a stool in two hours. The acid corrosive sublimate solution, 1 : 500, will do it in six hours, and a 5 per cent. solution of carbolic acid (about 8 ounces to the gallon) will do it in twenty-four hours. If solid fæces be present, they must be broken up and thoroughly mixed with the solution. Strong milk of lime, made by slacking fresh-burned quicklime and stirring up the fresh powder in twice its bulk of water, will kill typhoid bacilli in equal parts of a fresh liquid stool in about half an hour. If the problem is to deal with large masses of excreta, as in an old privy-vault, the chloride of lime or 5 per cent. carbolic-acid solutions are the best. Sulphate of iron is a deodorant for masses of excreta or sewage, but it is not a disinfectant, and is but slightly antiseptic. Its use is not to be recommended.

A 5 per cent. solution of sulphate of copper, a 10 per cent. solution of chloride of zinc, and a 15 per cent. solution of chlorinated soda will kill the ordinary bacteria, but not spores. Such solutions are more costly than the substances previously mentioned and present no special advantages. Most of the patent and proprietary disinfectants on the market are useless, and those that are not cost from ten to one hundred times as much as the satisfactory solutions above given. The physician has no guarantee that their composition remains constant, and had better confine his prescriptions to fresh-made articles of known composition and efficiency. For certain special and limited purposes, as in dealing with the micrococci of suppuration and occasionally to sterilize the hands of the obstetrician or surgeon, the solution of peroxide of hydrogen is convenient and useful. The hands of the surgeon and his assistants, as well as the skin of the patient, may be disinfected by washing first in a warm saturated aqueous solution of permanganate of potash, then in warm saturated aqueous solution of

oxalic acid, and finally in corrosive sublimate, 1 : 500. The body of a person dying of infectious disease should be wrapped in a sheet thoroughly saturated with the strong corrosive-sublimate or chloride-of-lime solution.

Isolation.

That it is desirable to prevent communication between healthy persons and persons suffering from communicable disease is generally admitted; but how this is to be done without causing additional suffering and danger to the sick, or great inconvenience and cost to others, is sometimes a difficult question. By the laws of Moses the leper was to be driven out of the community and his house, clothing, etc. destroyed by fire, but at present it is required that the leper shall be cared for as well as the community. As regards individual cases, when the family occupies a separate house, one room of which can be given up to the sick person and his attendant, it is always theoretically possible to provide such isolation as, combined with proper disinfection, is sufficient to secure protection for the rest of the family and of the community; but it is difficult, especially in mild cases of scarlatina, diphtheria, measles, etc., to make sure that such isolation and disinfection are properly carried out, and in tenement-houses and where the family occupies but one room it is practically impossible to do this, and therefore it is highly desirable that special hospitals be provided for the care of such cases.

One of the most important questions which the physician is called on to answer in scattered or so-called sporadic cases of the acute contagious diseases of children is as to the time during which the child should be kept isolated. This varies for each specific disease, and varies somewhat in individual cases, but the following may be considered as the minimum time, after the appearance of the eruption or other specific symptom, which should elapse before the child is permitted to be with other children: Scarlatina, 40 days; measles, 25 days; whooping cough, 40 days; mumps, 28 days; rötheln, 14 days. As regards diphtheria, the time is usually given as 40 days, but the fact is that it should date from the complete destruction of the specific bacilli, as shown by bacteriological examination, and the time required to demonstrate that such destruction has been effected may vary from ten days to eight weeks.

The isolation of a number of people—as, for instance, the crew and passengers of a vessel in what is known as maritime quarantine, or of a town in which an epidemic is raging—is occasionally useful for a short time to allow disinfection, vaccination, etc. to be carried out.

Food.

The force expended in a healthy man doing an average day's work is equal to about 3400 foot tons, of which 2840 foot tons are used in the production of heat. This force must be supplied by the tissues and fluids of the body, which, in turn, must obtain it from food. It is stored in the food in complex compounds of carbon, hydrogen, oxygen, and nitrogen, which can easily be reduced to simpler combinations, and in such reduction give out the

force, derived mainly from the sun's heat, which has been stored up by green plants.

The diseases connected with food may be due to defective or excessive supply, especially of certain principles, or to harmful or poisonous substances or living organisms contained in it, and include such various forms as rickets, scurvy, gout, alcoholism, and specific and parasitic diseases of various kinds. Certain substances, such as phosphorus, lime, iron, and potash salts, are required to maintain a healthy condition of the body—not to furnish force, but to supply structural material. Phosphate of lime constitutes about 50 per cent. of bone, and if the supply is deficient bone-softening, or rickets, may be produced. Iron is an essential constituent of red blood-corpuscles; if the supply is deficient, anæmia is the result. If the potash salts are deficient, scurvy is produced, especially if, at the same time, chloride of sodium is taken in excess. A continued diet of starchy foods and salt meats without fresh vegetables, fruits, or potatoes will usually produce scurvy.

The amount of food required daily by a laboring man doing moderate work should include about 125 grams of proteids, the same of fats, and 450 grams of carbohydrates or the starchy foods; and he will get this in about 20 ounces of meat, 22 ounces of bread, 10 ounces of potatoes, and 3 or 4 cups of coffee. The cooking of food is desirable to make it tender and soluble in the digestive fluids, to give it an agreeable flavor, and to kill parasites. The most dangerous animal parasite in meat is the trichina spiralis. It is killed by thorough cooking, and it is not worth while to take any special precautions against it merely in order that a few men may eat their pork raw with impunity. The flesh of animals dying of acute disease is not dangerous if well cooked, but it is not a desirable article of food. The flesh of tuberculous animals is somewhat dangerous even when cooked. Probably $\frac{1}{2}$ of 1 per cent. of the beef sold in market comes from animals in whom tubercle existed at the time of death. The systematic inspection of all animals which are to be slaughtered for food before they are killed, and of the meat after killing, is desirable. This can best be done in public abattoirs. As a general rule, animals affected with anthrax, septicæmia, glanders, cattle plague, swine plague, sheep-pox, and rabies should be killed and the bodies destroyed.

In cases of foot-and-mouth-disease, acute pneumonia, actinomycosis, dropsy, tuberculosis, and non-specific febrile disease the meat should be carefully examined after slaughtering to determine whether any part of it is fit for food or for industrial purposes.

The meat of animals dying of disease, and of very young animals, should not be used for food

Of all articles of food, milk is one of the most important from the sanitary point of view. It is used uncooked more than most other articles of food; it is often adulterated; it contains numerous bacteria, for which it forms an excellent culture fluid, and a number of typhoid fever, scarlet fever, and diphtheria epidemics have been traced to the use of milk from a particular dairy. It may also contain the bacillus of tubercle, and a certain amount of

the tuberculous meningitis and tabes mesenterica of infants is no doubt caused in this way. It may be sterilized by heating it, but the heat required to produce complete sterilization injures it as a food for infants, and raising it to a temperature of 160° appears to be sufficient to prevent the progress of the lactic-acid fermentation for as long as it is usually required to keep it. In case of a localized outbreak of typhoid fever the milk-supply should be investigated.

With the exception of milk, the adulterations of food practised in this country are of little sanitary importance. Ground spices, coffee, etc. are generally adulterated more or less, but not dangerously so. The most dangerous adulterations are those of drugs and of coloring matters.

Oleomargarine, if properly made, is not dangerous and is a useful article of food.

The production of a form of chronic arsenical poisoning by emanations or dusts given off from colored wall-papers containing arsenical pigments occasionally, though very rarely, occurs. It probably requires a marked peculiarity in constitution, as well as the ingestion or inhalation of the very small quantities of arsenic which are given off from such a source, to produce disease.

Exercise.

The usual estimate of the amount of muscular exercise required to keep a man in good condition is that it should equal about 150 foot-tons, or a walk on level ground of about nine miles. Each individual has his own peculiarities in this respect, and requires a particular amount to secure good appetite, complete digestion, restful sleep, and freedom from too great accumulation of fat. It depends on the amount and kind of food taken as much as upon anything else under ordinary circumstances. In addition to its general effects on health, it may be employed to strengthen particular groups of muscles for the purpose of correcting faulty development or to produce prompt co-ordination, or, as it is commonly called, "skill," in the performance of certain actions. It may also be used mainly for its effects on the nervous centres, which are quite as important as those on the muscles. The exercise used in training for a boat- or foot-race is designed not only, or even mainly, to increase the size and strength of the muscles of the arms, legs, and trunk, but to produce a heart, large blood-vessels, and lungs which will be competent to receive, propel, and aërate the greatly-increased quantity of blood which is forced to them in the violent effort of a "spurt." Owing to the valves in the veins, the contraction of the muscles of the extremities forces the blood in the veins inward to the heart, and when these contractions are rapid, continued, and strong, they exert a powerful pumping action on the blood, and at the same time much increase the normal quantity of carbonic acid contained in it. In the course of six weeks' training, with plenty of running, dumb-bell work, etc., a man gets a new heart, a bigger and stronger one, with larger orifices and pulmonary arteries and veins, provided always that his training is not overdone. If now, after the race is over, he ceases to take exercise, this large heart and arteries

must readjust themselves to the changed conditions, and there is some risk in the degenerations by which this change is accomplished. It is not wise to create in the system an artificial and excessive demand for exercise to secure comfort and pleasure, when such demand is not likely to be gratified in future years. The taking of exercise merely for its own sake soon becomes to most men a task for which they grudge the time, and to get rid of which they are glad to find an excuse. It is for this reason that companionship in exercise is desirable, or that it should be obtained in making exertion for some other object. For young men and boys gymnastic exercises are by no means a complete and satisfactory substitute for the ordinary out-of-door games and athletic contests, partly because they are not entered into with such zest and enjoyment, and partly because they do not exercise the brain so much. Work in the gymnasium should, however, be used as an accessory to games, and under competent direction and supervision it will effect much in developing special groups of muscles which are in need of increase of size and power, and it is also well suited to the needs of men of middle age.

Mere muscular strength and development furnish little protection against specific epidemic diseases.

Clothing and Bedding.

From the hygienic point of view the value and defects of particular kinds and styles of clothing are judged by the completeness with which they protect the person from the effects of extremes of temperature, the extent to which they interfere with the circulation or the shape or movements of the body or limbs, and the freedom with which they permit the exhalations from the skin to pass off into the surrounding atmosphere. The diseases due to insufficient clothing of the upper part of the body or of the limbs occur chiefly in young children and in women. Undue compression of certain parts of the body by tight lacing, close-fitting sleeves, garters, shoes, etc. occurs chiefly in women who try to follow the fashions of the day. As a rule, linen garments next the skin, or linen sheets, are not desirable, and by persons of rheumatic or neuralgic tendencies they should be carefully avoided. In cool weather, or when rapid changes in temperature are likely to occur, woollen under-clothing is best. It conducts heat badly and absorbs perspiration readily. During exercise in warm weather a man with woollen under-clothing will at first feel warmer than the man who has cotton or linen next his skin, but after a little time the difference in this respect will be small; and when the exertion ceases the man with the woollen under-clothing will be much less apt to catch cold or to have twinges of muscular rheumatism. The chief objection to woollen under-clothing in warm weather is that men are not likely to change it as often as is desirable. Pure wool under-clothing requires more skill in washing to prevent shrinkage and the loss of some of its desirable properties than is usually obtainable. Some persons, usually women, assert that they cannot wear woollen next the skin because of the irritation which it produces, but if fine woollen or merino garments be used, this objection almost always disappears in about two weeks. In very cold,

windy weather skins, furs, and leather give the greatest protection. It is not healthy to wear waterproof clothing continuously for any great length of time. In hot weather, in the shade, thin cotton or linen clothing is the most comfortable. In the sun, color is of more importance than texture or material as a protection against heat, white being the coolest; in the shade, color makes very little difference in this respect. Dark-colored clothing absorbs and retains odors more readily and persistently than light-colored clothing of the same material and texture, and wool more than cotton or linen. The most comfortable and healthy bed is composed of a hair mattress on metal springs, with cotton sheets and woollen blankets. Feather beds are not desirable. It is best that each person should have a separate bed. Clothing may be the means of transmission of infection, either from the homes of those who manufacture it or from those who have worn it, as by so-called second-hand clothing. The virus of small-pox, of scarlatina, and of yellow fever has been transmitted through clothing and bedding, and the disinfection of such articles in cases of contagious disease is of much practical importance.

Occupation.

Almost every occupation produces special liability to certain forms, and a certain amount of immunity from other forms, of disease or injury on the part of those engaged in it, but the net result of a particular trade or profession on the health and life of men is often very difficult to determine. Only men of considerable strength and vigor can undergo the muscular exertion required in certain forms of labor, hence weak and sickly men either do not engage in these occupations or leave them for lighter work. What are called easy, light occupations attract weak lives; hence the difference in the death-rate of blacksmiths and of clerks cannot be taken as the measure of the difference in healthfulness of the two occupations. The average age of those engaged in a particular occupation is also of great influence on the death-rate, which for this reason tends to be lower for medical students than for practising physicians. The influence of place of residence and of social status and habits, especially as to use of alcohol, is also very great in certain kinds of occupations. The most extensive and reliable series of data as to the relative death-rates in different occupations yet published is given by Dr. Ogle in the supplement to the forty-fifth annual report of the Registrar-General of England, and in a paper read before the Hygienic Congress in London in 1891. The following (A) is his table of comparative mortalities of men between twenty-five and sixty-five years of age in different occupations, the death-rate of clergymen, the lowest of all, being taken as the standard of comparison and represented by 100. The special causes of disease and injury directly connected with particular occupations may be classed as follows: viz. 1, accidents; 2, poisonous materials; 3, dust; 4, gases and vapors; 5, excessive temperature; 6, abnormal atmospheric pressure; 7, excessive use or strain of certain parts of the body; 8, special exposure to contagious or parasitic diseases. Of the poisonous materials, lead is the most important, as producing the greatest amount of

A.—*Comparative Mortality of Men (twenty-five to sixty-five years of age) in Different Occupations, 1881–83.*

Occupation.	Compar. Mortal.	Occupation.	Compar. Mortal.
Clergymen, priests, ministers	100	Carpenters, joiners	148
Lawyers	152	Cabinet-makers, upholsterers	173
Medical men	202	Plumbers, painters, glaziers	216
Gardeners	108	Blacksmiths	175
Farmers	114	Engine, machine, boiler-makers	155
Agricultural laborers	126	Silk manufacture	152
Fishermen	143	Wool, worsted manufacture	186
Commercial clerks	179	Cotton manufacture	196
Commercial travellers	171	Cutlers, scissor-makers	229
Inn-keepers, liquor-dealers	274	Gunsmiths	186
Inn, hotel service	397	File-makers	300
Brewers	245	Paper-makers	129
Butchers	211	Glass-workers	214
Bakers	172	Earthenware-makers	314
Corn-millers	172	Coal-miners	160
Grocers	139	Cornish miners	331
Drapers	159	Stone, slate-quarriers	202
Shopkeepers generally	158	Cab, omnibus service	267
Tailors	189	Railway, road, clay, etc. laborers	185
Shoemakers	166	Costermongers, hawkers, street-sellers	338
Hatters	192	Printers	193
Bookbinders	210	Builders, masons, bricklayers	174

disease. Manufacturers of white lead, painters and glaziers, plumbers, workers in rubber factories, and file-makers are specially liable to be affected from this cause with colic, local paralysis, and various obscure forms of disease of the nervous system and of the urinary organs. Tailors and seamstresses sometimes suffer from lead-poisoning from the use of sewing silk treated with sugar of lead, especially if they have the habit of biting off such thread. Chronic mercurial poisoning occurs in gilders, looking-glass makers, and hatters; necrosis of the jaws in workers in phosphorus, especially in match-makers; arsenical poisoning in zinc- and brass-founders and in workers in papers, feathers, etc. tinted with arsenical colors. Irritating dusts produce diseases of the lungs and air-passages which predispose to phthisis, as will be seen by the following table (B) of Dr. Ogle:

B.—*Comparative Mortality of Males in certain Dust-inhaling Occupations from Phthisis and Diseases of the Respiratory Organs.*

Occupation.	Comparative Mortality from—		
	Phthisis.	Diseases of Respiratory Organs.	Phthisis and Diseases of Respiratory Organs.
Coal-miners	64	102	166
Carpenters, joiners	103	67	170
Bakers	107	94	201
Masons, bricklayers, builders	127	102	229
Wool,- worsted-workers	130	204	234
Cotton-workers	137	137	274
Quarrymen	156	138	294
Cutlers	187	197	384
File-makers	219	177	396
Earthenware-makers	239	326	565
Cornish miners	349	231	580
Fishermen	55	45	100

Alcohol is also to be reckoned among the poisonous materials, as is shown by the following table:

Mortality of Dealers in Liquor (twenty-five to sixty-five years of age) from Various Diseases, compared with that of Men generally of the same Ages.

Diseases.	Mortality of—	
	Liquor-dealers.	Men generally.
Alcoholism	55	10
Liver disease	240	39
Gout	13	3
Diseases of nervous system	200	119
Suicide	26	14
Diseases of urinary system	83	41
Diseases of circulating system	140	120
Other diseases	764	653
All causes	1521	1000

Dangerous gases and vapors are evolved in chemical and color works, in the manufacture of sulphate of ammonia from the refuse of gas-works, in India-rubber works, etc. Excessive temperature and rapid changes of temperature affect glass-blowers, puddlers, and firemen in steamships and workers in certain mines, producing diseases of the respiratory organs and rheumatic affections. Abnormities of atmospheric pressure, and especially rapid changes in the pressure, affect workmen in compressed air, producing rupture of the membrana tympani and paralytic affections of the nervous system known collectively as the "caisson disease." The chief danger occurs in the rapid passage from a denser to a thinner air, producing tendencies to congestion and hemorrhages in internal organs. For the same reason persons having tuberculous cavities in the lungs are liable to attacks of pulmonary hemorrhage in passing rapidly by rail to high altitudes. For most of the special causes of disease in factories and workshops the specially important precautions are personal cleanliness and abundant ventilation. In many cases dust or vapors can be at once removed by fans or blowers, and in most cases dangerous and offensive gases connected with waste products can be avoided or converted into materials of value by proper methods of dealing with these products.

Habitations.

Physicians are rarely consulted in the selection of a site for a dwelling, and even more rarely in the preparation of plans. Occasionally, however, they are called upon for an opinion as to whether a particular house is unhealthy, and, if so, what should be done to improve it, or whether a change of residence is necessary to secure satisfactory results in the treatment of a particular case. In cities most men select their dwelling-places with special reference to cost, vicinity to their place of business, kind of neighbors, etc., rather than to sanitary conditions, with regard to which they have little choice. The sanitary character of a building-site is determined mainly by its elevation and exposure to prevailing winds and the dryness and kind of soil in the immediate vicinity.

An elevated site is desirable as securing abundance of fresh air and facilities for good drainage; but in the rural districts convenience of access and of water-supply must often be the first points to be considered. In some localities shelter from cold northerly winds, and in others from winds coming from over low marshy grounds, is very important. Rock, gravel, and pure sands are healthier sites than clay and alluvial soils, because they are dryer if sufficiently elevated. Damp sites are unhealthy, having a special tendency to produce or to aggravate diseases of the air-passages and rheumatic affections. The inhabitants of such sites are especially liable to pulmonary phthisis and to diphtheria, possibly because the specific bacilli and spores of these affections retain their vitality better in such localities, possibly because the slight colds and catarrhs which such sites tend to produce modify the respiratory tract so as to make it easier for the specific germs to effect a lodgment and to multiply and develop. Under the same general conditions of climate diseases of the respiratory organs are more fatal on damp soils than on dry ones.

Soil moisture or dampness refers to the water in soils that also contain air. When there is no air in the soil interstices and the water is continuous, it is called ground water. All soil contains a large proportion of interstices filled with either air or water: in coarse dry sand or gravel or in coarse sandstone this amounts to one-third of the bulk. When filled with air this air is always in motion, and enters buildings freely through the floors and sides of the cellars or basements, especially in cold weather, when the air in the house is warmer than that outside. Soil air always contains a greater proportion of carbonic acid than the atmosphere, and this proportion increases with the depth. Thus, while the atmosphere contains about 0.4 parts per 1000 of CO_2, the upper layers of the soil contain from 1 to 3 parts, and at a depth of fifteen feet it may contain from 50 to 70 parts.

In cities the soil of streets is liable to contain illuminating gas from leaky mains, and this may be drawn into the cellar of a house from a distance of from thirty to fifty feet. The excess of carbonic acid in soil air is greater in soils containing much organic matter, and is therefore, to a certain extent, a measure of the organic contamination of the soil; but it does not always depend on local oxidation processes, nor is it in itself a matter of much sanitary importance. It is, however, necessary to bear in mind this excess of CO_2 in cellars, due to soil air, in testing the air of rooms with reference to ventilation, for otherwise very erroneous conclusions may be drawn.

Like the soil air, the soil or ground water is continually in motion. It varies in height at different places and at the same place at different times. Where the ground water is always below fifteen feet from the surface, it is healthy so far as this is concerned. When the level of the ground water is above this, it is healthier when it remains at about the same level than when it fluctuates. In some places, as in Munich, typhoid fever increases as the ground water falls, but this is by no means always the case, and it probably depends to a considerable extent on the condition and amount of use of shallow wells.

As the soil water is constantly in motion, and for each locality this motion is tolerably uniform in direction and velocity, it follows that in privy-well and cesspool villages and towns it may be much more contaminated in one part of the town than in another.

Nearly every form of micro-organism may be found in the soil at different places and times, and their number and character depend on the moisture and temperature and on the presence of suitable food material. From the sanitary point of view the most important of these are the Plasmodium malariæ, the bacillus of typhoid, of tetanus, of anthrax, of tuberculosis, of diphtheria, and of cholera, and the nitrifying organisms. The pathogenic micro-organisms or their products may pass into the soil water, being washed down by rainfall, or into the air with particles of the surface soil blown about as dust. They cannot be drawn far through soil by air-currents, especially if the soil is slightly moist. To prevent ground air and dampness from entering the cellars of dwelling-houses, the floor, and the sides of the cellar up to ground level, should be laid with bricks soaked in asphalt. A cement floor is quite pervious to air when it becomes dry. In low sites, and especially in malarious regions in warm climates, it is better to have no cellar beneath the house, which should be raised on piers, posts, or arches. The natural processes for the purification of soil containing much organic matter of animal origin, such as the made ground in the suburbs of cities or the ground in the vicinity of leaky cesspools or of graves, is a slow one, requiring from three to eight or more years, according to the porosity of the soil and the accessibility of fresh air to the interstices. Hence, when a system of sewerage is introduced in a city which has previously been storing up its filth in cesspools, it requires some time for the nitrifying organisms to complete the work of purifying the polluted soil.

WATER-SUPPLY.

Water is sometimes considered as a food, because it is taken into the body through the alimentary canal, but it supplies no force for the production of either heat or motion. It is, however, the universal medium in and through which the processes of life occur and the products of vital action are removed and excreted. 58 per cent. of a man's body is composed of water; he must have from 60 to 90 ounces a day in his food and drink to maintain his weight and strength, and he needs a much larger quantity externally to keep his skin and his morals in good condition. In a fairly cleanly household the average necessary consumption of water per head per day is from 10 to 15 gallons. For all purposes the water-supply of a town should not be less than 18 gallons per head per day; if it is used freely and none is wasted, it will require about 25 gallons per head per day. The average supply in the larger American cities is more than three times this, the greater part being wasted through leaky fixtures.

This constant use of water by every living being makes the quality of the water used of great importance, as it is very liable to contain matters injurious to health. The most important of these are the micro-organisms which

cause disease, and especially those which produce diarrhœal and dysenteric affections, cholera, typhoid fever, and, sometimes, malaria.

Water may also contain poisonous salts, as of lead, or excessive amounts of magnesia and lime, giving rise to goitre or to calculus. No water in ordinary use is chemically pure: rain-water, snow, and hailstones contain organic matter and living micro-organisms. A good drinking water should have a bluish tint when in a layer of three feet thick; it should be limpid, cool, without odor when cold or when heated, and it is most palatable when it has a very faint taste of acid and of salt. A good water should not contain more than 20 parts of lime per 100,000, or it will be "hard," so that it will not easily form a lather with soap and is not well suited for laundry and cleansing purposes.

There is no simple, easy means by which any one can assure himself that a water contains nothing harmful, but in bad cases the sense of smell, taste, and sight will assure him that it is not fit to drink. By chemical analysis we can discover the amount and, to some extent, the source of the foreign matters present, and can usually tell whether it is contaminated with sewage or not. This last is indicated by the presence of an excess of chloride of sodium and by the long-continued production of free and albuminoid ammonia in distillation with an alkaline permanganate, indicating the presence of urea. Much care is necessary in obtaining the samples to be examined. Chemical analysis tells nothing about the living organisms in the water. Something may be learned about these by mixing a drop of the water with a little melted peptone gelatin, spreading the mixture in a tube or on the bottom of a thin, shallow glass dish, and cultivating the mixture. In this way it is possible to determine approximately the number of bacteria in a given quantity (as a cubic centimetre) of the water, and the nature of some of these bacteria can be discovered by subsequent pure culture methods; but it is rarely possible, by either chemical or bacteriological analysis, or by both combined, to make sure that a water is free from disease-germs, although it is often possible to be positive that it is polluted.

If it is suspected that a well or spring is being polluted from a neighboring leaky cesspool or privy-vault, the question can sometimes be settled by throwing a large quantity of crude carbolic acid or of common salt into the cesspool or vault. If there is communication, the peculiar odor and smell of the acid or a considerably increased proportion of the salt will be found in the well water.

The most reliable sources of what is ordinarily called a pure water-supply are springs and deep wells in the open country and streams coming from uncultivated and uninhabited uplands. Surface water from cultivated land is dubious; streams or ponds into which sewage is discharged, and springs and shallow wells in cities, furnish dangerous waters. The danger is mainly due to the possible presence of disease-producing bacteria which have passed from the bodies of sick people into the water through sewage contamination or as air-blown dust, but it may also be due to an excess of the products of organisms which in small quantity are harmless.

In a general way, it may be said that a well drains a funnel-shaped area, the radius of the top of which is equal to its depth, and this whether its diameter is two inches or three feet. The shape and area of the ground which it drains depend on the nature of the water-bearing strata or the velocity of the ground-water current, and on the amount that is drawn from it.

A general water-supply is desirable, because it usually gives a purer and more wholesome water than the wells or cisterns of a town, because it saves much labor, promotes cleanliness, lessens the danger from fires, permits of watering the streets, and increases comfort and happiness in many ways.

On the other hand, when a general water-supply does become dangerously contaminated, its effects are widespread, and it necessitates the provision of means whereby the large amount of water brought in and made foul by use can be taken out again without producing nuisance or danger to the town itself or to its neighbors. A general water-supply may become polluted at its source, or while it is in an open stream or pond, or while stored in reservoirs, or while in the distribution-pipes. The pollution which occurs in reservoirs is due to the growth and decay of various species of algæ or of fresh-water sponges, producing unpleasant odors. Uncovered reservoirs more frequently become affected in this way than covered ones, light being necessary for the development of the algæ which produce them. While in the distributing pipes the water may become contaminated by sewage if the pipes are leaky, and especially if the supply is intermittent. Such contamination may be suspected when a sudden outbreak of typhoid fever occurs, confined to the houses supplied by a particular water-main, and there is no other circumstance common to these houses, such, for instance, as a common milk-supply.

The typhoid bacillus has been known to pass many hundred feet beneath a mountain and infect a spring at its base, and to preserve its vitality for several weeks in excreta thrown out on snow, and then, through the melting of the snow, pass into a stream and produce an extensive epidemic.

When a running stream has been polluted by sewage, a process of self-purification occurs by sedimentation, by the action of bacteria, and of microzoa which feed upon the organic matters. The rapidity and completeness with which this natural purifying process goes on depend on the amount of dilution of the sewage, the presence or absence of fine particles of clay, which produce sedimentation, and especially on the amount of oxygen present in the water, which determines the character of the bacteria which flourish in it. If there is abundance of oxygen, those bacteria which require it for growth will multiply and consume the organic matter to the exclusion of other forms. Such bacteria are called ærobic—that is, air-loving—bacteria, and among these are the nitrifying organisms, which will be referred to hereafter in speaking of the filtration of sewage.

When it is necessary to use water which has been polluted by sewage, it may be rendered harmless by boiling, and thus sterilizing it, or by certain methods of filtration and aëration. The only small household filters which can be relied on to remove bacteria are those made of unglazed porcelain, and

these will only do so for two or three days, at the end of which time they must be thoroughly cleansed and sterilized. On the large scale the cheapest and most satisfactory filters are constructed of sand, but their action must be intermittent, as will be explained in speaking of sewage filtration. Spongy iron also makes a good filter, and a combination system by which iron is showered through the water in a revolving cylinder, with subsequent aëration and sand filtration, gives good results.

The freezing of water does not destroy the vitality of the micro-organisms contained in it. It kills some of the soft micrococci and bacteria, but only a portion, and has little or no effect on spores. The bacillus in typhoid will preserve its vitality and powers of development in ice for several months; hence, ice cut from a sewage-contaminated pond may be very dangerous.

The jurisprudence of water-supplies is in an unsatisfactory condition in the United States. The common law of the subject rests on contradictory decisions of different courts, and where there is not clear and definite statute law upon the subject it is very uncertain in any given case as to how far manufacturing or other interests of more or less public importance will be allowed to override the health interests of individuals or of small communities. The general principle is, that a person living on the banks of a stream has the right to demand that the water of this stream shall continue to come to him in its natural purity and volume, but that, if pollution has been going on for twenty years without complaint or attempt at interference, what is called a prescriptive right to continue such pollution is established.

The fact that a person or corporation owning property on the banks of a stream does not use the water does not prevent them from bringing an action to protect themselves against the acquirement by others of a prescriptive right to pollute the stream, thereby depriving them of their rights in future. It has also been decided in one case that the principle applies to subsoil water while on its passage to springs or wells, and that therefore the placing a cesspool or drainage from gas-works in such a position as to pollute a well is good ground for action for damages.

When it is possible to prove to the satisfaction of a court that actual disease and death have been caused by the pollution of a water-supply by sewage, no doubt the nuisance can be stopped and damages collected in many cases; but it is rarely possible to prove this. In some cases it is a question whether it is not best for the public to abandon a stream to pollution, so long as it does not injure the public health. It is generally admitted that the discharge of excreta into a stream the water of which may be used lower down for drinking purposes is unlawful, but, practically, it is not possible to prevent a small amount of this contamination in most cases. What amount of contamination is excessive and unnecessary is a question to be decided separately for each particular case.

So far as statute law is concerned, the best form is probably that of the

State of New York, which authorizes the State Board of Health "to make rules and regulations for protecting from contamination any and all public supplies of potable waters and their sources within the State."

Sewage-Disposal.

Water-supply, and its pollution and purification, are closely connected with the subject of sewage-disposal. By "sewage" in this connection is meant water made foul by use in habitations and manufactories or by street-washings. It is a complex liquid, containing a large amount of organic matter and innumerable micro-organisms, but varying much in composition in different places, or in the same sewer at different hours of the day. Chemically, and as regards the amount of organic matter, the sewage from towns where it is not allowed to connect water-closets with the sewers does not differ greatly from that from water-closet towns. Ordinary sewage has been drunk with impunity; diluted sewage, as found in the shallow wells of most small towns and villages is constantly imbibed with only occasional bad results. The sewage from a single house rarely contains the specific bacteria of cholera, typhoid, or dysentery, but that from a large city will rarely be free from those of typhoid, and never from those capable of producing intestinal irritation.

In considering the question of the disposal of the sewage of a particular locality there must be taken into account the probability of its containing specific causes of disease, and the communication of these to water-supplies; its liability to produce offensive odors; its effects on fish or on the fitness of a stream for manufacturing purposes; and its value as a fertilizer. When there is no general water-supply the amount of sewage produced is comparatively small, and it is usually disposed of on the premises by means of a cesspool or privy-vault or by being thrown on the surface of the ground or into the gutter. To remove it entirely from a town some system of water-carriage is necessary, and this requires a general water-supply; while, as mentioned above, a general water-supply requires some kind of system of sewers to remove the fouled water. 1000 adults excrete each day about 250 pounds of fæces and 375 gallons of urine. Practically, the amount of sewage from a community may be taken as equal to the amount of its water-supply. In considering the merits of different systems of residential sewage-disposal the chief points to be borne in mind are as follows: 1. Fresh sewage contains a large amount of dead organic matter in complex forms of combination. 2. These complex forms are to be decomposed and recombined into simpler forms, such as nitrates, ammonia salts, etc., in which the combined nitrogen is in a form suitable to nourish plants. 3. The natural process of effecting this is by the action of bacteria. 4. The bacteria which grow and multiply best when there is little or no free oxygen present—*i. e.* the anærobic bacteria—do not effect this decomposition into simple salts, but produce substances which are more complex, more dangerous to health, and more offensive to the sense of smell than are the products of those which grow best in abundance of free oxygen—the ærobic bacteria. 5. In most cases it is not desirable to prevent

the action of the ærobic bacteria, which should be favored as much as possible.

In cities, towns, and villages it is not desirable to turn the sewage into cesspools. If these are watertight, which is rarely the case, the ærobic bacteria can act only on the upper surface; offensive gases are generated below, and, as the vaults must be emptied from time to time, the cost of doing so is considerable, especially if there is a general water-supply, and the final disposal of the matters removed is still a difficult problem. If the cesspools are leaky, the surrounding soil becomes polluted, affecting the soil air and the soil water, which are liable to contain specific disease-germs and are certain to contain unpleasant gases and vapors. Taking cities of the same size and density of population, the annual death-rate in the unsewered cesspool cities is from 3 to 8 per 1000 greater than it is in sewered cities. For cities, and especially for large cities, the best method for removing the sewage is by water-carriage in a system of watertight channels or sewers. For suburban residences, country institutions, temporary encampments, and in very cold climates the so-called dry systems of sewage-removal may be used: where there is no system of sewers with which to connect, earth-closets are the best for this purpose, as a rule.

When sewage is removed by water-carriage, it may be finally disposed of by allowing it to flow into a neighboring stream or large body of fresh or salt water; by spreading it over the surface of land for the purpose of fertilizing it and of raising crops, which is known as broad irrigation or sewage farming; by spreading it beneath the surface of land through a system of small, open-jointed earthen pipes, which is called subsurface irrigation; by filtering it through soil; by treating it with various chemicals to purify it; and by combinations of these methods.

The turning of sewage into a stream or lake is often the cheapest method, so far as immediate cost is concerned; but it dangerously pollutes the water, is generally undesirable, and is becoming more so as the country becomes more thickly settled.

Sewage farming is, theoretically, the best means of sewage disposal where a sufficient quantity of suitable land is available, because it uses the sewage as a fertilizer, and thus utilizes the stored force in its nitrogen compounds. This form of stored force is essential to vegetation, and therefore to animal life; the amount of it in and on the earth is limited, and when destroyed it is not easily replaced. It steadily diminishes in a soil cultivated without fertilizers.

At present the commercial value of sewage as a fertilizer is, in most localities, insufficient to repay the cost of its collection and application; but as population increases and the price of fertilizers rises the value of sewage will increase. To obtain satisfactory purification and fair returns from crops the amount of land required for sewage farming is 1 acre to from 75 to 150 persons, depending on the porosity of the soil and the depth above the level of the subsoil water.

Subsurface irrigation is especially useful for country houses, asylums, etc.,

where there is a slope from the building to suitable ground adjacent, of which 1 acre is sufficient for the sewage from 75 persons.

The slow, intermittent filtration of sewage through sand, in such a manner as to promote the growth throughout the filter of masses of nitrifying organisms is at present one of the best known methods of sewage-disposal. The application of the sewage must be intermittent, so that there shall always be abundance of air in the filter. Each acre of such a filter, properly constructed and managed, will purify the sewage coming from about 1000 persons; and the fluid which escapes from it will be a clear, odorless water, containing inorganic salts in solution, not susceptible of putrefaction, and free from specific pathogenic bacteria and their dangerous products.

By the addition of various chemicals, such as lime, alum, sulphate of iron, etc., about one-half of the organic matter and a considerable proportion of the bacteria of sewage may be removed; but the results are not as satisfactory as those obtained by intermittent filtration, and it is more costly.

Sewers may be constructed to receive only the water fouled by use in habitations, forming what is called a separate system; or to receive also the water of rainfall from roofs, yards, and streets, forming what is known as the combined system. Most large cities have the combined system. The separate system has its collecting branches made of pipes from six to twelve inches in diameter, laid with watertight joints; it is much cheaper in construction than the combined system, and is specially applicable to localities where the proportion of length of street to number of houses is large, as in villages and small towns. It is desirable where the sewage is to be disposed of by irrigation or filtration, as the amount of sewage should be as constant as possible in such cases.

To keep sewers in good condition and free from foul odors the sewage must be delivered to them fresh, and not as an overflow from a cesspool; the grades must be such as to secure a constant flow and to prevent stagnation of the liquid at any point; and they must be well ventilated.

The good effects of sewers on the health of a town are due not only to the removal of sewage, but to the fact that they act also as drains, and tend to prevent the subsoil water from rising above the level at which they are laid. This is true even where they are watertight tubes, as drainage takes place through the loose soil immediately surrounding them.

House Sewerage.

The main pipes placed in a house for the removal of excreta are called soil pipes, and these, with the pipes and fixtures connected with them, are usually spoken of as forming the system of house drainage. It is better, however, to restrict the term "drainage" to the removal of surface and soil water, and to call the system above referred to that of house sewerage. It consists of fixtures, such as water-closets, urinals, slop-sinks, bath-tubs, kitchen-sinks, wash-tubs, etc., of the waste pipes leading from them, of cisterns or tanks for flushing them of traps, of special ventilating pipes, and of the soil pipes as far as their

connection with a sewer or cesspool outside the house. The essential feature of a satisfactory system is that no air from the interior of the waste or soil pipes or from the sewer shall escape into the house or into any part of its water-supply; that all foul matters turned into the system shall be washed rapidly away without stagnation at any point; that the liability to obstruction of any of the pipes shall be as small as possible; and that if it does occur it can be easily located and removed. It is moreover desirable that the waste and soil pipes shall have a constant gentle current of fresh air passing through all parts of them as far as possible, in order to favor the growth of the ærobic bacteria in the slime which lines them, and thus to prevent the development of those organisms which produce foul odors as well as of those which cause disease, as explained in the section on Sewage-disposal. All this requires a proper plan of arrangement and connections, good materials and good workmanship, and care in use, with occasional skilled inspection to make sure that all the parts remain in good order. From such a system there is no special danger to health. As regards plan and arrangements, the plumbing regulations of most of our large cities are now fairly in accord and are satisfactory, the main points being as follows:

1. Soil pipes must be extra heavy cast-iron or standard wrought-iron pipe, not less than four inches in diameter and free from cracks, holes, and other defects; they must have a continuous fall toward the sewer and must be so put together as to be air- and water-tight at all joints.

2. Soil pipes must be extended full size up to and through the roof, and be freely open to the outer air at the top.

3. It is not desirable that the sewers should be ventilated through the soil pipes or through rain-water pipes in any case where the air escaping at the top of the pipe is liable to enter a window of the same or of an adjacent house. In most cases it is better to cut off the sewer air from the soil pipe by a trap between the house and the sewer, and to provide a fresh-air inlet to the soil pipe just inside of this trap.

4. Every fixture should have a trap on its waste pipe fixed as close to it as possible, and from the top of this trap there should be a ventilating pipe of a size not less than that of the waste pipe to which the trap is attached, which ventilating pipe should continuously incline upward and open above the roof. A mechanical trap which merely prevents siphonage is not a satisfactory substitute for the ventilation of the trap and waste pipe.

5. All water-closets or slop-sinks should be flushed from a special tank or cistern, and never directly from a water-supply pipe.

6. Waste pipes from refrigerators, from safes placed beneath fixtures, or from tanks or cisterns except flushing tanks, should not be connected with soil pipes, but should discharge in the open air.

7. The arrangement of the waste, ventilating, and soil pipes should be such that they can readily be inspected at all points.

About half of the houses having a sewerage system have one with defects in it of some kind which permit of the occasional discharge of soil-pipe air into

the house; hence the discovery of such a defect in a house in which there is sickness is no proof that the latter is caused by the former. In a well-sewered house the chief danger to health connected with the system occurs when the fixtures have been unused for two or three months, and the traps and interior of the pipes have become dry, so as to give off dust-particles which are carried into the rooms. The best-water closet is one of the all-porcelain wash-out forms, of which several varieties are in the market. The worst form is the pan closet. Trapless closets should be avoided.

Ventilation.

Most persons of average cleanly habits in this country would object to being compelled to wear under-clothing that had just been removed from the body of another man, or to use another person's toothbrush, or to eat food that had been partially masticated by another. They do not, however, often object to drawing into their noses, mouths, and lungs air that has very recently been inside another man's body; and upon the whole it is fortunate that they do not, for they cannot very well help doing so under the ordinary conditions of civilized life. The evil results of the continuous inhalation of impure air are not, in most cases, such as to attract notice unless the impurity is very considerable or the conditions of moisture and temperature connected with it are such as to produce evident discomfort. The injury inflicted on the body by breathing air deficient in oxygen and contaminated with animal exhalations is usually not perceptible until after a considerable period of time, and is then often attributed to other causes. The proof that this injury occurs has been obtained by comparison of the statistics of disease for a series of years among men living in unventilated with those of men living in well-ventilated barracks, prisons, etc., and also among cavalry horses kept in well- and ill-ventilated stables. The diseases which are especially produced or aggravated by defective ventilation are chronic inflammatory affections of the throat and lungs and certain forms of contagious disease, more especially typhus fever and phthisis. With regard to phthisis, this is due in part to the fact that the probabilities of inhaling the specific bacillus or its spores are greater where a number of men or animals are repeatedly breathing air containing the dried sputa and other excretions of their companions, and partly because the inhaling of air loaded with dead or dying organic matters tends to accumulate in the air-passages materials well suited for the nourishment of the specific germs, which in the absence of such food-material would be killed by the living tissues with which they would come in contact.

In ordinary life it is difficult or impossible to separate the effects of impure air from those of insufficient or improper food or clothing, or from those of general want and cleanliness, as, for instance, in studying the causes of the excessive mortality in a dense population, as in a tenement-house; but if the importance of respiration to life, the immense surface which the air-passages and air-cells present for the lodgment of particles, and the favorable conditions which these present for the growth of bacteria so far as moisture and

temperature are concerned, be considered, it is evident that the purity or impurity of the air breathed must be an important factor in the preservation or loss of health and energy.

Ventilation is the continuous and more or less systematic changing or renewal of the air in a room or other enclosed space. It may be used to remove watery vapor, as from damp walls or from the drying-room of a factory, or to remove dust or offensive or dangerous gases or vapors produced in certain manufacturing operations; but it is usually provided for the purpose of diluting and removing the products of exhalation and respiration of man and to regulate the temperature of apartments. To effect it the external air must be introduced in a continuous current and diffused throughout the room, and a corresponding quantity of air must be continuously taken out.

As a rule, we must take the outer air as we find it: at night we must use night air; in cities we must take it from the streets. It is true that by special appliances we can draw the air down through a tower and can filter it through cotton or through water-spray, but this is rarely necessary.

Perfect ventilation would ensure that a man inhaled no air which had recently been in his own lungs or in those of his companions. Good ordinary ventilation does not aim at this perfection: it merely ensures that the fresh air comes in in sufficiency, and is so thoroughly mixed with the air in the room that the products of exhalation and respiration are so diluted that when a man having a normal sense of smell comes into the room from the outside air he will perceive no unpleasant odor.

The matters given off from the skin and lungs consist of carbonic acid, watery vapor, dried epithelial scales, and certain nitrogenous products of decomposition belonging to the ammonia and amine groups. It is these last which are the source of danger and of odor. The carbonic acid has no odor, and in the proportion in which it exists in a crowded, unventilated room is not dangerous, but it increases in proportion to the dangerous nitrogenous matters; its amount is easily measured, and hence we judge of the effects of ventilation by the proportion of carbonic acid found present. This proportion should not exceed 2 parts in 10,000 over and above the amount which was in the air when it entered the room. If the air enters the room directly from the outer air, it will usually contain from 3 to 4 parts of carbonic acid per 10,000, and in that case, if the room is well ventilated, the proportion will not exceed 6 parts in 10,000 in the air in any part of the room. But if the air has come from the cellar or through an underground passage, it may contain from 7 to 12 parts of CO_2 per 10,000 as it enters the room; and this should be constantly borne in mind in attempting to measure the amount and completeness of the ventilation of an apartment by means of carbonic analysis.

The great majority of people suppose that ventilation means simply the removal of foul air, and that this can be effected by putting in some kind of an opening, or tube, or flue through which the foul air will either flow out or may be forced out. In very few private dwellings, even large and costly ones, are there any special provisions for the admission of fresh air to the several

rooms, and in the immense majority of school-rooms, lecture-rooms, theatres, and other places of public assembly, either there are no special arrangements for the supply of fresh air, or these are entirely insufficient for the purpose.

The amount of fresh-air supply required for a room depends upon the number of people who are to occupy it, and whether it is to be occupied only an hour or two at a time, or for several consecutive hours, or permanently. In a hospital ward which is permanently occupied and which requires the most, the air-supply should be not less than 1 cubic foot per second per bed, or 3600 cubic feet per hour per person. For bed-rooms, barrack-rooms, prison-cells, etc. the supply should be 3000 cubic feet per hour per head. For school-rooms, lecture-rooms, etc., which can be thoroughly aired out after two hours' occupancy, the supply may be from 2000 to 2400 cubic feet per hour per head. If double or triple these quantities can be furnished, so much the better, but as a rule this can only be done in warm weather, when the windows can be left freely open. The reason for this is that in cold weather the room must be kept warm, and the larger the amount of air that is passed through it the more fuel it takes to heat it.

If a room is warmed by hot air brought into it by flues and registers, as from a furnace or from steam or hot-water radiators in the cellar, it is said to be heated by indirect radiation. If the heating surfaces are in the room itself, it is said to be heated by direct radiation, and in this case no arrangements are usually made for warming the incoming fresh air, or indeed for providing any fresh air at all. If, however, fresh air is brought in so as to surround and be warmed by the stove, the back of the fireplace, or the steam radiator, the room is said to be heated by the direct-indirect method.

To get the requisite amount of fresh air into the room, flues and registers of sufficient size must be provided, and if the room be a large one, they should be at several different points, in order to secure a good mixture of the fresh with the foul air. The velocity of the air in the flues should be from 4 to 6 feet per second; as it comes through the register it should not exceed 4 feet per second. The usual rule is to allow about 24 square inches per head for the inlets—that is, 1 square foot for six persons—but this is a scant allowance, especially for hospitals. In a hospital ward for twenty-four beds the combined area of the inlet flues should be at least 6 square feet.

In rooms heated by indirect radiation the only way in which the temperature can be lowered if it gets too hot is, in most cases, to close the register and so shut off the supply of fresh air, or else to open the window, which is often undesirable. In a properly-constructed heating apparatus there is a by-pass around the radiator, so that by moving a valve the fresh air can either be brought around it or through it, and be admitted cold, or hot, or at any desired temperature, so that the register need never be closed.

In the direct-indirect method, if the heating is by steam or hot water, the usual position of the radiators is in the window-breast, and the air is admitted beneath the window-sill; if by stoves, the air is brought in through an air-box placed beneath the floor, one end open to the outer air, the other opening

beneath the stove, which is surrounded by a jacket of tin, or sheet iron, to force the air to ascend along the sides of the hot stove. This method is perhaps the best for country school-houses.

In the direct method the heat is supplied by an open fire or by some kind of stove or radiator, and the fresh air is admitted at some other point or points and is not warmed. In this case the opening for the incoming air should be so placed and shaped that the current will be directed upward toward the ceiling. The simplest mode of doing this is by raising the lower sash of the window four inches, closing the opening thus formed by a board, and allowing the air to come in through the crevice thus formed between the upper and lower sashes. In most houses the fresh-air supply comes in through crevices about the doors and windows, through the cellar from the soil into the halls and stairways, and directly through the walls of the house. Through the outer wall of an ordinary brick house, plastered but not painted or papered, about 7 cubic feet of air to each square yard pass per hour if the room is ten degrees warmer than the outer air. If the wall is damp, or is painted or papered on the inside, very little air can go through it. Stone walls are much less pervious to air than brick ones.

In most dwelling-houses the outlets for foul air are the chimney-flues, and if the room have an open fireplace connected with such a flue, it is in most cases sufficient, even if there be no fire in the fireplace. In hospitals, schools, and other rooms where a number of persons are assembled, special outlet flues should be provided. The movement of the air through the room and in these outlet flues is generally produced by the difference in temperature between the air in the room and that of the outer air. Air expands 1 part in 491 of its volume for each degree Fahrenheit that it is heated, and, if it is free to expand, a cubic foot of warm air is therefore lighter than a cubic foot of cold air. The result of this is that the heavier cold air tends to flow in below and push up the lighter warm air. The pressure thus produced depends on the height of the column of warm air, being equal to this height multiplied by the difference in temperature between the warm and cold air divided by 491, and the velocity, if there is no friction, equals eight times the square root of this. Thus, if the height from the fireplace to the top of the chimney is 20 feet, and the difference in temperature between the air of the room and the outer air is 20° F., the theoretical velocity of the upward flow of the air in the chimney-flue would be $\frac{8\sqrt{20 \times 20}}{491}$, or a little over 7 feet per second. The actual velocity will depend on the amount of friction at the points of entrance of the air into the room and in the outlet flue. If one fourth be deducted for this, the velocity in the above case would be 5.38 feet per second. If the flue were 1 foot square in cross-section, 5.38 cubic feet of air would pass through it every second.

If the air in the flue and room is colder than the external air, it will fall instead of rising; the current will be reversed. It is not desirable to place upcast flues in outer walls, for the air in them is liable to be chilled and the

upward current checked. It is also not desirable to have two or more separate upcast flues in a room, for the cold air is liable to flow down one of them, so that it becomes an inlet instead of an outlet. The velocity of the air in an ordinary smoke or ventilating upcast flue should be about 6 feet per second to produce the best and most economical results. At the top of the flue it is best to have a little greater velocity, say 8 feet per second, to prevent possible interference by the wind. The area of the outlet flues should therefore be about the same as that of the inlet flues, giving from 24 to 36 square inches per head.

The movement of air necessary for ventilation may be produced not only by the ordinary differences of temperature between the exterior and interior of a building, but by wind; by heat specially applied for the purpose, as by steam-pipes, furnaces, gas-jets, etc.; by steam- or water-jets; or by fans and propellers of various kinds moved by machinery. The special mechanical means for forcing air in or drawing it out are only used in mines, tunnels, and large public buildings. Wind is an important aid to ventilation, but is not to be relied upon. In warm weather, when doors and windows can be freely opened, nothing can take its place, but when these are closed it produces its effects either by increasing the inward flow of air through crevices, walls, etc., or by modifying the flow through upcast shafts, its effects depending on the position and shape of the openings at the top of these shafts and on the velocity of the current escaping from them. As a rule, the top of an exit ventilating flue or shaft should be covered in such a way as to prevent the entrance of rain or snow, for if the wall of the flue is damp, much heat is taken up in the evaporation of the water and the ascending current is thus chilled and checked.

To secure good ventilation in a room it is necessary not only to introduce and remove the requisite quantity of air, but to secure a thorough distribution and mixing up of the air in the room, and to do this without causing draughts which will be unpleasant to the inmates. Air has a strong tendency to adhere to surfaces against which a current of it strikes: it does not rebound like a billiard ball from a cushion, but spreads out in a thin sheet on the surface of the wall, roof, ceiling, or floor against which it impinges. When it becomes chilled against the surface of a window, it flows downward in a thin sheet, giving the sensation to one seated by it of a current of cold air leaking in through the sash. If fresh warm air be introduced through registers in the floor, it rises directly to the ceiling, where it spreads out, and gradually descends as it becomes chilled. A living man is usually from twenty to thirty degrees warmer than the air of his room in winter, and therefore acts as a little stove, causing an ascending current of air. The air which he exhales is also warmer than the surrounding air, and rises. It is true that it contains more carbonic acid than the surrounding air, and that carbonic acid is heavier than air of the same temperature, but as diluted and warmed in the breath it is lighter. There is no accumulation of carbonic acid near the floor of an inhabited room. Ventilation dilutes the gases and vapors in a room, but it has not much effect on the suspended particles, including bacteria, except for

a few moments after a dust has been raised. Ordinarily, unless specially filtered, it brings in about as many bacteria as it takes out.

The ventilation of soil pipes is referred to in the section relating to House drainage. To test the sufficiency of the ventilation of a room the determination of the proportion of carbonic acid in the air in different parts of the room, as compared with that in the air as it enters the room, is the best method. To determine the amount of air entering and leaving the room by the special supply and exit flues, an instrument called an anemometer is used to measure the velocity of the current in feet per second, and this velocity, multiplied by the area of the flue or opening, stated in square feet and fractions of a square foot, gives the number of cubic feet passing per second. Most anemometers will not record a velocity of less than 2 feet per second. The direction of currents in the room is determined by toy balloons, by smoke, and by the fumes of nascent muriate of ammonia. So far as the impurities due to respiration and exhalation from the skin are concerned, the normal sense of smell gives a good test, for if no odor is perceived when first entering from the outer air the ventilation is good. Care must be taken not to confuse the sense of discomfort created by an excessively warm and moist air with that due to an excess of organic impurities; a hot moist air may be pure, and a cool air very impure.

Disposal of the Dead.

For the great majority of American communities the best method of disposing of the dead is to bury them in the ground in such a way as to favor their decomposition into gases, water, and soluble salts. No attempt should be made to preserve the bodies, as by embalming, by the use of metallic coffins, etc. Under ordinary circumstances there is no reason to believe that cemeteries are a source of danger to those who live in the vicinity, or that they cause dangerous pollution to water-supplies. Overcrowded and badly-managed cemeteries in the midst of large cities have in times gone by produced nuisance and perhaps disease, but this is not a valid objection to the ordinary methods of burial in rural cemeteries. A cemetery is not a nuisance *per se*, but a legislature has a right to pass laws regulating interment both in private and public burying-grounds, and it has the right to forbid the continued use of any piece of ground for burial purposes and to order the removal of the bodies already buried therein. The arguments in favor of cremation are in the main theoretical, or appeal to the emotions rather than to reason; on the other hand, the chief objections that have been made to cremation, as that it destroys stored force that came from the soil and should be given back to it, or that it destroys the evidence of crime, are also theoretical and of little value. The chief dangers and discomforts which the dead cause to the living occur prior to burial in preserving the bodies and in connection with funerals. In the crowded habitations of the poor the keeping of dead bodies among the living for several days prior to burial causes much discomfort and sometimes danger. Each city should have a public mortuary where the dead can be properly cared for prior to interment. In all cases of death from contagious disease

the funeral should be as private as possible, and there should be no gathering of friends in the infected house. Funeral processions and parades are invariably more or less injurious to all concerned, are a heavy burden on the poor, and in cold or stormy weather are a fruitful cause of disease.

Sanitary Jurisprudence.

Constant yielding to the appetites and desires of the moment cannot be indulged in with safety to the health of the individual, and in like manner in a community a certain amount of personal individual liberty must be surrendered to preserve the health and comfort of the mass of the people. That the State, when it does act, should do so with regard to the interests of the many rather than of individuals is the theoretical rule, but the practical question is, often: To what extent is it best to allow the community to suffer in order not to interfere with individual benefits? and the answer to this differs greatly for different places and at different times.

As a general rule, in the United States, the power to control nuisances and to regulate matters affecting health depends upon what is called the police power of the several States, which is exerted either through specific enactments by State legislatures, forming what is called statute law; through rules and regulations framed by municipal or other local authority in accordance with State legislation; or through the interpretation by the courts of statutes or of prevailing customs, forming what is called the common law.

That part of the common law which relates to public hygiene is summed up in what is called the law of nuisance as set forth in the decisions of the courts, which are based on the principle that a man must so use his liberty of action and his property as not to cause injury to the health of others. The most difficult and doubtful point in the application of this principle is to determine the cases in which an individual is entitled to compensation for restriction of his liberty for the public benefit.

Under what is called the "right of eminent domain" the government may demand the services of any of its citizens or may take private property for the public good, but it must furnish a reasonable compensation for the service or property thus taken. But under what is called the police power it is usually held that certain uses of property may be forbidden, or certain services required by the State, without entitling the person whose liberty is thus restricted to compensation therefor. This is the case with regard to laws prohibiting the storage of gunpowder near habitations or highways, or forbidding the erection of wooden buildings in certain parts of cities, or requiring that the plumbing in a house shall be arranged in a certain way, etc. "In abating a nuisance property may be destroyed and the owner deprived of it without trial, without notice, and without compensation. Such destruction for the public safety or health is not a taking of private property for public use without compensation or due process of law in the sense of the Constitution. It is simply the prevention of its noxious and unlawful use, and depends upon the principles that every man must so use his property as not to injure his

neighbor, and that the safety of the public is the paramount law. These principles are legal maxims or axioms essential to the existence of regulated society. Written constitutions presuppose them, are subordinate to them, and cannot set them aside." (*New Jersey Repts.*, 8 C. E., Green, p. 255).

The refusal of compensation for loss to an individual caused by action of the State under the police power is sometimes in conflict with the important legal principle that if a man is compelled to give up to the public his time, his labor, or his property, or any value therein, he has a right to demand from the public compensation therefor.

Under the common law there is much difficulty in many cases in deciding as to what amount or degree of danger or discomfort constitutes a nuisance: one court is not bound by the decisions of another one, and different judges have very different ideas as to the relative importance of general business interests, of public health, and individual rights, and decide according to what they deem expedient for that particular place and time.

For these and other reasons connected with the uncertainty and cost of legal proceedings under the common law it is necessary to have positive, definite statute law to secure as clear and precise definitions as possible of what is forbidden and of what is to be done to prevent or to get rid of nuisances. In connection with such regulations it is usual to provide some special means for their enforcement in the shape of a health department, board of health, or health officer. The health department of a city, if it is to be really efficient, should have certain quasi legislative and judicial as well as administrative powers, and should have charge of the registration of vital statistics. Much of the information which it requires to enable it to do its work properly must be obtained directly or indirectly from medical men, and hence the organization, powers, and duties of such departments are of special interest to physicians.

A municipal corporation in making sanitary regulations must not exceed the proper and necessary powers delegated to it by the State legislature; but the State may expressly authorize the city to pass local laws and to be the exclusive judge of the necessity for these laws. Under such authority from the State a city may regulate the size, plans, ventilation, and plumbing of the habitations of its citizens, even to the extent of making it impossible for persons of limited means to find shelter within its boundaries. Tenement-house regulations may produce this result, and it is a question of expediency as to where the line shall be drawn. A city cannot be kept healthy unless a reasonable standard of what is fit for human beings to live in is fixed and maintained; there must be a limit to the lowest kind of habitation which it is permissible to furnish, just as there is for unwholesome food. In each case the liberty of the seller and the buyer is to a certain extent interfered with, but neither has any legal claim on the community for compensation on that account.

The question as to whether a State, or a city acting by authority of the State, can by statute compel physicians to report to the local authorities the

names and residences of all persons afflicted with contagious or infectious disease who are their patients, under penalty and without compensation, has been decided in the affirmative in 1887,[1] by the Supreme Court of Errors of the State of Connecticut, but the wisdom and justice of this decision are very questionable, especially as applied to States having no regulations as to the qualifications of persons permitted to practise as physicions.

[1] See the State *vs.* N. E. Wordin, *Twelfth Annual Report Connecticut State Board of Health*, 1890, p. 249.

EPHEMERAL FEVER AND SIMPLE CONTINUED FEVER.

By WILLIAM PEPPER.

Definition, Synonyms.—These terms are used to describe forms of febrile disturbance which, so far as can be determined, are not due to specific infection or to any inflammatory lesion. They are unattended with definite lesions or with characteristic eruptions; their symptoms are usually mild and their termination favorable. When the affection lasts only from twenty-four to seventy-two hours, it is called ephemeral fever, while the duration of simple continued fever or febricula is more commonly from seven to twelve days, though it may not exceed four or five.

History.—With the older writers these forms of fever occupied a prominent position. As diagnosis has become more accurate and our knowledge of the variations in the course of the infectious fevers more minute, many cases which would formerly have been regarded as ephemera (ephemeral fever) or synochus simplex (simple continued fever) are found to be abortive forms of some infectious disease or to be dependent on a latent local lesion. All careful observers will, however, agree that, after making full allowance for these sources of error, cases are met with not infrequently which must still be referred to one or the other of the above headings. It must be admitted that such reference is always made with the feeling that a more refined diagnosis than is yet possible might show in any such case some mild infection or some obscure irritation.

Etiology.—Children and adolescents are much more liable to these febrile attacks than older persons, yet cases are met with in adults or even in advanced age. It is, indeed, a matter of common observation that certain individuals, of whatever age, exhibit the phenomena of fever with extreme facility from the operation of apparently trifling causes. The heat-regulating mechanism in these subjects is so readily deranged that it constitutes a strong susceptibility, amounting almost to an idiosyncrasy. In such persons it is probable that areas of local irritation too slight and too limited to produce recognizable symptoms of functional disturbance may suffice to cause a mild fever of short duration.

It is often suggested, in studying closely the symptoms of the more important fevers, that this element of individual susceptibility plays its part there

also, and helps to explain the wide variation in the degree of pyrexia induced in different cases of apparently equal degree of infection.

Extreme mental or bodily fatigue, great excitement or anxiety, undue exposure to the direct rays of the sun, may be reckoned among the well-ascertained causes of ephemeral and simple continued fever. The most severe results of these causes are observed in tropical climates, and especially in the case of young and robust persons who have not yet become fully acclimated. Indeed, it is probable that some of these cases are rather to be regarded as mild cases of insolation or thermic fever. The susceptibility of the system is increased by such influences, the power of the heat-controlling centres is lowered, there is notable increase in the amount of the results of tissue-waste to be disposed of, so that the causation of pyrexia even here is more or less complex.

Again, it is apparent that different individuals present widely different degrees of liability to derangement of the chemical processes concerned in digestion and assimilation, and that in some subjects the development of ptomaïnes with strong pyrogenic properties is readily induced. For instance, I have observed several attacks of violent fever, of short duration, but with a temperature of from 104° to 106°, produced in a man past middle age by the ingestion on each occasion of a moderate amount of cheese and beer. It is obvious that in all such cases the existence of more or less gastric irritation is to be counted upon, and that this is responsible for a share of the febrile disturbance.

In some individuals the susceptibility to noxious vapors, as sewer-gas or the emanations from putrescent organic matter, is so great that fever may be induced by exposure to their influence.

Symptomatology.—The onset in ephemeral fever is abrupt and is not preceded by any prodromes. There is rarely a chill; in nervous children a convulsion may occur at the beginning of the attack. The fever rises rapidly to 101°, 103°, or even 105° F. I have seen it reach 106° within eighteen hours after the onset. It is attended with headache and dulness or marked hebetude. Restlessness, mild delirium, and irregular muscular twitching may occur, especially in children. The face is flushed, the skin hot and dry, the pulse rapid, full, and tense. The tongue is coated white; the tonsils are occasionally reddened and somewhat swollen. Appetite is lost; nausea and vomiting may occur. The bowels are costive, and typical febrile urine is secreted in scant amount. There is no characteristic eruption, but in children with delicate skin and vigorous circulation there may be a marked erythematous flushing of the surface. Herpes often appears on the lips. At the close of twenty-four, forty-eight, or seventy-two hours the fever terminates by rapid subsidence or by abrupt crisis. Free perspiration, diarrhœa, or copious urination marks the defervescence. The symptoms clear away promptly, and convalescence is rapidly completed. In many instances the whole process is much milder than as above sketched.

Simple continued fever may be less abrupt in its onset, and for the first two

or three days the fever may rise gradually till it reaches 102.5° or 103.5°. The ascent is, however, more rapid as a rule than in typhoid. The nervous symptoms are mild. Sleep is disturbed and slight nocturnal wandering may occur. Headache and some degree of dulness are present. Catarrhal symptoms are not marked. Dulness of hearing is infrequent. Occasional cough and a few bronchial râles may be present. The pulse-respiration ratio is fairly preserved, and may be represented by 96 : 24 : 103° in ordinary cases. The heart's action retains its tone, and the pulse, at first full and bounding, merely grows softer, but rarely feeble or rapid. The tongue remains moist, though heavily coated. Appetite is much impaired, and thirst is rarely marked. Vomiting rarely occurs unless provoked. The abdomen is but slightly if at all meteoric; the bowels are commonly torpid, though if intestinal catarrh coexists a tendency to looseness may be present. The spleen is but little if any enlarged. The urine is moderately febrile in character, is often passed quite freely, and contains a trace of albumin only in a small proportion of cases. Slight epistaxis occasionally occurs. There is no characteristic eruption. Sudamina are common, as is also an eruption of herpes on the lips and face. Pale bluish or slate-colored spots, several lines in diameter, not elevated above the surface and not modified by pressure, are sometimes seen, but have no special diagnostic value, as they may be observed in other diseases.

The fever usually continues ten or twelve days. It may, however, end much sooner, as in six or even four days, or much more rarely it may be protracted to fourteen or fifteen days. The daily maximum is usually in the evening; an inverted type with morning maxima is, however, not rare, especially in children. Hyperpyrexia is very uncommon, and the average maxima are 102.5° to 103.5°. The daily range of temperature may be so marked—2.5° or even 3°—as to make the case resemble a malarial remittent. Some cases of so-called infantile remittent fever are undoubtedly of simple continued type, while others are abortive typhoid. Defervescence is not so abrupt as in ephemeral fever, and yet is commonly more rapid than in typhoid. The temperature often becomes subnormal for a day or two, with abnormally slow pulse. Critical discharges as of sweat, or from the bowels, or of urine heavily loaded with urates, or in the form of epistaxis, or of hæmorrhage from the bowels or the uterus, may attend the defervescence.

More severe grades of simple continued fever are sometimes met with when the range of temperature is much higher and the nervous symptoms are more pronounced. This type of the disease is especially apt to occur in the tropics, where it has long been known under the name of ardent continued fever. Even in the more severe type, as met with here, death is of rare occurrence, but the violent cases observed by Murchison and others in India, and especially among robust young European new-comers, not infrequently ran into a state of profound stupor with heart failure, and terminated in death by the sixth or eighth day or even earlier.

Diagnosis.—Ephemeral fever in children often simulates scarlatina in the

abruptness of its onset and the sudden development of high fever, with vomiting and restlessness. But, although there is a vivid febrile flush, the characteristic eruption of scarlatina is wanting, and there is no sore throat or swelling of the glands at the angles of the lower jaw. Great anxiety is, however, often caused for twelve or twenty-four hours.

Both in ephemeral and simple continued fever it is necessary to exclude acute gastric or gastro-intestinal catarrh. A certain amount of functional disturbance, anorexia, nausea, even vomiting and diarrhœa, may attend these types of fever; but a careful consideration of the circumstances preceding the attack, of the proportion between the degree of fever and of gastro-intestinal disturbance, and of the effect of remedies to allay the mucous irritation, will lead to a correct diagnosis.

It is of course necessary, before pronouncing a diagnosis of ephemeral or simple continued fever, to exclude by careful examination the existence of any local inflammatory affections, as of the kidney, lungs, pleuræ, or heart. The fact that in children especially, but occasionally also in older subjects, rheumatic fever may occur without arthritis, and that in this form of the disease endocarditis is very likely to be present, warns us that such cases may be mistaken readily for ephemeral or simple continued fever. A child of ten years who seems flushed and drooping, with slight hoarseness and occasional cough, but still about the room and without definite complaint, is found to have a temperature of 104.5° F. and an acute mitral murmur. Prompt and energetic treatment is followed by abrupt fall of temperature on the third day, but the endocarditis requires a month before complete cure is effected. Similar cases are not rare, and their true nature is often overlooked. The affection is regarded as an ephemeral fever, and only long afterward does the detection of organic heart disease show that it has been an acute rheumatic fever with latent endocarditis.

Simple continued fever must be carefully distinguished from malarial remittent fever and from typhoid. When the time and place of the attack and the character of the fever suggest a malarial nature, the absence of marked enlargement of the spleen, the failure of full doses of quinine to produce decided effect on the course of the case, and the failure to detect the malarial organisms in the blood, will dispel the suspicion. Herpes is about equally frequent in the two affections.

In the article on TYPHOID FEVER attention is urged to the cases of an abortive type which may resemble greatly simple continued fever. In ordinary cases also it may be for several days difficult or impossible to decide which disease exists. The prodromes are less marked in simple continued fever; the temperature is apt to rise more rapidly; the spleen does not enlarge so decidedly; the Ehrlich reaction is likely to be absent; epistaxis and looseness of the bowels are more rare; and no characteristic eruption makes its appearance, while, on the other hand, herpes is of far more frequent occurrence. It must be remembered, however, that in some cases of typhoid the eruption is postponed or even absent, and that the abdominal symptoms

may be but slight; so that the differential diagnosis may remain in doubt until the somewhat abrupt defervescence at the tenth or twelfth day leaves it still uncertain if the case has been one of mild irregular typhoid or of simple continued fever.

Prognosis.—The prognosis in these forms of fever is uniformly favorable in this country. It is only when a case of unusual severity occurs in a very frail subject, and especially if in infancy or old age, that a fatal result need be feared. In the tropics, where the affection assumes a much more violent type, death not rarely occurs, or if life is spared convalescence may be protracted, and serious sequelæ, chiefly affecting the nervous system, may linger.

There are no characteristic anatomical lesions. When death occurs, the only changes are those of intense internal congestion with serous effusions.

Treatment.—Absolute rest in bed must be insisted upon. This is essential, not only for the more speedy cure of simple fever, but to avoid all chance of damage in case an obscure local inflammatory lesion or an irregular typhoid fever is developing. The diet is to be carefully restricted and exclusively liquid. Water may be allowed freely as called for by thirst, unless marked irritability of the stomach exists. In that event small pieces of ice may be swallowed; or small amounts of carbonated water, of equal parts of milk and lime-water, of liquid peptonoids, may be relied upon. A short course of fractional doses of calomel, gr. $\frac{1}{10}$, every two hours for one or two days, followed by a mild saline if no loose movement occur, or repeated small doses of liquid effervescing citrate of magnesium, may be given with advantage at the outset. Later it is usually preferable to overcome constipation, if it should exist, by laxative enemas or suppositories.

In cases where the symptoms suggest the existence of malaria, and pending the examination of the blood, full antiperiodic doses of quinine should be given. But except for this purpose it is not indicated in these simple fevers unless asthenic symptoms supervene. Small and frequently repeated doses of aconite, alone or with spirit of nitrous ether, effervescing or neutral mixture, or solution of acetate of ammonium, may suffice to moderate the fever. Spongings of the surface with cool water, or more vigorous hydrotherapy, as fully described in the article on TYPHOID FEVER, should be used according to the grade of the pyrexia. In the ardent continued fever of the tropics the early and systematic use of cold baths must evidently be insisted upon, and cardiac sedatives may be required in addition. Headache and restlessness may be relieved by cold applications to the head, by a hot mustard foot-bath or a sinapism to the nucha, and, if necessary, by proper doses of chloral and potassium bromide. A few doses of antipyrine, gr. 5, or of phenacetin, gr. 3, may exert a happy febrifuge and tranquillizing effect.

When the early symptoms suggest the possibility of typhoid fever or the presence of gastro-intestinal catarrh, the use of silver nitrate or some other of the remedies recommended for their surface action or antiseptic properties (see TYPHOID FEVER), should be promptly instituted, and laxatives should

be avoided. Stimulants are rarely required save in debilitated subjects or in cases of specially adynamic type.

Convalescence is usually prompt and uncomplicated: a mild tonic may be given with advantage, and careful attention to personal hygiene should be insisted on.

TYPHOID FEVER.

By WILLIAM PEPPER.

Definition.—Typhoid fever is a specific, infectious febrile disorder, sporadic or epidemic, often communicated by the contagium from the stools, characterized by lesions chiefly of the intestinal and mesenteric glands and the spleen, with the constant presence in them of the bacillus of Eberth. The disease is marked clinically by a variable febrile course which lasts three or four weeks, a rose-colored macular eruption, and cerebral, pulmonary, and abdominal symptoms.

The general tendency is to restoration to health after slow convalescence, but relapses are not infrequent; there are numerous complications and sequelæ, and death may occur from various causes.

Synonyms.—Probably a hundred names have been applied to this disease both before and since the establishment of its essential difference from typhus and other fevers. No more descriptive term has been used than that given it by Huxham, "slow nervous fever," but this has become obsolete. The title "typhus abdominalis," so generally employed by German writers, is objectionable, as implying a relationship with typhus fever (called "typhus exanthematicus" by way of contradistinction) which the accurate observation of recent years has completely disproved. "Enteric fever" is likewise objectionable, because it implies that the intestinal lesion is the basis of the disease, instead of only one of its localizations. In spite of all objections to the name "typhoid fever," on account of its vagueness and of its seemingly indicating a resemblance to typhus fever, this term, given to it by Louis in 1829, seems the most appropriate one, and has passed into such general use that it is desirable that it should be adopted uniformly.

History.—It has been claimed by Murchison and other writers that typhoid fever was known to Hippocrates, and that Galen described it under the title of "hemitritœus." Nothing definite, however, is heard of it before the seventeenth century, when Spigelius seems to have encountered the affection, and to have observed in several post-mortem examinations the characteristic intestinal lesions. Bartholin, Willis, Panarolus, Baglivi, Hoffman, and Sydenham also appear to have been acquainted with typhoid fever. In the next century are to be noted the writings of Huxham, Gilchrist, Manningham, Lancisci, Morgagni, and many others, which clearly describe either the symptoms or lesions now known to be characteristic of the disease. There was, however, at that time no recognition of it as a distinct entity or as other than a mere variety

of continued fever. Even the close association between the characteristic symptoms and the intestinal lesions does not appear to have been pointed out by any one before Bretonneau, who began his observations in 1818. Some years later Louis added greatly to a proper understanding of the affection, but even yet no one had determined sharply the points of distinction between typhoid and typhus fevers.

To Gerhard and Pennock of Philadelphia, writing in 1837, we are indebted for a thorough establishment of the separate existence of typhoid fever and for clearly distinguishing between the two affections. As a result, the individuality and true nature of typhoid fever were appreciated in America sooner than in either France or England. In Germany, it is true, Hildenbrand in 1810 showed that there was a difference between this disease and typhus, but regarded them as varieties simply, without establishing their independence, and for years after him German writers shared this view. Not until 1849 did Jenner definitely demonstrate in England their non-identity, though Stewart in Scotland wrote of it in 1840, and the French observers were beginning to grasp the fact at about the same time.

Since 1850 the facts in the case have been fully recognized the world over, and our exact knowledge has been increased by numerous valuable contributions from various countries.

Etiology.—The study of the causation of typhoid fever is of much practical and scientific importance. There are certain predisposing causes which exert a powerful influence. It is one of the most widely distributed of infectious diseases, and is, in fact, ubiquitous. Although it may occur in all climates, it is especially prevalent in the temperate zone; indeed, it may be stated to be constantly present there in greater or less degree. Yet it cannot be said that geographical locality of itself exercises any influence upon the frequency of its occurrence. The disease is certainly not so unusual in the tropics as was formerly supposed, and it is quite common in Iceland, Norway, Sweden, and Finland. The great vitality which its specific poison possesses, and the manner in which this is diffused by running water, explain in large part the extraordinarily wide dissemination of the affection.

Season exerts a marked influence upon the frequency of the occurrence of typhoid fever. Although met with in winter and spring, it is especially frequent throughout the late summer and autumn months. According to Murchison, out of 5988 cases seen in the London Fever Hospital during twenty-three years, 2461 occurred in autumn, 1490 in summer, 1278 in winter, and 759 in spring. Bartlett shows that of 645 cases admitted to the Lowell Hospital during a period of eight years, 250 occurred in autumn, 163 in summer, 130 in winter, and 102 in spring; 98 cases being reported in October alone, 92 in September, and 86 in August, while 48 was the greatest total number in any one of the other months of the year. According to Osler, over 50 per cent. of the 1889 cases in the Montreal General Hospital and of the 1381 cases in the Toronto General Hospital were admitted in the autumn months. Elaborate statistical tables of epidemics in various parts of the world, prepared by

Hirsch, prove the same tendency to the occurrence of typhoid fever in the late summer and the autumn. In consequence of this prevalence in the fall the disease early received in many places the name of "autumnal fever."

Typhoid fever is apt to prevail after hot and dry summers. Pettenkofer and Buhl showed that the disease was more common when the ground-water was low, which then allowed the germs to develop rapidly in the soil and filter through into surface wells. The explanation of the method of the action of low water-level, as given by Buchanan and Liebermeister, is that the lower the water is, the greater amount of solid matter must be suspended in it. Should there, then, be germs in the soil, the water will contain them in larger proportion, and be to that degree more poisonous. It is probable also that when the hot, dry seasons break up and are followed by damp changeable weather, the resisting power of the community is lowered, and, further, that catarrhal conditions, which favor the entrance of the poison from the intestine into the general system, are especially liable to occur.

It must be clearly recognized, however, that the disease does not always have any connection with dry weather and a low water-level, and that epidemics often occur without reference to the state of the ground-water. This is indeed but what would inevitably result from the varied manner in which contamination of water- and milk-supply may occur.

Baumgarten suggests that the dust of dry seasons may disseminate the germs; and the suggestion adds to the probability that in some cases the bacilli may enter with the inspired air.

The configuration of the ground and the elevation above the sea are apparently entirely without influence in the production of typhoid fever.

There is no reason to believe that sex exerts any distinct influence. However, as lads and young men are most apt to congregate in cities, where the cause of typhoid fever is most constantly and powerfully present, they naturally furnish a larger proportion of the cases which form the basis for statistics.

The disease occurs at all periods of life, but particularly between the ages of fifteen and twenty-five years, becoming progressively less frequent after the age of thirty-five. This is probably due, in large measure, to the fact that those who are especially susceptible to the poison have already suffered from the disease at an earlier age, or perhaps, with even more probability, that individuals of riper years have already become immune through constant exposure to the germs. Typhoid fever is not infrequent between the ages of thirty-five and fifty years, and is occasionally met with even up to extreme old age. It is far more general in early life than is usually recognized. Murchison reports its occurrence in an infant six months of age, and Charcellay reports two cases in infants but a few days old, while several observers have discovered evidences of the disease in the fœtus. I have observed several cases during the first year of life, and have seen patients recover from well-marked attacks at the age of seventy-two and even seventy-five years. Hamernyk records a case in a patient aged ninety years.

It is important to note the difference in individual susceptibility to the poison of the disease. It is altogether probable that few inhabitants of large cities have not had, more or less frequently, some portion of the poison pass through their digestive tract. It is a matter of common observation that young men and women who have recently moved into cities, and who are subjected to the influences of change of residence, of habits, and of diet, are specially prone to the affection. Louis found that of 129 cases, 73 had not resided in Paris over ten months, and 102 not over twenty months. The same influence of recent residence has been observed by Murchison and others. (It has also been noticed that those who are apparently not susceptible to the disease may lose this immunity by changing their residence, and consequently may be attacked by it on returning to the house or locality previously occupied, and in which they had formerly been constantly exposed without danger.) Then, too, I am familiar with instances which indicate that certain families may exhibit a high degree of susceptibility, or the reverse, in successive generations.

A point which deserves careful consideration is the probability that the presence or absence of a catarrhal state of the intestinal mucous membrane at the time of the admission of the poison to the bowel may play an important part in determining the occurrence or non-occurrence of infection by the germ. In this connection it is interesting to note the frequency with which catarrh of the tonsils or pharynx precedes diphtheritic infection, or a slight fissure or abrasion of the skin an attack of facial erysipelas. I have seen more than one instance in which a patient, confined to bed with what could only be regarded as a simple, non-specific gastro-intestinal catarrh, has apparently received the infection of typhoid fever from contaminated water or milk, and developed the symptoms of a well-marked attack of the disease. It is also possible that some of the relapses which are so frequent in this disease are due to the admission of small portions of fresh poison, which, in the susceptible catarrhal state of the mucous membrane of the bowel, can penetrate the epithelial lining and reach the lymphoid tissue with especial ease.

Apart from the predisposing influence of this intestinal catarrh, the state of the individual's health has little if any influence on the development of typhoid fever. It is indeed possible that depressing influences, such as overwork, prolonged anxiety, home-sickness, may reduce the tone and resisting power of the system and render it more susceptible to the poison of this as of some other infectious diseases. On the other hand, overcrowding, filth, destitution, and intemperance seem to be without special predisposing influence.

The consideration of the exciting causes of typhoid fever is wholly controlled by the fact that a special micro-organism, observed and described by Eberth and by Klebs, and after them by Koch, Gaffky, Arthaud, Pfeiffer, Friedländer, and many others, has been shown to be constantly associated with the disease. The organism is a small bacillus, of about one-third the diameter of a red blood-corpuscle in length, one-third as thick as long, rounded at the extremities, and sometimes exhibiting at one or both ends, or, according

to Arthaud, in the centre, a shining rounded body, possibly a spore, but possibly also only a degenerative alteration of the protoplasm. It occurs singly or in filaments composed of a number of bacilli joined end to end. The descriptions of it vary considerably, owing to the fact that the bacillus itself varies with the culture medium. All observers, however, agree on its motility as a characteristic feature. Löffler was able to demonstrate that this motion was due to the presence of a vibratile cilium. The bacilli are found chiefly in the spleen, intestinal and mesenteric glands, and liver. Pfeiffer was the first to discover them in the stools. They are, however, rarely detected before the period of actual ulceration, when they become much more numerous. According to Chantemesse and Widal, they exist in great numbers in the passages from the tenth to the sixteenth or seventeenth day, but disappear, as a rule, after the twenty-second day. They have been observed in the kidney, and Neumann and Karlinski found them in the urine. They have been discovered in the expectoration in certain cases, and also, though rarely, in the blood. Rütimeyer reports their presence in blood taken from the rose-colored spots. They have also been reported as occurring occasionally in many other parts of the body, as in the meninges of the brain and of the spinal cord, the substance of the cord, the heart-muscle, lungs, and testicle. They have been found, further, in pus from an encapsulated peritonitic abscess, in periosteal abscesses, in empyema, and in serous pleural effusion.

Perhaps in this connection it can be best recorded that Widal and Chantemesse found bacilli in the placenta from a woman who aborted at the fourth month on the twelfth day of an attack of typhoid fever; Neuhaus in the liver and spleen of the fœtus; Eberth in the fœtal blood from various parts of the body; and P. Ernst in the spleen and the blood from the heart in the case of a child prematurely born of a mother with typhoid fever, which died suddenly on the fourth day of life. The mother had received an injury some days before labor which had probably produced a lesion of the placenta. The experiments of Fränkel upon guinea-pigs led him to believe that the bacilli could not be transmitted from mother to fœtus unless there had been an injury to the placenta; and Eberth holds much the same view.

The bacilli of Eberth will produce pure cultures on potato, gelatin, agar, and in blood-serum and bouillon. They grow rapidly in sterilized milk, and become quite large. They have been found to live in milk for thirty-five days, and in butter for twenty-one days. In fact, as Heim has shown, there is scarcely any article of diet which does not form an excellent culture medium for this bacillus.

Very few of the cultures are characteristic, that on the potato being the most so. Even this, however, is so like that of the colon bacillus that much confusion has arisen and still exists, especially as this bacillus, like that of Eberth, penetrates at times into different tissues of the body. There is no doubt that the colon bacillus has repeatedly been mistaken by able observers for the typhoid bacillus. The uncertainty has indeed gone so far that Vaughn concludes, from an elaborate series of experiments, that the Eberth germ is not

a specific micro-organism, but a modified form of any one of a number of other closely-related germs. In this opinion, which seems improbable in the light of what we know of other infectious diseases, he is upheld by some other investigators.

It has been claimed that successful inoculation experiments have been made, but this matter does not appear to be positively determined as yet. The typhoid bacilli unfortunately possess tenacious vitality. They have been known to remain active and virulent in parts of the organism for as long as fifteen months after the convalescence of the patient. Outside of the body it seems undoubted that they may retain their vitality for weeks in water, and may even increase in number, while in illy-drained soil they are capable of multiplication and growth, and thus continue to live indefinitely. Although they are killed by exposure for twenty minutes to moist heat, they are not killed by heavy frost. Prudden has shown that they may retain their vitality in ice for months, and Seitz that they will grow at a temperature of 37.4° F. They develop rapidly in milk, without altering its appearance in any respect. It would appear that they will continue to live in fæces for extraordinarily long periods. Magnant reports a small epidemic of fourteen cases which he could ascribe only to the careless emptying of a privy-well into which the stools of a typhoid-fever patient had been emptied a year before. Uffelmann says that in one instance under his observation the bacilli had certainly remained alive and virulent for over a year. He made some interesting experiments by adding pure cultures to fæces under different conditions, and found the bacilli still living after four months. Karlinski's experiments, while indicating a shorter duration of life than this for the bacilli, still prove their great hold upon it. It has been found, too, by Grancher and Deschamps that typhoid germs, placed upon the surface of frequently moistened ground, will penetrate to the depth of fifty centimetres, and there retain their life for five and a half months.

Just how long the bacilli may live in ordinary water is not positively known. Under favorable circumstances they may persist twenty to thirty days. Hochstetter even found them live twelve days in a syphon of seltzer-water. It is certain that they will live a shorter time in running water than in cisterns or reservoirs.

Sunlight proves quite destructive to the germs. Janowsky found that cultures ceased to develop after four to eight hours' exposure to light.

The bacilli enter the system by the way of the intestinal mucous membrane. This is certainly true in the vast majority of cases. That they may occasionally enter by way of the respiratory tract has been asserted, but never proved. That they may be transmitted by way of the placenta from mother to fœtus has already been stated.

As to the exact mode of action of the bacilli after their admission to the intestine, further investigations are needed. They may possibly multiply in the intestinal contents under favorable circumstances. But very probably also they immediately penetrate the mucous membrane and lodge in the lymphatic tissue

of the bowel, as well as in the mesenteric and other lymphatic glands and in the spleen and liver. Here they grow at the expense of the tissue and produce necrosis. During their growth they develop certain toxic agents, Brieger describing a ptomaïne (typhotoxicon), and later, with Fränkel, a toxalbumin; and Vaughn a ptomaïne which produces vomiting, purging, and rise of temperature in dogs. It is probable that the constitutional symptoms of the disease are the result of the action on the system of these or analogous toxic products.

Typhoid fever is not contagious in the ordinary sense of the term. There are no exhalations from the skin or lungs which can impart the disease. The infectious product is contained in the discharges from the bowels, and, more rarely, in the matters vomited or expectorated. It must be admitted that those who handle these discharges or the linen soiled by them may in this way acquire the disease. A very striking instance of this has been reported in which the fever prevailed extensively for twelve years in one of two German artillery barracks, but very few cases occurring in the other. Finally, it was found that the linings of the trousers of almost all of the soldiers were soiled with dried fæcal matter, and that this clothing, thus previously contaminated, had been used by the men who were later attacked. Thorough disinfection of the clothing was now employed, and from that time on no more cases developed. So, too, if the alvine discharges are placed where they can dry and the germs become diffused through the air, it is probable that they may enter the mouth and be swallowed with the saliva. As already stated, it has been suggested that the increased amount of dust in the atmosphere after hot, dry summers may be an additional source of occasional infection. Undoubtedly, however, it is chiefly by the germs gaining entrance directly to the flowing streams of water or soaking through the ground and entering sources of springs, and thus contaminating water used for drinking purposes, that the disease spreads. So many outbreaks have been studied critically and traced to this cause that it is needless to do more than refer to the instances recorded by Murchison or by Hutchinson (Pepper's *System of Medicine*, vol. i. p. 250, *et seq.*). A very interesting observation is that made by Mosny, that the mortality from typhoid fever in Vienna diminished from 1.2 per 1000 to 0.11 per 1000 after the introduction of spring water; but that after the water of the Danube was again introduced, though temporarily, an epidemic broke out, which was localized in those parts of the city supplied by this water. Very similar observations have been made on the effect in Paris of temporary employment of the river-water for drinking purposes. As further instances may be mentioned the existence in 1887 of typhoid fever in towns along the Ohio River for a distance of over eight hundred miles, and the discovery by Rushford and Cameron of the bacilli in the water-supply; as also the epidemics reported by Brouardel, Passerat, Vaughn and Novy, and Chapin, in all of which bacilli were found in the water. In the last-mentioned both Prudden and Ernst found them in the water-filters of the houses in which the disease had appeared.

One of the most remarkable epidemics which has ever been reported is that which occurred at Plymouth, Pennsylvania, in 1885, and which was carefully studied by L. H. Taylor. In this instance a mountain-stream which supplied a population of about 8000 with drinking-water became infected by the entrance of typhoid germs from a single patient living close to its edge, miles away from the town itself. As a result more than 1000 cases developed, at the rate of 50 to 100 a day, and nearly 100 persons died. Another, though small, epidemic has quite recently been carefully studied by Seneca Egbert of the Laboratory of Hygiene, University of Pennsylvania. In this a small manufacturing village of about sixty houses was severely infected, the infection arising from a single case brought ill to one of the houses, and spreading by the soaking into the sandy, sloping soil of the fæcal matter, and the contamination in this way of the various wells from which the drinking-water was obtained. About 50 cases occurred, of whom quite a number died.

It is by this contamination of the water-supply that many virulent epidemics in boarding-schools, hotels, and public institutions are to be explained.

Infected milk is also a frequent mode of conveyance of the poison. The milk may become polluted by the water with which it has been diluted or which has been used to cleanse the cans, or the germs may be introduced directly into the milk from the hands of the milker, soiled with the discharges of a typhoid-fever patient whom he or she is engaged in nursing. The latter is evidently a less common method of infection. Instructive instances of epidemics due to infected milk have been reported by Murchison, Cameron, and Ballard. More recently Almquist reported an epidemic in Sweden where 104 cases with 11 deaths occurred among persons all of whom received milk which was in all probability contaminated. Another milk-epidemic is recorded by H. E. Smith as occurring at Waterbury, Conn., and Littlejohn published the account of an epidemic of 63 cases traceable only to the milk supplied from one dairy. Numerous other instances of infection from this source have been recently reported. Dr. L. H. Taylor of Wilkes-Barre, Pa., has favored me with the notes, as yet unpublished, of such an epidemic occurring under his observation. Quite a number of cases occurred in this epidemic, but only in a limited portion of the town. A careful investigation showed that the disease could not be traced to the water-supply, which was exceptionally pure. Further study revealed the fact that the greater number of the patients had received milk regularly from a certain farm, that a number of retailers of milk in the neighborhood had procured milk from this source, and that a popular druggist, who dispensed milk-shakes to the inhabitants of that part of the borough, also bought his milk from the farm. It was discovered also that a number of persons living upon the farm had been sick with typhoid fever, and it seemed beyond question that this arose from using the water from a well on the place which examination showed was impure. This same water was constantly employed to wash and cool the cans; and there could hardly be

a doubt that it was by the milk, contaminated in this way, that the epidemic was brought about.

Upon the whole, the evidence does not seem satisfactory in support of Pettenkofer's view that the typhoid germ, as discharged from the patient's body, is not in an active state, but must remain in the soil and undergo certain changes before becoming capable of originating the disease. If time is required for any such changes to take place in the germ, it is certainly very short in many instances.

There are certain reports of outbreaks of typhoid fever which were considered to be due to the use of poisoned meat. Cayley has collected a number of instances of this nature. Careful study of these cases seems to show, however, that the possibility of the poison having been introduced in the usual methods cannot be excluded. It is true also that there are numbers of instances recorded in which typhoid fever has arisen sporadically, perhaps in sparsely-settled regions, apparently without any conceivable means of infection of the patient with a typhoid germ. Metcalf reports such an instance occurring on an island in the Pacific Ocean, where a patient fell ill with typhoid fever, although there had been no occurrence of the disease for certainly fifteen months, and although no possibility of infection even from this case could be discovered. Numerous epidemics, too, have occurred in which the disease could in no way be traced to any outside source. A conspicuous instance occurred recently at the military academy at Chester, Pa., where 14 cases of typhoid fever developed among 132 students. The cases I saw with Dr. Ulrich, the physician in charge, were of very grave type. There were 5 deaths. The epidemic was investigated with extreme care and thoroughness by Dr. John S. Billings, who discoverd no source for the infection. Such instances as these have led a number of writers of note, with Murchison especially prominent among them, to argue that cases may develop independently of pre-existing typhoid fever. In the absence of more intimate knowledge of the life-history of the Eberth bacillus it seems unwise to try to pronounce final judgment on this point. It has been suggested that this bacillus, possibly in an imperfect state of development, is widely diffused in nature without reference to cases of typhoid fever. Coming in contact with the results of the decomposition of organic matter, it develops actively, and acquires a pathogenic power which enables it when introduced to a susceptible system to produce typhoid fever. It is needless to repeat that the discharges from even a single case of the disease may contain so many and such virulent bacilli as to be able to infect an extensive water-course or spread the disease to hundreds. In the face of an extraordinary diffusibility in damp soil, by running water, and by milk it is safer at present to say that, although the origin of typhoid without direct connection with a pre-existing case is possible, the evidence at hand does not justify us in asserting that it occurs.

Morbid Anatomy.—The lesions of typhoid fever are generally divided into two groups: those characteristic of the disease, and those which may be

regarded as secondary changes, chiefly due to the effect upon the tissues of the constitutional infection and the long-continued fever.

I. The characteristic post-mortem changes are seen in the lymphatic structures of the intestine, in the mesenteric and other lymphatic glands, and in the spleen.

A. The alterations taking place in the solitary and agminated glands of the intestine are usually divided into four stages.

(1) *The Stage of Infiltration.*—In this there occurs a hyperplasia of the lymphatic follicles, chiefly of the lower part of the ileum and the cæcum, but sometimes also in the lower part of the jejunum, the colon, and even the rectum. In some cases the large intestine is the portion chiefly involved. It has been claimed that the process has been observed in the duodenum and stomach also. The gray-red, hyperæmic, and pearl-like solitary follicles, enlarged to the size of a pin's head or that of a pea, project above the surface of the mucous membrane. Their capillary blood-vessels are greatly dilated, and finally become choked with blood-cells. Later the follicles undergo a great increase of their cellular elements and grow firmer, anæmic, whitish, and opaque. The infiltrating cells are largely of the nature of lymph-corpuscles, but some are very large and may contain ten or more nuclei. The glands of Peyer's patches also become more prominent, and form flattened oval projections. They retain their normal outline, and are separated sharply by upright or overhanging edges from the surrounding mucous membrane. The infiltration may extend beyond the glands to the membrane, the blood-vessels of which become injected, and it may reach even the muscular or serous layer. The changes in Peyer's glands are more or less widely diffused. The lower part of the ileum is in all cases chiefly involved, and in mild cases a few patches in this region are the sole seat of the infiltration.

The first stage begins early in the disease. Murchison has detected it in two cases dying at the close of the first day of the attack. It reaches its height about the middle of the second week. In a large number of glands resolution now takes place, the cellular elements becoming fatty and granular, and being absorbed. The plaques may gradually become less swollen, preserving meanwhile their even surface; but as the retrogression takes place more rapidly in the follicles than in the cellular infiltration of the interfollicular tissue, the former are very apt to seem depressed and a reticular appearance is given to the plaques. It is perhaps still more probable that this appearance is due to a necrosis of the follicles, leaving little pits. The "shaven-beard" appearance also may be produced by the deposit of pigment, the result of hæmorrhagic extravasation, in the depressions in the follicles. The plaques may exhibit this pigment even years after recovery from the disease.

(2) Should resolution not occur the *stage of necrosis* develops. The blood-vessels become compressed by the surrounding cellular infiltration, and in consequence of lack of nourishment the follicles die and form sloughs. This process may occur in all or in only some of the glands of the patches, and

may be superficial or extend even to the serous layer of the intestine, finally producing perforation of the bowel. The solitary glands undergo the same change to some extent. The process is most marked at the lower part of the ileum, and in bad cases the greater part of the mucous membrane in this region may be in a sloughing condition. The necrotic tissue is sharply demarcated from the surrounding parts, has a yellowish, greenish, or brownish color, and becomes softer. The neighboring tissue is often decidedly hyperæmic.

The second stage rarely begins before the middle of the second week, and reaches its height toward the end of this week.

(3) Following the necrosis and directly dependent upon it is the *stage of ulceration*. The sloughs loosen and gradually separate, beginning at the periphery, and finally, at about the end of the third week, become completely detached, leaving ulcers of varying sizes and shapes. Sometimes a whole Peyer's plaque is involved, producing an oval ulcer of corresponding form. More frequently several irregularly-shaped ulcers, separated by bands of mucous membrane, may be seen in one plaque. At the lower part of the ileum the ulcers often run together to a great extent, and occupy almost the entire circumference of this portion of the bowel. The solitary glands likewise undergo ulceration, producing ulcers of a rounded form. The walls of the ulcers are hyperæmic, swollen, and often overhanging. The floor varies in character according to the depth to which the necrosis has penetrated, being smooth and usually of a gray color if the ulceration be superficial, showing the parallel lines of the muscular fibres if the mucosa has been entirely penetrated, and being smooth and transparent if the serous layer be reached.

The ulceration of the solitary follicles is apt to be well marked in the colon, and especially in the cæcum, where the ulcers are often very numerous. Eichhorst has observed a case in which the only ulcer discoverable anywhere was at the tip of the vermiform appendix. The ulceration may extend so deeply that perforation may take place into the peritoneal cavity. This was found to have occurred in 5.7 per cent. of the 2000 autopsies on cases of typhoid fever made at the Munich Pathological Institute, and in 21.2 per cent. of the 64 autopsies made at the Montreal General Hospital.

(4) The *stage of cicatrization* follows immediately upon that of ulceration. It usually begins at about the commencement of the fourth week and continues for two or more weeks. The walls of the ulcers become less swollen, and attach themselves to the subjacent tissue. Delicate gray granulations cover the floor of the ulcers, and sometimes secrete pus. Later the granulations are replaced by connective tissue. The cicatrices thus formed remain as smooth thin spots for years, often exhibiting pigmentation. Epithelium covers the cicatrices, and villi may even grow upon them, but the true adenoid tissue is probably never replaced.

Any one of the stages described does not exist at one time in the intestine to the exclusion of other stages. Different glands may be found illustrating two or more stages. The neighborhood of the ileo-cæcal valve is the portion

of the bowel usually exhibiting the most advanced stages of the glandular lesions. Again, the same Peyer's patch may be undergoing cicatrization in one part, while sloughing is still proceeding or ulceration actually spreading in another part. Such a condition of course prolongs the stage of healing very greatly, and may lead to perforation after convalescence is seemingly well under way.

B. Contemporaneously with the early changes in the intestine, alteration takes place in the mesenteric glands, especially in those in the vicinity of the part of the bowel most affected, and usually, though not always, in proportion to the degree of involvement of the intestinal glands. Intense hyperæmia is followed by swelling due to cellular infiltration. The soft, swollen glands, of a bluish-red color, may vary from the size of a bean even to that of a small hen's egg. On section the central portion is often of a lighter shade than the periphery. At about the time of ulceration in the intestine resolution begins to take place in the mesenteric glands, the histological process being identical with that seen in the intestinal follicles. The color then becomes paler and yellower, and the swelling diminishes, although the glands are apt to continue hyperæmic and firmer in consistence. Where the swelling has been very great spots of necrosis with softening occur, especially in the central portion, but the puriform fluid thus formed becomes absorbed if the process has been limited to a small area. When, however, it is extensive, a large part of a gland breaks down and later is transformed into a cheesy and, finally, calcareous mass. Sometimes a liquefied gland bursts into the peritoneal cavity.

Glands in other parts of the body also sometimes become congested and enlarged. Particularly is this true of the retroperitoneal and bronchial glands and those in the fissure of the liver. In fact, any of the lymphatic glands may occasionally undergo this change to some extent. According to Liebermeister, the lymphatic follicles at the root of the tongue and in the tonsils are often affected in the same way early in the disease, but almost always undergo resolution.

C. The spleen nearly always becomes enlarged in typhoid fever. Birch-Hirschfeld, however, states that this enlargement not uncommonly fails to occur in elderly persons. It may also be absent when the capsule has been thickened by previous inflammation and the organ has become firmly adherent to surrounding parts. The increase in size begins in the middle of the first week, and reaches its height toward the end of the second week, the organ being then two or three times its normal dimensions. In the fourth week diminution in volume begins, and dimensions nearly normal are reached by the end of the fifth week. The degree of enlargement varies much in different epidemics, being greater in the severer outbreaks. The enlargement begins with hyperæmia, the organ being tense, firm, and of a uniformly deep-red color when cut. The capillaries and veins are dilated, and the sinuses contain an accumulation of red and of white blood-cells.

Gradually the splenic tissue grows softer and more granular, and finally almost diffluent on section, and in the second and third weeks the Malpighian

bodies, often hyperplastic, appear as small grayish points. Blood-pigment is now very abundant. Numerous large multinuclear cells are found in the veins, and very many splenic cells containing red blood-corpuscles or fragments of them are present. Sometimes as many as twenty corpuscles thus encysted may be discovered.

As the spleen grows smaller the capsule becomes wrinkled and often covered by grayish lines. The splenic tissue becomes paler, firmer, and often browner, and the stroma is more apparent. Hæmorrhagic infarcts are present in a proportion of cases variously estimated at from 3.6 to 7 per cent. These infarcts may sometimes soften and rupture. Rupture of the spleen may also occur from mechanical injury. In the 2000 Munich cases already referred to rupture took place in 5 instances.

II. The lesions of the second group—*i. e.* those not characteristic of typhoid fever, but more or less frequently seen after it—may be briefly discussed.

Cadaveric rigidity occurs early, and is very persistent in patients dying at the height of the disease, but it is only slight when death has taken place in the later stages. The degree of emaciation varies, and is not infrequently only slight even after two or three weeks of fever. Post-mortem ecchymoses, generally dark in color, but pale after a protracted illness, are usually abundant in the dependent portions of the body. The characteristic lenticular spots are never seen after death, but sudamina are often visible. Abscess or gangrene of various parts of the body may occasionally be found. The voluntary muscles are dry and of a dark-red color in the earlier periods of the disease. In the third week yellowish spots and grayish, wax-like streaks appear in them, or an entire muscle may be transformed into a shining, gray, friable mass. Hæmorrhages and abscesses sometimes occur in the substance of the muscle, due to the rupture of the degenerated fibres. Rupture of the muscle itself has been reported.

The peculiar histological changes in the muscles—seen in other long-continued febrile conditions as well—were first pointed out by Zenker, who described two forms: the one a granular degeneration, the other a waxy variety. The first is the more common, but the two are often associated. In the granular degeneration the fibres are filled with granules which are in part albuminous and in part fatty. In the waxy form the striæ disappear completely, and the muscle is transformed into a glistening, waxy mass. The muscles most apt to be affected are the adductors of the thighs, the recti abdominis, the pectorals, the diaphragm, and those of the tongue. These degenerative changes are most marked in the second, third, and fourth weeks. Later than this but little evidence of the process can be found. The cause of the degeneration is usually believed to be continued hyperpyrexia.

The muscle of the heart is affected in a similar manner, though the granular degeneration much exceeds the waxy in frequency. The organ is dilated, flaccid, soft, and has a pale-yellowish, "faded-leaf color," as it is commonly described. The degeneration takes place in patches, fibres seriously diseased,

with the striæ invisible, lying adjacent to others scarcely at all affected. The papillary muscles are those oftenest attacked.

Myocarditis is not uncommon, and proliferation of the muscle-nuclei with infiltration of the connective tissue occurs. Dewevre found granular degeneration of the myocardium in 16 out of 48 cases. Endocarditis and pericarditis are uncommon.

Thrombi are frequent in the chambers of the right side of the heart, as also in the veins of the body, particularly the femoral, but rarely in the cerebral sinuses. The minute arteries of the body exhibit sometimes an endarteritis or a fatty degeneration.

According to Ponfick, the marrow of the bones exhibits at times changes similar to those of other lymphoid organs. It exhibits numerous large cells which contain many red blood-corpuscles and, later, pigment. Periostitis sometimes may be seen, and Helferich has observed chondritis of the ribs in a number of instances.

The pharynx and œsophagus may sometimes be congested and exhibit ulcers late in the disease. A diphtheritic deposit is occasionally observed on the pharynx. The stomach is in some cases congested. Softening is also found at times, but is very probably a post-mortem change. Ulceration sometimes occurs, but is rare. The same conditions have been observed in the duodenum. The jejunum and upper part of the ileum may be congested or may be paler than normal, but rarely present any other alteration, except the typhoid ulceration sometimes present. Great gaseous distension of these parts is uncommon. The lower portion of the ileum is in a catarrhal condition and is more or less collapsed. Its mucous membrane is reddened, particularly near the ulcers, and sometimes exhibits post-mortem softening. The mucous membrane of the cæcum and colon may be of normal appearance or of a pale color, or sometimes injected and softened. Flatulent distension of the colon is usually marked.

The liver shows evidences of parenchymatous degeneration. Early in the affection it becomes hyperæmic. It is often softer than normal, and the outlines of the lobules are indistinct. Usually it is somewhat pale, and the cells under the microscope are granular and full of fat, with the nuclei indistinctly outlined. In advanced cases the organ approaches the appearance seen in acute yellow atrophy. This was seen in three of the Munich cases. Wagner has described small lymphomata, and Handford small necrotic areas in the organ in persons dying during convalescence, while Hoffmann found numerous multinucleated cells as well as small mononucleated cells, a condition which he regarded as evidence of a regenerative process. Embolism, abscess, and emphysema have been reported as rare occurrences. The mucous membrane of the gall-bladder may exhibit a catarrhal or diphtheritic inflammation, or ulceration of it may occur. The bile is thin and watery when the disease has lasted three to four weeks.

Hoffmann states that the pancreas and salivary glands early become larger and firmer, and, on section, browner. Under the microscope are found an

increase in the number of cells and a granular degeneration of them. Suppuration of the parotid gland occurs as one of the complications of the disease.

Peritonitis is, of course, found in cases where death has followed perforation of the bowel. Under these circumstances it is usually general, with considerable plastic and sero-purulent effusion. In rare instances the lesions of peritonitis, even of the most advanced degree, may be found, although no perforation has occurred, and no starting-point for the inflammation exists in connection with any ulcer which, though not actually perforating, is so deep as to involve the serous membrane. I have seen this several times, but only in young subjects. It appears as though it depended upon a true localization of the morbid process in the peritoneum in these particular cases, just as at other times it may occur in the pleura. Recently I have seen with Dr. George S. Gerhard a boy, aged fifteen years, who in the course of a desperately severe attack of typhoid had sero-plastic pleurisy on the right side, together with peritonitis which resulted in a very large indurated mass occupying the right hypochondriac and median region, apparently composed of enlarged glands, inflammatory exudation, and agglutinated coils of intestine. (See Fig. 7). The slow resolution of this large mass occupied several months, but complete recovery followed finally.

The kidneys exhibit a parenchymatous degeneration similar to that in the liver. They are commonly slightly swollen, rather pale and flabby, and somewhat cloudy on section. There is present a granular and fatty degeneration of the epithelial cells of the tubules, particularly of those of the convoluted portion. Infarcts are occasionally seen. Miliary lymphomatous nodules may rarely be met with, similar to those in the liver. These may also be sometimes found in the peritoneum. Miliary abscesses may develop from the lymphomata in the kidney, and some observers have found the bacilli in the pus from these. A diphtheritic inflammation has been observed in the pelvis of the kidney. Osler noted this in 3 of his 64 autopsies. The bladder likewise may exhibit a diphtheritic inflammation, and a vesical catarrh is not unusual. Orchitis is sometimes seen.

In a considerable number of cases the larynx exhibits ulceration, situated usually on the posterior wall or on the epiglottis, or even involving the vocal cords. According to Eichhorst, bacilli can be found in the ulcers. Diphtheritic inflammation of the larynx is not unusual. Œdema of the glottis may occur and may require tracheotomy. This operation had been found necessary in 20 per cent. of the Munich series. Affections of the trachea are rather rare. Catarrhal inflammation of the bronchi is almost always present. Hypostatic congestion with splenization of the lungs is very common. Abscess and gangrene of the lung are seen with comparative rarity. Pulmonary œdema is common, infarcts not rare, and broncho-pneumonia and croupous pneumonia are very frequent in some epidemics. Pleurisy of any form it not usual.

Alterations of the nervous system are unimportant. Meningitis is very rare. A case is reported by Kamen in which the bacilli of Eberth appear to have been the sole cause. The brain-substance and membranes may early

become hyperæmic and œdematous. Later the convolutions may be somewhat atrophic. Numerous capillary hæmorrhages can be found in the cortex in some cases, but larger cerebral hæmorrhages are rare. Imridi could find only fifteen reported cases in addition to one observed by himself. Meningeal hæmorrhages may occur. Meynert has described a granular change, Popoff an infiltration, and Hoffmann a pigmentation of the ganglion-cells. The peripheral nerves may exhibit parenchymatous changes. Levin claims that the ganglia of the trunks of the pneumogastrics frequently exhibit an inflammatory process, and he believes that it is on this that such symptoms as laryngitis, paralysis of the pharynx, cardiac irregularity, and the like depend.

Clinical Description.—The conditions under which typhoid fever occurs usually render it difficult to determine the length of the period of incubation. The general consensus of medical opinion places it at about two weeks, but it is sometimes certainly less than this. In several instances under my observation, where the poison was unusually concentrated and virulent, I have felt satisfied that it did not exceed four or five days. Griesinger reports three cases in which this period seemed to last but one day, but it is exceedingly doubtful whether these instances have any real value, as the diagnosis was uncertain. More probably instances of typhoid fever, though not certainly so, were the cases in the outbreak at the school of Clapham to which Murchison refers. In this local epidemic twenty cases developed four days after exposure to the infection.

On the other hand, the period of incubation is said to extend sometimes to three or four weeks, although there are manifestly great difficulties in the way of determining the date at which the last portion of typhoid poison, which may have been the effective dose, was admitted to the system. It is possible, too, that in certain cases the germs may for a long time lie dormant in the intestine or even in the tissues until a favorable occasion arises for attacking the system.

The stage of invasion may last as long as two weeks, while in other cases, and especially if the poison be concentrated and active, the disease will attain marked severity within two or three days from the initial symptom. The sudden onset of the attack without or with such brief prodromal symptoms is rare. It is more apt to occur in children than in later life, except in cases of a very malignant type. Oftener the invasion is so gradual that it is difficult to determine the day from which the actual beginning of the disease should be dated.

The premonitory symptoms—some of which, at least, are commonly exhibited—consist of increasing sense of weakness and fatigue on exertion, light and disturbed sleep, confusion of ideas, failure of appetite, occasional colicky pains in the abdomen, a tendency to slight looseness of the bowels, nausea, coated tongue, epistaxis, bronchial cough, severe headache (which is frequently occipital), a sense of weary aching in the limbs, and not rarely a decided degree of dulness of hearing, especially toward the close of the stage of invasion. When these symptoms are present in any marked degree it is

evident that they possess a certain diagnostic importance, as in no other disease are there such varied prodromes extending over so many days. I have repeatedly been led to anticipate the approach of typhoid fever by the unusual dulness of hearing and by the persistent occipital headache coming on after a few days of general malaise. The pulse may not be disturbed during this stage, but if the temperature be taken it will usually be found that there is slight evening elevation.

The actual onset of the disease is rarely abrupt, but more frequently so in children than in adults. It may be marked by some chilliness and evidence of fever, but rarely by an outspoken rigor. The occurrence of decided fever is usually the evidence of the beginning of this stage, but as the prodromic symptoms very often merge gradually and imperceptibly into those of the actually developed disease, it is a common custom to date the beginning of this stage from the time when the increasing sense of weakness leads the patient to take to bed. As this, too, is a variable date, depending upon the severity of the attack and the will-power of the patient, it often happens that the case must be regarded as already in the third or fourth day before the confinement to bed begins or before medical aid is first sought.

The fever gradually increases day by day, usually presenting an evening exacerbation, with a remission in the early morning hours, until by the end of the first week it reaches 103° or 104° F. It must be borne in mind, however, that the temperature not at all infrequently rises with much greater rapidity.

The appearance during the first week is listless and apathetic; the hearing is dull; headache is often intense; and the patient lies, much of the time, with the eyes closed as though in sleep. Delirium is apt to occur, especially at night. In severe cases more marked nervous symptoms present themselves. The respirations are but moderately accelerated; the pulse is increased in frequency, but not always in proportion to the increase of temperature. It is full, of low tension, and often dicrotic. The tongue is coated, appetite is lost, thirst is moderate; the abdomen is moderately distended, and pressure in the right iliac fosssa will usually disclose some gurgling sounds and tenderness. Constipation is present in perhaps the majority of cases at first, but during the first week, if not indeed from the outset, diarrhœa sets in, with yellowish and liquid stools. The spleen is distinctly enlarged toward the end of the first week. At about the seventh day or later a characteristic eruption of rose-colored spots appears, usually first upon the upper part of the abdomen. There is occasionally cough, sometimes quite severe, and auscultation shows a few scattered râles. The urine presents a febrile character and is diminished in amount, especially if diarrhœa be present, and sometimes contains a small amount of albumin.

In the second week the symptoms become aggravated. Headache is apt to be replaced by an increased tendency to torpor and somnolence. Delirium is present, usually of a mild, wandering type, though it may be violent. The temperature remains high and presents a more uniform course, though still marked by daily remissions. The pulse is now more rapid, less full, and less dicrotic.

The tongue is apt to lose its coating and to become red and more or less dry. It is protruded with difficulty, and often exhibits tremor. Tremor is also seen in the limbs. The spleen increases in size. Râles in the lungs are more abundant. The abdomen grows more distended.

In the third week the temperature becomes of a distinctly remittent type, the morning fall growing more marked, and the height of the evening elevation gradually lessening. The other symptoms of the previous stage persist.

In some cases all the symptoms become worse toward the end of the second week and in the third week. The stupor grows more extreme; the patient can scarcely be roused at all; the tongue is very dry and is covered with a brown crust; the teeth are coated with sordes; the pulse is rapid and feeble; subsultus tendinum is marked; the urine and fæces are often passed unconsciously or there may be retention of urine. Weakness is progressive and great; muscular relaxation is marked; emaciation is often extreme, and there is a tendency for bed-sores to develop. Such a condition is fitly termed the typhoid state. It will be seen later that it may develop in other diseases.

These symptoms may persist until the fourth week and the patient die, if, indeed, death does not occur earlier. In cases which recover there is a gradual improvement, commencing with the opening of this week or perhaps sooner. The temperature is now even more decidedly remittent, and finally distinctly intermittent, the morning temperature reaching normal, although the evening temperature is still rather high. The mental condition clears up; other nervous symptoms improve; the tongue becomes more moist; appetite returns; the distension of the abdomen disappears; diarrhœa lessens and the stools become darker in color, and constipation is finally apt to supervene; the hypertrophy of the spleen diminishes; the pulse improves in strength and lessens in frequency; the eruption, which had developed in successive crops, ceases to appear.

Convalescence begins with the entire disappearance of fever, often marked by a subnormal morning temperature. It is gradual and often tedious, lasting into the fifth and sixth week, and sometimes not beginning until then. Various sequelæ may now occur, just as different complications may develop during the course of the disease. During convalescence, too, the patient is subject to sudden temporary elevations of temperature, produced by excitement, overexertion, or indiscretions in diet. These recrudescences last a day or two only, and are to be distinguished from true relapses, which exhibit other symptoms of the primary attack besides the mere febrile reaction.

Consideration of Special Symptoms.—*General Condition and Appearance.*—The expression of the face in typhoid fever is characteristic. Even from the beginning there is a drowsy, listless appearance with heavy eyes. If, however, headache be severe or fever high at the onset, the expression at that time may be excited and anxious, the eyes bright, and the pupils dilated. In very mild cases the physiognomy may be but little altered at any time.

When the disease is fully developed, if of the ordinary type, the patient lies quietly, more commonly upon the back, with the eyes often closed, and

with a peculiar placid, sleepy, and heavy expression, unless there be active delirium, when jactitation may be marked. The face is often pallid or there may be a circumscribed flush on one or both cheeks. If confined to one side, this may indicate a higher degree of congestion of the corresponding lung. The flush comes and goes, and is often brought out or made worse by the administration of food or stimulant.

The general strength of the patient is usually prostrated from the beginning. In grave cases weakness becomes so extreme as the disease advances that the patient lies utterly helpless on his back or slides down in bed. In very mild cases, on the other hand, there may be but very little prostration. The patient may be about or may rebel against confinement to bed. Cases are met with in which the patient has kept about until very shortly before death.

Emaciation frequently becomes great, or even extreme in cases which have lasted several weeks. According to the studies of Cohin, there takes place at first a systematic loss of weight, varying with individuals, the loss bearing a uniform relation to the course of the temperature. Later the patient begins to gain weight, the constant increase being an evidence of convalescence. Zieniec found as a result of the study of 384 cases that there was an average daily loss of weight of 0.6 per cent. which continued while the fever lasted and even longer. In the event of delirium or other threatening symptoms, or of the development of complications, the daily loss became 1 to 1½ per cent. If the increase in weight during convalescence suddenly ceased, a relapse was probably indicated. In fatal cases the total loss was 22 per cent. of the body weight.

Skin, Muscles, etc.—The skin is often persistently hot and dry throughout the whole course of the disease, but more frequently more or less sweating occurs. There may be sudden flushings or sudden outbursts of perspiration. Sweating is more common in typhoid fever than in almost any other of the acute diseases except malaria, relapsing fever, and rheumatism. It is usually slight, occurring at night or on awakening in the morning or after the employment of the bath, but it may develop at other times, may be limited to the face and head, or may affect also the trunk or extend to the entire surface. In severe cases, marked by a high degree of nervous ataxia and exhaustion, the body may be bathed in sweat continuously for many hours or even for several days. A special sudoral form of typhoid fever has even been, though unnecessarily, described by some observers.

The characteristic eruption of typhoid fever demands close study, as upon it the diagnosis depends in many cases. It consists of isolated round or lenticular, rose-colored, slightly elevated spots, which first appear usually on the seventh or eighth, but occasionally not until the tenth or twelfth, day of the disease, and then continue to make themselves visible in successive crops. They are rarely to be discovered after the middle of the third week. They are, as a rule, first found upon the upper part of the abdomen and lower part of the chest, and may be limited to that region. Occasionally these parts do not exhibit any rash, while other portions of the body do. The

spots often also appear on the sides of the trunk and on the back, and sometimes upon the extremities. In very rare instances they are spread over the entire surface, and I have seen face, trunk, and extremities closely dotted over with them. When thus copious the spots may, to a slight extent, be confluent by twos and threes by the edges. They are soft and very slightly elevated papules of a pale, rose-red color, varying in diameter from 1½ to 2 or 3 lines, disappearing rapidly on pressure and returning promptly when the pressure is removed. Each spot lasts three to five days, and then gradually fades, leaving sometimes a brownish stain. Fresh crops appear at intervals of three to five days. There is no uniformity in the amount of eruption nor in the number of successive crops, nor does the extent of eruption or the number of crops or of individual spots bear any relation to the gravity of the case. Murchison has counted as many as one thousand spots in a single case. Generally, however, the number is quite limited, and careful search may sometimes fail to detect more than two or three spots during the whole course of the disease.

The eruption is sometimes entirely absent throughout the case. This happens oftener in children than in adults. Murchison reports its presence in 4606 of the 5988 cases of typhoid fever which occurred in the London Fever Hospital during twenty-three years, and probably careful search would have shown it present in still more of them. Eichhorst failed to miss it entirely in over one thousand cases under his own observation.

Although I admit that the observation is doubtful, owing to possible want of sufficiently frequent and careful search, it is my opinion that in different outbreaks and in different seasons there may be great difference in the amount of the eruption, and that in some of our epidemics it has not been extremely rare for the typical typhoid spots to be almost or entirely absent, and this especially in young children.

We should never conclude that no rash is present until after repeated and careful examination, not only of the abdomen and chest, but of the back and thighs as well. The importance of this critical examination cannot be overestimated. Occasionally some of the rose-colored spots may be capped by a small vesicle with turbid contents.

Certain accidental eruptions may be seen in typhoid fever, and it is important not to confound these with the true rash.

Sudamina, or minute pearly vesicles, occur more frequently in this than in any other of the infectious diseases. They are, however, in no sense characteristic of it, since they may appear in any affection that is attended by copious sweating. They are as likely to develop in mild as in severe cases of typhoid, and may be present at almost any stage of the disease, though they are not usually met with until after the twelfth day. They occur most commonly on portions of the surface where the cuticle is tender and where there is normally a tendency to perspiration. Thus they are seen about the epigastrium, the hypochondriac regions, the axillæ, the neck, or about the groins and the inner surface of the thighs. They are so minute and delicate that it

is often necessary to view the surface obliquely and in a good light in order to detect them, and they may sometimes be perceived by the finger when they cannot easily be distinguished by the eye. In some cases they are very copious, and may appear in several successive crops. As sweats are not uncommon in typhoid fever, the appearance of sudamina cannot be regarded as heralding a break in the pyrexia.

The contents of the sudamina may later become turbid or milky; so that, as they dry up, thin, grayish scales are formed, which readily desquamate. Slight desquamation may also occur in cases in which the rose-colored spots have shown a tendency to fade very slowly.

An erythematous eruption of a faint scarlet color is sometimes present in the first week of typhoid fever, and has even been mistaken for the rash of scarlet fever. It is particularly liable to occur on the abdomen and chest, but may sometimes be detected on the extremities as well. Petechiæ rarely occur, and oftener develop independently of the rose-spots than exist as transformations of them. Urticaria is sometimes seen. Herpes labialis occurs occasionally, but by no means so often as in cerebro-spinal fever or malarial fever. During the height of the disease it is often possible to elicit a distinct red streak with white edges by drawing the finger across the cheek or brow: this closely resembles the *tâche cérébrale* of meningitis.

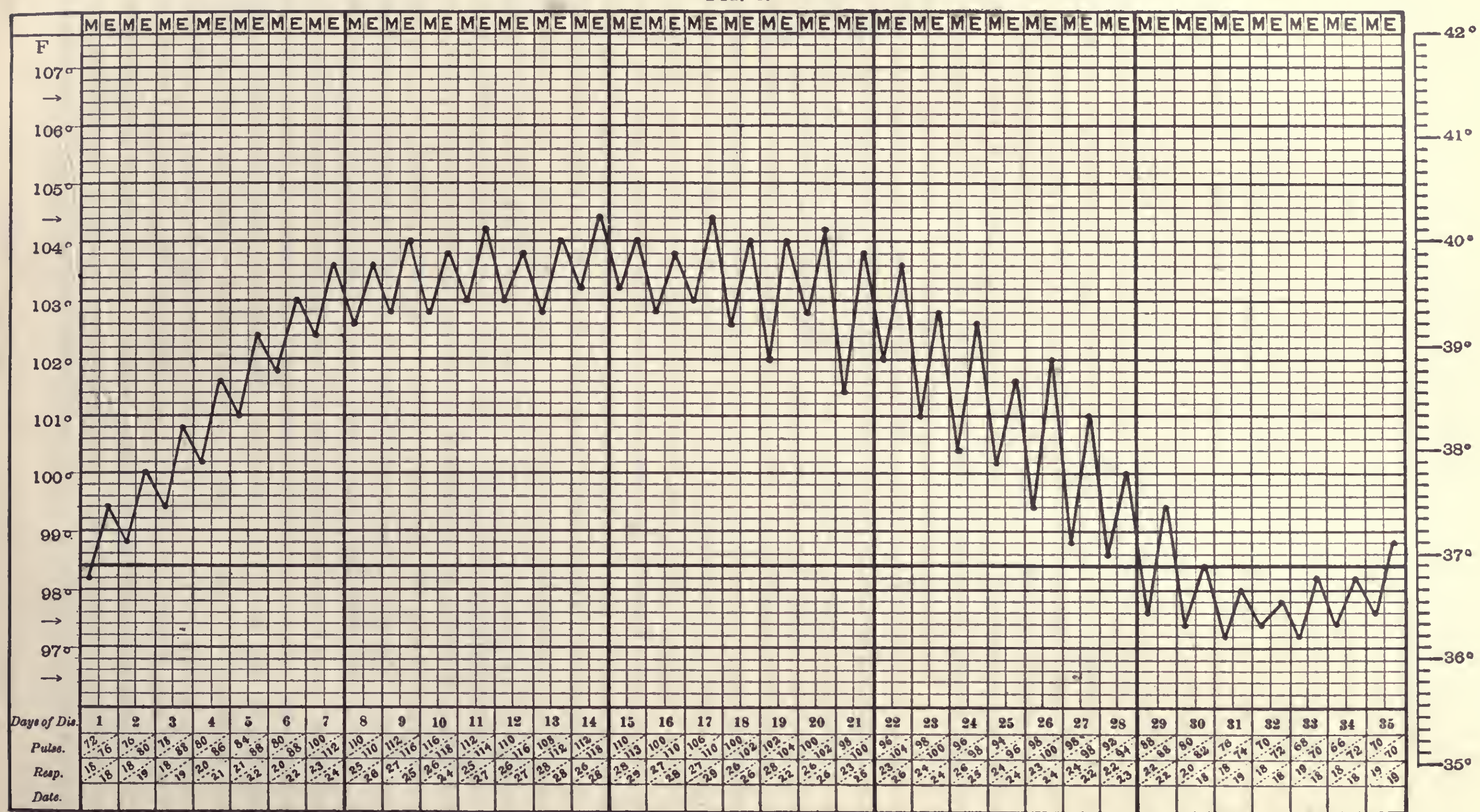
It is pertinent to mention the occasional occurrence of subcutaneous mottling. This is seen especially where the skin is very fair and sensitive, and is apparently a vaso-motor phenomenon occasioned by exposure of the surface of the body. Portions of the skin are unusually white, while there is in other places a more or less extensive pinkish injection, thus producing a mottling which at times is extremely marked. It has, however, no special significance. The same may be said of the pale-blue, subcuticular patches, "peliomata," or "*tâches bleuâtres*" of French writers. They vary in diameter from three to eight lines, are of irregularly rounded form, not at all elevated, of a uniform tint throughout, and not affected by pressure. They are most abundant on the abdomen, chest, and thighs, and their appearance is often very striking. They are by some referred to the action of body-lice.

Œdema of the skin of portions of the body may develop in typhoid fever as a result of several causes, prominent among which is venous obstruction. Nephritis and anæmia may also produce a more general œdema.

It has been claimed that the skin exhales a peculiar odor in typhoid fever. It was described as "of a semicadaverous and musty character" by Nathan Smith. A number of writers agree that a peculiar odor is present, although perhaps more deny its existence. I am myself convinced that a characteristic odor is often to be noticed about patients with the disease, especially in cases attended with sweating.

Temperature.—The course of the fever presents many variations and irregularities; still, the careful study of a large number of temperature-charts shows that the pyrexia is more or less characteristic. For our first knowledge of this fact we are largely indebted to Wunderlich. The accompanying chart (Fig. 3)

Fig. 3.



Typical Chart: Step-like ascent, the maximum being reached in seven days; then continuous fever with slight diurnal variation during the second week; and, finally, more markedly remitting temperature and gradual descent until the normal is reached, about the twenty-eighth day; subnormal temperature during early period of convalescence.

represents diagrammatically what may be called the typical pyrexia. In the early stages of the disease the curve exhibits a gradual ascent, occupying about one week, during which each successive daily maximum and minimum is from one and a half to two degrees higher than the corresponding points of the previous day. By this step-like ascent, with a daily variation likewise of fully one and a half to two degrees, a temperature of 103° to 104° F. is reached by the close of the first week or sometimes earlier. Following this initial period there is present, for about two weeks, a febrile movement of a more uniform severity, often spoken of as the fastigium. The maximum daily temperature now oscillates about the maximum temperature of the preceding period. The morning remissions are much less marked, although the daily range is still one to two degrees. The course of the fever during this period is marked by occasional fluctuations in which the temperature drops considerably below the average or else rises to the point of hyperpyrexia. During the third week the fever begins to fall gradually, but by more irregular steps than it showed in the initial rise. In a typical case the evening exacerbations are, for a time, as great as before, but the morning remissions become daily more marked. Very soon the evening maximums also begin to grow less by about half a degree every day, while the morning remissions are still more decided. There may be a difference of as much as two to four degrees between the daily maximum and minimum, and by the close of the third week or in the fourth week the morning temperature is nearly or quite normal, or even sometimes subnormal, though an evening exacerbation is still present. The pyrexia thus often has a somewhat intermittent character. The evening temperature continues to fall gradually, and with considerable regularity in typical cases the normal is attained about the twenty-eighth day of the disease. It is not unusual during the period of defervescence to have an evening maximum higher than that of the previous day, but followed by a more abrupt fall on the following day.

While a temperature curve possessing these features may be regarded as the type, it must be understood that there are many variations, and that a typical temperature chart is not often seen. This will be understood when the complex character of the pyrexia in typhoid fever is considered. Not only is there the general infectious process, with the morbid chemical changes in the blood and tissues and the disordered nervous action affecting the production and dissipation of caloric, these acting as the exciting causes of the primary fever, but there are often, even from an early period in the disease, widespread lesions which develop with irregular rapidity and influence powerfully the febrile movement, producing what may be called the secondary fever. In addition to these is the operation of numerous and varied accidental factors influencing the temperature curve, such as indiscretions in diet, the occurrence of intestinal hæmorrhage, temporary nervous excitement, profuse diarrhœa, free epistaxis, and the development of complications. I know of no disease in which it is more difficult to appreciate the origin and meaning of the pyrexia.

In certain rare cases a high temperature, even 105° F., may prevail almost continuously, day after day, for two weeks, and yet be unassociated with any

grave nervous symptoms or evidences of heart failure. I have observed this most frequently in young and sensitive women, in whom it was apparently due to a peculiar disturbance of the nervous system, since there were no marked pulmonary or intestinal symptoms to explain any considerable portion of the elevation. On the other hand, it is not exceptional to meet with cases, especially of patients of phlegmatic disposition, where all the symptoms are fairly well marked, and yet the temperature does not exhibit a corresponding rise. Undoubtedly, there is, however, a general correspondence between the gravity of the case and the height of the fever; and this is true whether the attack owes its severity to a high degree of infection or to a marked development of local lesions. Cases where the temperature is throughout little above the normal are generally of mild type, although, as will be seen later, there is danger in them, as in others, of grave complications arising.

The most characteristic feature of the temperature curve of typhoid fever is the gradual initial rise. This is important in its bearing on prognosis, but especially in relation to diagnosis. There are many cases of influenza and other affections in which, about the close of the first week, the symptoms closely resemble those of typhoid fever, but in which the fact of a more abrupt onset is a guide to the avoidance of a serious error. It must not be forgotten, however, that a rapid initial rise in temperature to 103° or 104° F., with or without preceding chill, may occur in typhoid fever also. This is, at times, met with in very grave cases, but it may also be noticed in those of ordinary severity, especially in children or when there is an unusual degree of pulmonary or gastro-intestinal irritation at the beginning of the attack. An implicit dependence upon the typical mode of ascent during the first week may readily lead to mistakes. As an illustration of the caution requisite I may mention two cases of typhoid seen in consultation as these pages go through the press. Of four children—two girls aged nineteen and eight, respectively, and two boys aged seventeen and fifteen, respectively—the older girl was taken suddenly ill in the night with vomiting, and the next morning had a fever of 105° F.; the younger boy was taken ill the following morning, and before night his temperature reached 104.6° F. The girl, on subsequent inquiry, stated that she had not felt bright and strong for a week, but twenty-four hours before the onset she had been to a large dinner-party. The boy had continued to bathe in the ocean and to play tennis until the day preceding the attack. It is quite certain that, had the temperature been taken regularly during the previous week, some slight ascending fever would have been found, since in both cases within thirty-six hours of the apparently abrupt onset copious eruption appeared, showing that the seventh or eighth day of the disease had probably been reached. Yet for the practical purpose of early diagnosis the attacks seemed as sudden as though of acute gastritis.

The fluctuations which occur during the second and third weeks are difficult of explanation. As already stated, the curve usually presents marked diurnal variations of from one degree to two degrees between the minimum and maximum. The shorter the time that the temperature re-

mains high in each twenty-four hours, the better is the fever borne as a rule. The indication is unfavorable when a high temperature is maintained almost continuously. On the other hand, extreme variations, as from three and a half to five degrees, are usually associated with nervous atony and with marked sepsis from the intestinal ulceration. The most extreme daily variations of temperature I have noted in this disease amounted to seven degrees for several days in succession in a fatal relapse complicated by extensive catarrhal pneumonia. In some cases the appearance of successive crops of eruption and the variation in the intensity of the abdominal symptoms correspond with exacerbations of fever, and suggest a relation between the latter and the varying intensity of the intestinal lesion.

Hyperpyrexia, or fever above 105° F., is much less common in typhoid fever than in typhus, scarlet, or relapsing fever. When present it usually indicates a high degree of danger, and the cases in which it occurs more than a few times exhibit a large percentage of mortality. Nevertheless, it is not infrequent to have recovery follow where a temperature of 106° F. has been reached several times during an attack, provided that the fever has not remained too continuously so high. Very high initial temperatures indicate intense infection or violent nervous disturbance, or an early complication, such as marked gastric or pulmonary catarrh. During the second or third week hyperpyrexia is more common than at any other time. When the temperature rises with less decided remissions toward the close of the second week, or remains high during the third and fourth weeks, it indicates continuance of grave lesions or the occurrence of reinfection; and such cases are very unfavorable. As death approaches it is not unusual to note a progressive rise of temperature (see Fig. 4.), which may reach 107° or even above 110° F., as in a case reported by Wunderlich. In such cases the body remains warm for a long time after death. On the other hand, when death is about to take place by collapse the temperature sinks to normal or even below it.

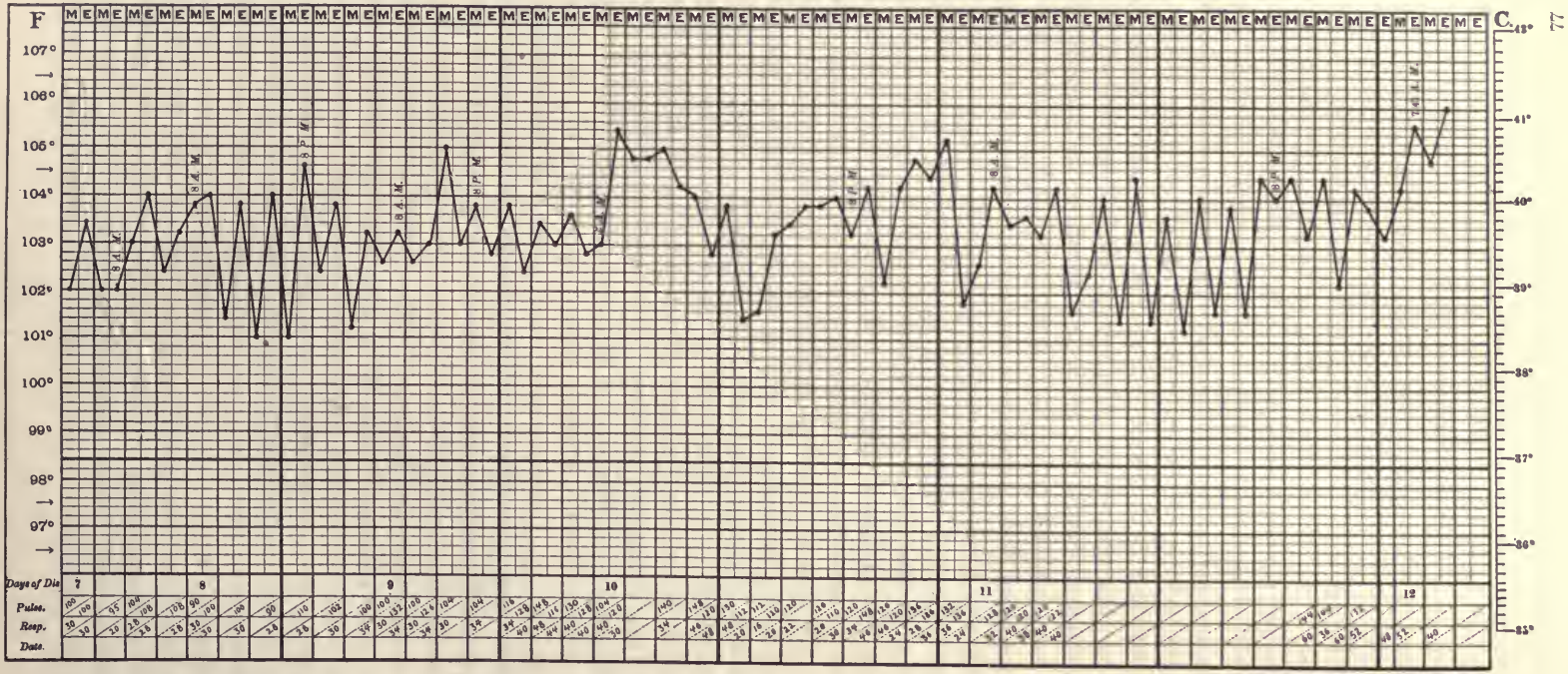
It is important to observe the time when the daily maxima occur. The study made by Ampugnani of hourly charts from 200 cases of typhoid fever shows that the maximum temperature occurred between three and six o'clock in the afternoon. The maximum is followed by a gradual fall during the night, so that the minimum is reached between four and eight o'clock in the morning.

The tolerance of the fever by the patient depends much upon the length of the remission. There is often a marked difference between successive days in this respect.

Some cases present two maxima in each twenty-four hours, the temperature pursuing a very rapid and irregular up-and-down course. The temperature is said to be inverted when the daily maximum occurs in the morning and the minimum in the evening. This is not unusual in cases occurring under the age of twelve years. It may, however, be present at any period of life, and has no special significance.

There is no crisis or abrupt fall of temperature in the normal curve of

FIG. 4.



Highly irregular, atypical curve, with hyperpyrexia. Patient admitted on sixth or seventh day; soon delirious, then incontinence of fæces, retention of urine, and marked nervous symptoms. Diarrhœa not excessive, but tympany marked. Treated with repeated cold sponging, enemata of turpentine, hypodermic administration of strychnine, caffeine, and digitalis. Fever did not abate, nervous symptoms and general depression increased, and death resulted on the twelfth day of the disease.

typhoid fever. Any sudden drop must therefore be viewed with suspicion. It may indicate the approaching development of a serious complication, as when, owing to some carelessness in nursing, the patient has been allowed to become chilled and there is to be an attack of pneumonia. It may mark the occurrence of severe intestinal hæmorrhage, and the temperature may fall suddenly as much as five, seven, or, in rare cases, even nine or ten degrees, with marked evidences of shock, before the bloody discharges occur to furnish the positive explanation. It may attend the occurrence of a perforation of the intestine. Occasionally a fully-developed case which is pursuing apparently the usual course will culminate early in the third week in a rather rapid fall in temperature to the normal, and this be followed by uninterrupted convalescence. These abortive cases may owe their short duration to the slight degree of intestinal lesion and to the early stoppage of infection from that source, as well as to the absence of the secondary fever which extensive ulcerative processes would naturally produce.

It is never safe to consider the disease ended until the temperature has been at or a little below normal both morning and evening for several days in succession. If the temperature continue to rise even to 99⅖° or 99⅗° F., though the morning temperature be normal or somewhat subnormal, it must be understood that some lingering trace of the disease is still in the system or that some complication is present. Occasionally a post-typhoid anæmia may account for this daily slight evening rise, or the evening fever is purely nervous in origin, and will be cured by allowing the patient to abandon the bed.

It often happens that after the temperature has fallen to the normal there will be an irregular rise, which on close examination will be found due to the occurrence of phlebitis, periostitis, a latent pleurisy, or some other sequel. In other instances, after convalescence has been established for two or three days, the temperature will rise again rather suddenly, and remain elevated for a day or two without any severe constitutional disturbance being present and without the action of any complication or sequel. These recrudescences are due to various slight causes, as fatigue, excitement, indiscretions in diet, etc. In the event of a true relapse of the disease the temperature, after being strictly normal for several days, will begin for a second time a gradual, step-like ascent, reaching 103° or 104° F. by the sixth or seventh day, and then pursue, for a week or ten days, a fluctuating course similar to that in the original attack; this being followed by a gradual decline to the normal again. Lastly, there are certain cases of typhoid fever, to which reference will be made again, in which the temperature never rises above normal.

The *Nervous Symptoms* deserve minute study. It occasionally happens that throughout the course of a case the mind will remain clear and the special senses almost normal, but such instances are exceptional. Usually there appears at an early stage of the disease a mild degree of drowsy dulness, styled hebetude. The patient looks and seems sleepy, and will lie quietly, with the eyes closed, paying little or no attention to his surroundings and rarely making any remark. If addressed he opens his eyes leisurely, and in a slow and

deliberate voice returns an appropriate answer. He seemingly relishes liquids when given, yet would go a long time without asking for nourishment or drink. When roused he soon falls back again into the same somnolent state, so that it is possible to administer food and remedies at regular intervals without interfering with his rest.

Headache is one of the most frequent of the symptoms of typhoid fever, and is often complained of bitterly at the onset. It is, as a rule, most severe in the occipital and cervical region, but at times extends anteriorly. It may be so violent as to arouse fears of meningitis, especially when combined, as it sometimes is, with retraction of the head, twitching of the muscles, and allied symptoms. In some cases it persists throughout the greater part of the attack and constitutes the most troublesome symptom. More commonly it subsides as hebetude develops, which after the first week renders the patient too dull to notice it clearly even if it exists. Headache appears to be as frequent in children as in adults. Its presence does not seem to be any indication of the severity of the attack.

Vertigo often accompanies headache, but usually disappears with it. Wakefulness at night, with restlessness, is usually complained of during the early portion of the attack. It may appear again later in the disease, associated with wandering or more violent delirium. It is at times a troublesome symptom, and, though the patient may seem dull, there may be little or no actual sleep. If this condition persist exhaustion is apt to ensue. It is most important to bear this fact in mind, because the dull appearance of the patient may mislead the attendants into the belief that he is getting sufficient sleep. The symptom is one which should receive early and efficient treatment.

Delirium of some sort may be observed at times in the majority of cases. It may be present from the start, but it oftener does not appear until toward the close of the second week, following headache and somnolence and preceding stupor. Its mildest form is simply a slight confusion of ideas, particularly noticeable toward evening or during the night or on awakening from sleep, the patient being at other times perfectly rational. The most characteristic form is that of the wandering type. The patient talks to himself rapidly, softly, and unintelligibly, and often appears to be holding a conversation with imaginary persons; and this delirium may last uninterruptedly for hours.

The wandering delirium may alternate with periods of somnolence, or, on the other hand, there may be outbreaks of active, noisy delirium, which are peculiarly liable to be attended by such efforts to leave the bed that forcible restraint becomes necessary. Occasionally maniacal delirium occurs early in the disease. It has sometimes been the first symptom noticed. A case is reported by Motet in which a patient was sent to an insane asylum before the true nature of the febrile disease was discovered.

Even when the patient has seemed almost rational during the day, it is necessary that a close watch be kept during the night, as then he often dreams of being away from home and that he is summoned to return, or he awakens with the notion that he is not in his own room, and rises quickly to go thither.

This is a very common delusion, and in this confused state serious accidents may happen to the patient from stepping out of windows or falling down stairs while trying to follow some imaginary summons or to escape from some apparently strange and uncomfortable place. It is necessary to impress this fact very clearly upon the attendants, not only in private practice, but in hospitals, since fatal results from this source are not infrequent.

In children or in young and sensitive women a violent form of delirium is sometimes met with which must be regarded as partly hysteroidal in type. There is extreme restlessness and agitation of the whole body; the patient talks rapidly and with utter and wild incoherence; at the same time there is a peculiar quality of voice and of expression, conjoined with a less degree of violence of the associated symptoms than would seem naturally to accompany such excessive delirium; which indicates the presence of a large emotional element. Such cases, although very alarming in appearance, recover habitually.

In the second or third week of the disease in severe cases somnolence, which preceded delirium to some extent and then alternated with it, may deepen so as to replace it to a great degree. The active delirium entirely ceases, and only a low, muttering form remains. Finally the patient settles into a state of more or less deep coma.

It is only in grave cases that such stupor ensues or that intelligence is so wholly lost that it becomes impossible to rouse the patient so that he will protrude the tongue when requested. Profound stupor may, however, exist for fully two or even three weeks, and then gradually clear up as the fever declines and the case approaches convalescence. Coma vigil, a state in which the patient, although in deep stupor, lies with the eyes open, fixed, and staring, is much more rare in typhoid than in typhus fever. It indicates intense nervous irritation combined with exhaustion, and is of grave omen. When it occurs it is usually toward the close of fatal cases which have been marked by violent nervous symptoms.

The organs of special sense present few disorders. Ringing and buzzing in the ears are frequent in the early stages, and allusion has already been made to the dulness of hearing frequently observed in the early days, and which is apt to continue as a marked symptom. This deafness usually occurs in both ears, and is due partly to a catarrhal condition of the Eustachian tubes and partly to the blunted mental sense-perception. Deafness in one ear is apt to be a more serious sympton. Vision is rarely affected. Sometimes there is slight haziness, or there may even be double vision. There is at times decided photophobia. Injection of the conjunctivæ is rare. The pupils are usually dilated, in contradistinction to the contracted pupils of typhus fever. The dilatation develops in the latter part of the second week, and very often accompanies delirium, though it may occur without it. Occasionally the pupils are unequal, and after stupor comes on they may become much contracted. Strabismus is sometimes seen.

Epistaxis is a common symptom, and is often one of the earliest ones, though it may occur at any period in the disease. It may vary in amount

from a few drops only, just sufficient to stain the handkerchief or the pillow, to a hæmorrhage of a profuse nature.

There is evidently a morbid condition of the nasal mucous membrane which disposes to it, and which is aided by the altered crasis of the blood. Even when no blood has escaped, the nails of the patient may show traces of it. It thus has considerable diagnostic value. Although epistaxis occurs occasionally in other infectious diseases, and is absent in some cases of typhoid fever, it is incomparably more frequent in the latter affection than in any other acute disease. It does not, as a rule, afford any relief to the symptoms of the disease, and is, indeed, rarely free enough to affect the system, although I have occasionally seen apparent temporary relief to severe headache and restlessness from a free epistaxis in the early stages of the disease. On the other hand, when there is already decided debility any considerable loss of blood is to be dreaded. In hæmorrhagic cases epistaxis is one of the commonest forms of bleeding, and even where there is no blood lost from any other surface epistaxis may be so profuse and obstinate as to induce dangerous or fatal exhaustion.

The sense of taste is often greatly impaired, owing both to the blunted perception of taste and to the thick coating of the tongue.

Cutaneous hyperæsthesia sometimes occurs, particularly in women and children, but it is not seen as often as in typhus fever or cerebro-spinal fever. It may be so severe that the slightest touch causes great suffering. It can occur at any time during the attack. Its principal seat is the superficies of the abdomen and the lower extremities. Cutaneous anæsthesia has been reported in rare instances.

With the headache already mentioned there is at times violent pain extending down the spine. Tenderness over the spinous processes may be associated with this. Pain in the extremities, particularly the legs, is of quite common occurrence, especially at the commencement of the disease. Toward the close of the first week, however, it subsides, and it is only in exceptional cases that much pain is complained of in the later periods. As a rule, patients looking back upon their attacks of typhoid fever do not speak of them as painful.

Tremulousness and weakness of the muscles, as seen in the hands, lips, and tongue, are very often present. Most marked in the severer cases, they may occur even in those patients whose mental faculties are entirely preserved. They are commonest in the old and feeble and in the intemperate.

Clonic spasmodic movements are present only in the later periods of the graver cases. Subsultus tendinum is one of the symptoms of this class, as is also twitching of the face. The condition becomes most marked and nearly constant when the low muttering delirium of the latter stages develops. Carphologia is also one of the severer symptoms. In it the patient gropes in the air after imaginary objects or picks at the bed-clothes as though to remove something from them. Obstinate hiccough may be seen toward the last stages of grave cases, or sometimes, indeed, as an early symptom. It is usually a sign of evil omen. General convulsions are unusual, being chiefly met with

toward the end of grave cases, and oftener in children than in adults. Recovery, however, may take place after them.

Rigidity of various groups of muscles is frequently seen in severe cases. In some there are marked retraction and stiffness of the muscles of the neck, and even of those of the spine. This may be as marked as in cerebro-spinal fever, but does not, nevertheless, call for a description of a special cerebro-spinal type of typhoid fever. Sometimes spasmodic constriction of the muscles of the pharynx prevents swallowing. Trismus, spasm of the glottis, and rigidity of the extremities have also been reported. I have noted in some cases an extreme degree of general muscular rigidity, with a fixed ecstatic expression of face. This may be met with in cases of hysterical type, when it bodes no special danger; or, on the other hand, it may be seen toward the close of fatal cases where there has been great nervous irritation in the earlier stages.

According to Hughlings-Jackson and Money, the knee-jerk is never absent in typhoid fever.

The Digestive Symptoms are numerous and of the greatest importance. There is no other disease in which anorexia is more marked or persistent. It is complained of during the initial stage, and lasts until convalescence begins. Usually it is only an indifference to food and not an actual aversion, and it is generally possible to administer a fair amount of nourishment, especially in the form of milk or light broth which has no decided taste. In mild cases, where there is an unusual retention of intelligence throughout the disease, I have frequently observed continuance of more or less decided appetite. Thirst is generally marked in the early stages, but later, when the mental faculties are greatly obtunded, water is no longer asked for.

The tongue, as oftenest seen in typhoid fever, is enlarged and flabby, not rarely tooth-marked around the edges, and with a whitish or yellowish coat. The papillæ are not especially prominent. The edges are generally unnaturally red, and there often is a red triangular area near the tip. At about the middle or end of the second week it may lose its coating entirely or in spots, and become bright-red, dry, clean, glazed, and sometimes fissured; but more frequently it grows brownish, especially in the centre, and may finally become coated all over with a thick, cracked, brownish crust which renders its protrusion very difficult. Toward the beginning of convalescence it becomes gradually more moist and the crust is slowly gotten rid of. It is not at all unusual, however, in cases of moderate severity for the tongue to remain moist and only slightly coated throughout the whole course of the disease.

The viscidity of the secretion of the mouth causes it to dry and be deposited as sordes upon the teeth and lips. This is particularly liable to occur when the typhoid state is well developed, but is not at all characteristic of typhoid fever alone. The lips are generally dry, and often crack and bleed if picked. The gums rarely bleed. The pharynx is commonly the seat of marked catarrhal irritation, and the mucous membrane is swollen and congested and secretes a thick mucus. The tonsils may be enlarged at the same time.

Nausea and vomiting sometimes occur, especially at the beginning, but are not common, in my experience, unless excited by injudicious feeding or medication. Late in the disease vomiting is even more rare, except as the result of peritonitis or of ulcer of the stomach. The morbid irritability of the stomach is at times marked, and I have seen violent nervous symptoms, even convulsions, produced by minute amounts of solid food. Rarely vomiting is so persistent that death may follow from exhaustion.

Tympanites is a very frequent symptom. Generally it does not develop before the second week, but sometimes is seen earlier than this. It varies in degree from slight meteorism to extreme distension, sufficient to interfere with breathing and heart-action and to cause extreme distress. It is generally most marked in severe cases, especially if diarrhœa be a prominent symptom, but it may develop independently of this. It is due to the influence of the intestinal ulceration paralyzing the peristaltic movements of the bowel, to the degeneration of the muscular coat of the bowel, and to the production of gas from decomposition of food and of the intestinal discharges. Tympanites once developed is apt to persist, though often varying in degree upon different days.

Abdominal pain and tenderness are very commonly observed. The pain may be due, as stated, to abdominal distension or may be directly produced by the ulceration of the bowel. Fugitive griping pains often occur among the earliest symptoms. Tenderness on pressure is chiefly found in the right iliac fossa, and is caused by the intestinal ulceration in this region. Nevertheless, severe ulceration may sometimes be present without producing tenderness.

Gurgling is often elicited by pressure in the right iliac fossa, and is due to the presence of gas and liquid in the lower part of the ileum. It may occur in any disease accompanied by diarrhœa.

Diarrhœa must be considered one of the cardinal symptoms of typhoid fever. It may be one of the early manifestations of the disease, perhaps present even upon the first day of the onset, or possibly among the prodromes, but it more frequently develops toward the end of the first week, and sometimes not until late in the disease. It may last for a few days or may persist throughout the whole attack. Its severity varies greatly, the movements numbering two to four daily as an average number, but in many cases reaching ten or twelve or even more in the twenty-four hours. The evacuations are rarely accompanied by pain, and tenesmus does not occur. Occasionally, brief griping pains will precede each movement of the bowels, and I have known the occurrence of frequent painful contractions of the rectum. The gravity of the case is in direct proportion to the severity of the diarrhœa, although two or three loose movements daily need cause no special uneasiness, provided they be small and unattended with any symptoms of exhaustion.

Even in cases of decided severity, however, diarrhœa may be entirely absent. In my own experience the bowels are more often quiet than is generally taught; and, indeed, constipation is not rarely present to such a degree as to require attention. Even in such cases, however, the fact must

be borne in mind that the intestinal tract of every patient with typhoid fever is in an irritable condition, and that drugs given to open the bowels act with unusual activity.

The severity of the diarrhœa bears little proportion to the degree of the ulceration. A very extensive ulcerative process, followed by perforation or fatal hæmorrhage, may occur in cases in which there has been no diarrhœa or any other abdominal symptom. In fact, the looseness of the bowels depends rather on the degree of catarrh, particularly of the large intestine, than upon the ulceration. In one case where death occurred after perforation the patient, who was sixty-three years of age, had throughout such constipation that firm evacuations were secured on alternate days by enemas.

The characteristic stools of typhoid fever are of a light ochre-yellow color, thin, offensive, alkaline, and often ammoniacal. Their appearance often suggests a comparison with thin pea-soup. On standing they separate into two layers, the upper being thin and serous and containing albumin and soluble salts, and the lower being a flaky sediment consisting of remnants of food, blood-cells, epithelial débris, crystals of triple phosphates, and sometimes portions of sloughing tissue. This characteristic appearance of the evacuations is best seen after the middle of the second week. Before this the passages are more apt to be brownish in color. Sometimes the stools are frothy or pultaceous. Blood may be present in sufficient quantity to give them a very dark-red or almost black color. In some cases the stools are passed involuntarily.

Intestinal hæmorrhage of some degree is a symptom seen in from 3 to 7 per cent. of all cases, according to different estimates. It is always a cause of anxiety, and may prove a symptom of the greatest gravity. It may vary in amount from a few drops to a quantity sufficient to prove rapidly fatal. It occurs oftenest between the close of the second week and the beginning of the fourth week, but may be seen as early as the fifth or sixth day. The early hæmorrhage is due to intense congestion of the intestine or to disintegration of blood within the vessels, and is usually, though not always, of small amount. It appears sometimes to be the result of a hæmorrhagic diathesis, and is then accompanied by epistaxis and hæmatemesis. When due to disorganization of the blood it is apt to be combined with petechiæ and bloody urine. The bleeding later in the affection is produced by the opening of small blood-vessels as a result of the intestinal sloughing.

Hæmorrhage is most common in cases which have previously been severe and in which diarrhœa has been marked. The blood is bright red if passed at once, or dark and clotted or, perhaps, tarry if retained for several days. Sometimes extensive internal hæmorrhage takes place, and death occurs without blood having been voided at all. The symptoms of a large hæmorrhage usually come on unexpectedly. They consist of a sensation of sinking and faintness, great prostration, pallor, sudden reduction of temperature by several degrees and even to below normal, feeble pulse, coldness of the extremities, and a temporary improvement in the nervous symptoms. If death in collapse

do not occur, the temperature rises again within twenty-four hours, with a reappearance of the nervous symptoms as they existed before the accident.

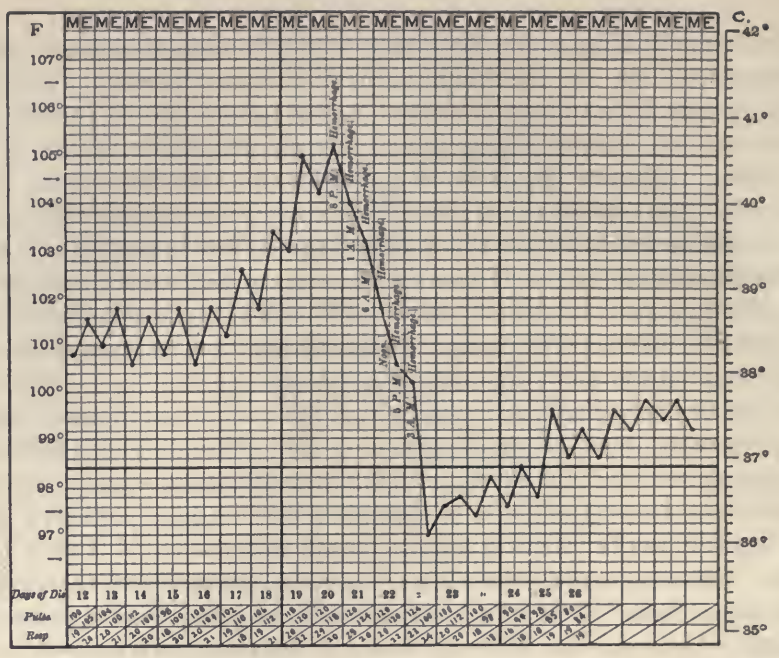
Occasionally I have known a marked rise of temperature to precede by a few hours the occurrence of hæmorrhage. In one case under the care of Dr. W. R. Batt, which was apparently doing well with a temperature not exceeding 102° F., fever increased on the eighteenth day, and on the twentieth day the temperature reached 105° F. Hæmorrhage occurred first on the twenty-first day, and within thirty-six hours there were six very large discharges of blood. The temperature fell gradually to 97° F. This was followed by high fever and the evidences of peritonitis. Convalescence was not completed until the one hundred and fourth day. (See Fig. 5).

It is obviously necessary to recognize two different forms of hæmorrhage in typhoid fever which are of widely different gravity, because, while Graves and Trousseau do not seem to have regarded it as a very dangerous symptom, many observers have found it followed by death in a very large proportion of cases. If the blood passed be bright in color and small in amount, and if there be no evidence of shock to the system nor any increasing distension of the abdomen due to accumulation of blood and development of gases, there is ground for hope that it has come from the large bowel as the result of a small follicular ulceration. I have seen many such instances where any alarm at the occurrence of a hæmorrhage of moderate amount has proved needless, since the favorable course of the case was in no way disturbed by it. So, too, even when the hæmorrhages are large and repeated and have induced most alarming collapse, so that life seems almost extinct, the case is not necessarily lost, since reaction may be secured and recovery follow a cessation of the discharges.

Enlargement of the spleen is present in most cases. It begins at about the middle of the first week, is greatest at about the end of the second week, and diminishes during the third and fourth weeks. It is often very considerable, and the organ may even reach three times its natural size. The enlarged spleen is smooth, not indurated, and is often slightly tender on pressure. It can usually be detected by careful palpation below the margin of the ribs from the earliest days of the disease. If, however, tympanitic distension develop, it often becomes impossible to feel the spleen even though decidedly enlarged. Increased area of splenic dulness can be demonstrated by percussion even more constantly, and it is only where tympanites grows extreme that careful light percussion fails to detect the enlarged organ.

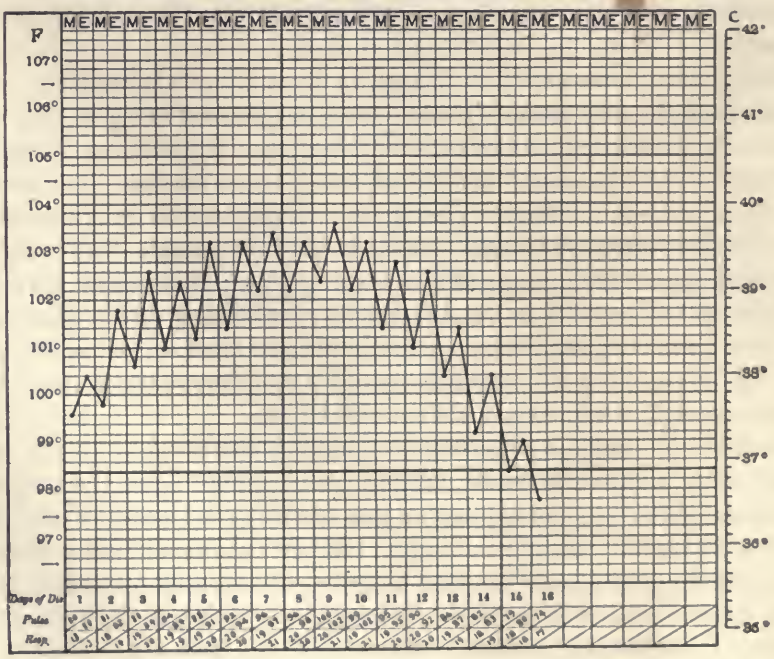
Of the *circulatory symptoms* there is nothing characteristic in the heart-sounds, except that as the disease advances in adynamic cases the quality of the first sound changes and approaches that of the second sound, while a faint systolic murmur may become audible. The true condition of the circulatory strength of the patient can often thus be best determined by constantly watching the nature and alterations of the first sound. Palpitation of the heart may result from the disturbance of the nervous system. In a case recently under my observation there were daily paroxysms at almost the same hour, attended

FIG. 5.



H. B——, male, æt. 34, case of moderate severity, without marked diarrhœa, began Dec. 6th. Temperature range moderate until Dec. 24th, when it began to rise, reaching 105° on Dec. 26th. Hæmorrhage on the 27th, and five others during next thirty-six hours, with fall of temperature to 97°. Administration of large doses of oil of turpentine, one ounce during the thirty-six hours of hæmorrhage. Delirium and unconsciousness for nine days. Final recovery. Discharged March 20th.

FIG. 8.



Temperature-chart of Case of Abortive Typhoid Fever.

by pallor of the face and followed quickly by intense determination of blood to the head and by orthopnœa.

The pulse is increased in frequency, and often in proportion to the elevation of temperature. It rises in the evening with the temperature and falls in the morning, but besides this it is subject to many variations from time to time, and is readily modified by slight influences. Its rate is generally somewhere between 100 and 120 per minute. A velocity above 120 indicates a decided condition of cardiac weakness. It is not infrequent, however, to find the pulse but little accelerated even when the temperature is high. This occurs more often in typhoid fever than in any other of the infectious febrile diseases. On the other hand, the pulse may be unduly frequent in mild cases when the temperature is but little elevated. It is at first full in volume and very often markedly dicrotic. This existence of dicrotism is particularly characteristic of typhoid fever. As the disease advances and weakness grows greater the frequency of the pulse increases. In severe cases it may often reach 140 or 150 a minute, and recovery yet ensue, though a rate as high as this renders the prognosis very grave. At the same time it loses in force and becomes compressible and small. If exhaustion progresses, it becomes yet faster, running and almost impereeptible. At the same time duskiness of the skin and coldness of the extremities indicate the great weakness of the circulation. This local coldness may exist even when the general temperature is high, and constitutes a decided danger signal. Collapse may rapidly develop from this condition. A sudden slowing and weakening of the pulse may also indicate a tendency to collapse. In a case to which reference will be made more particularly the pulse fell in a lad of seventeen years to 28 for a period of three or four hours, attended with subnormal temperature, 95° to 97° F., and with respiration of from 6 to 8 per minute.

As the patient approaches convalescence the pulse diminishes in rapidity, and after convalescence is established not infrequently becomes abnormally slow. This post-typhoid bradycardia need excite no anxiety, even when, as I have frequently observed, it continues during several weeks at a rate of 50 or 45. Cases are met with where the pulse falls as low as 35. I have seen this condition especially in hospital practice and in the cases of men of strong, muscular frame and phlegmatic temperament. Exertion will usually cause a marked rise in the pulse-rate. Indeed, it is not rare to find persistent rapidity of pulse continuing as the temperature falls, and even for some time after convalescence is otherwise complete. This may be the result of mere cardiac irritability, or may be caused by the slow disappearance of serious lesions of the cardiac muscle. It occasionally happens that when these latter have been severe the heart's action becomes so rapid and feeble on exertion as to necessitate long-continued rest and care.

The blood shows little alteration in the early stages of typhoid fever, but in the third week a decided diminution in the number of red blood-cells and in the percentage of hæmoglobin takes place. The number of leucocytes is not materially affected, and this fact, as Osler has pointed out, may be of value

in distinguishing the disease from septic and inflammatory processes in which leucocytosis occurs.

The *respiratory symptoms* always demand close watching. Bronchitis is nearly always present in greater or less degree, and varies remarkably from time to time during the course of the case. Although it may be said in general, therefore, that the rate of respiration advances with that of the pulse and of the temperature, we must be prepared for many variations which have no serious significance. Marked rapidity of breathing may sometimes be explained by the encroachment on the thoracic area from displacement of the diaphragm, due to tympanitic distension. Mere nervous influences may cause it also, and I have occasionally seen severe paroxysms of dyspnœa without corresponding disturbance of pulse in cases of the hysteroidal type. It is doubtless connected in some cases with the changes in the cardiac and respiratory muscles. It is not rare to find the breathing at 36 or 40 when the pulse is not above 85 or 95. Great rapidity of breathing may even be associated with slow pulse, an extreme instance of which, noted by Murchison, showed the respirations without discoverable pulmonary lesion to be 48, while the pulse was at the same time only 42. Abnormal slowness of respiration is less common. I attended in consultation with Dr. Mecray of Camden, N. J., a case in a lad of seventeen years, already referred to, where at the tenth day of a relapse there occurred on six successive days, beginning at midnight and lasting for three or four hours, an alarming fall in the respiration, as low as 6, 7, or 8 in the minute, and at the same time a drop in the pulse to 28 or 30, and in the temperature to 97° and even to 95½° F. Death was imminently impending, and was averted only by colossal doses of strychnine.

Analyses of the expired air have revealed nothing of importance save the presence of ammonia in the later stages of some cases.

Auscultation may reveal scattered bronchial râles from the onset of the case; indeed, the physical signs of bronchitis may be so marked as to divert attention from the constitutional nature of the disease. As a rule, however, they increase as the fever advances. In the second week and later harshness of respiratory murmur and sonorous, sibilant, and mucous râles may be expected, especially at the base posteriorly. Not rarely on turning the patient on his side or raising him to a sitting posture there will be heard in this region a fine, dry, crepitant expansion râle. This will disappear after a few breaths, dispelling the momentary fear of incipient pneumonia. Percussion resonance may be impaired slightly over the base, owing to imperfect expansion; but distinct dulness appears only in case of pneumonia or pleurisy. Bronchial catarrh and pulmonary congestion become so extensive and severe in some cases of severe adynamic type, and prove so obstinate, that in view of the attending irregular fever the fear of developing tuberculosis may be entertained. Cough is a very irregular symptom. It is sometimes severe and harassing in the early stages, but later may be comparatively slight, even when examination of the chest shows marked catarrh and congestion.

The *urine* is usually diminished in quantity, high-colored, and of increased

specific gravity in the earlier stages of the disease. The diminution may persist until convalescence, or the urine may become light-colored and be excreted in larger quantities about the end of the second week. As convalescence is more nearly approached, and during it, the specific gravity falls very considerably, the reaction is feebly acid or is alkaline, and a quantity of urine decidedly greater than normal is passed. The amount of urea excreted is increased in the early stages, and often throughout the attack. It is not affected by the existence of diarrhœa, but may be reduced by the occurrence of an inflammatory complication. It is frequently diminished during convalescence. Uric acid is increased while the attack is in progress, but diminished during convalescence, while the reverse is true of the chlorides. A febrile albuminuria is very common, and, as will be seen later, the complication of infectious nephritis is not rare. The diazo-reaction described by Ehrlich depends upon the existence in the urine in typhoid fever of certain aromatic bodies which are capable of producing definite color reactions with the diazo-compounds. Ehrlich considered the reaction characteristic of typhoid fever. It is true that it is very commonly present in this disease, but it may also frequently be observed in tubercular meningitis and in some other conditions. It is, for example, rarely absent in measles. To employ the test two solutions are kept—one a ½ per cent. solution of sodium nitrite, the other a ½ per cent. solution of hydrochloric acid saturated with sulphanilic acid. Just before using, 40 parts of the first are mixed with 1 part of the second. The hydrochloric acid acts upon the sodium nitrite and liberates nascent nitrous acid, and this, acting upon the sulphanilic acid, produces diazo-benzene-sulphonic acid. Equal parts of the mixture of the two solutions and of urine are now thoroughly shaken in a test-tube and overlaid with ammonia. If the reaction develops, a deep-red ring forms at the junction. The color varies from a carmine to a deep garnet. In normal urine the ring which forms has no tinge of red.

In very severe cases of typhoid fever, with unconsciousness, the urine is passed involuntarily. Retention of urine is often an early symptom and demands catheterization. As soon as marked hebetude appears the region of the bladder should be percussed daily, as partial retention may occur even when there is occasional discharge from overflow.

Complications and Sequelæ.—Many of the conditions already described as symptoms or as pathological lesions might with equal propriety be considered among the very numerous and varied complications and sequels of the disease.

Of the complications involving the dermal, muscular, and osseous systems, bed-sores deserve first mention, as they are frequent and troublesome in severe cases. They depend upon the imperfect nutrition of the skin, the emaciation, the constant pressure over bony prominences, and, in the case of the nates, the great difficulty in keeping the parts perfectly clean and dry. Patients may die from the exhaustion caused by bed-sores after having survived the fever. The only way to avoid them is by daily careful examination of all dependent parts and by the prompt adoption of preventive measures.

Atrophic lines (lineæ albicantes) may develop in the skin during convalescence, especially in children and young adults. I agree with Bouchard that they are, at least usually, the result of stretching due to the rapid growth after the fever has ceased. A very similar condition was described by Wilkes as atrophic in nature. Not infrequently abscesses of the skin, subcutaneous tissue, or muscles form.

Herpes labialis is conspicuous by its absence. Its presence, in fact, offers a strong presumption against the diagnosis of typhoid fever. It is to be borne in mind, however, that it may sometimes occur in this disease, though with nothing like the frequency with which it occurs in pneumonia, malaria, and cerebro-spinal fever.

Temporary falling of the hair is a very common sequel, but permanent baldness is rare. The new hair often lacks lustre at first. It occasionally happens that curly hair has grown in cases in which it was previously straight, but this condition need not be permanent.

The nails often exhibit transverse markings after recovery, indicating the impairment of nutrition which existed during the attack.

Rupture of muscles, often followed by hæmorrhage into them, occasionally happens.

Periostitis of different bones, but especially of the tibia, is an occasional sequel. It may subside or may go on to necrosis. Keen has collected the records of 37 cases of necrosis of the tibia following typhoid fever. In a series of cases I have observed obstinate periostitis of the sternum or of the crests of the ilia, or in two instances, judging from the location of the pain and from the effect of movement of the trunk, of the front of the spinal column. Swelling and even suppuration of the joints are sometimes seen.

A tendency to grow stout, temporarily or permanently, is a not infrequent sequel to typhoid fever. On the other hand, patients may remain permanently leaner than before, and never regain robust strength.

Meningitis and cerebral hæmorrhage have already been referred to in the remarks on Morbid Anatomy as rare complications. It is essential to appreciate, however, that the nervous symptoms of typhoid fever, though they may be very grave, are seldom connected with any actual organic lesions of the membranes or substance of the nerve-centres. When meningitis does occur, it may result from suppuration in the temporal bone, or it may be pyæmic or tuberculous in nature. It is not to be denied also that in rare instances a certain degree of meningitis is set up as a part of the special typhoid lesions. The nutrition of the brain and cord often suffers severely, however, from the prolonged fever, systemic infection, and sustained reflex irritation from the local lesions.

Mental defects, varying from mere impairment of intellect to a high grade of confusional insanity, may appear during convalescence. At times the patient emerges from the fever with mental weakness and poor memory, which persist for weeks or even for months. Or, again, when convalescence seems to be advancing normally a true post-typhoid insanity develops. There

are confusion of ideas and inability to recognize friends or familiar sights. Hallucinations are not rare, and mild or even maniacal delirium may occur. In the great majority of cases recovery follows, though the symptoms may be alarming and obstinate.

Slowness of speech is at times present after typhoid fever, and temporary aphasia may occur, especially in young children. In rare instances I have known epileptic convulsions to follow, and hystero-epilepsy has also been reported as a sequel.

The organs of special sense sometimes exhibit alterations. Otorrhœa is not infrequent, especially in children. Sloughing of the cornea may rarely occur, especially if coma vigil exist. Temporary blindness not depending upon this has been observed.

Neuralgias and hyperæsthesias of any kind are not common sequels.

Paralysis of different forms is seen as a complication or sequel. It generally does not develop until several weeks after convalescence begins, although it sometimes comes on during the height of the disease or even at its commencement. It usually depends on a neuritis, and almost any nerve may be attacked. The paralysis may be limited to a single nerve, or it may assume a paraplegic or even hemiplegic form. Recovery generally ensues after weeks or months. Some of these cases may be due to a poliomyelitis, or a sudden hemiplegia may possibly in some instances be the result of a thrombosis or an embolism.

Muscular tremor and chorea are occasional sequels.

Among the complications of the digestive system may sometimes be observed ulceration of the tongue and of the mucous membrane of the cheeks. Cancrum oris has been reported in a few cases, and aphthous stomatitis also, although it is rare and is seen only in very debilitated conditions or in patients treated in unhygienic surroundings. Alveolar abscess is liable to develop. Diphtheritic inflammation of the pharynx and œsophagus is a dangerous complication not rarely met with. Dysphagia may be due to this, to a paresis of the muscles of deglutition, to a cellulitis of the neck, or, especially in children, to pharyngeal hyperæsthesia.

Parotitis, usually unilateral and suppurative, is an occasional and dangerous complication. It generally begins during the third or fourth week, and is seen, as a rule, only in severe cases. Both glands may be involved at once, or first one and then the other. Suppuration is very apt to occur, in which respect this secondary form of parotitis differs widely from mumps. Liebermeister states that parotitis has become much less common since the introduction of antipyretic methods.

Profuse gastric hæmorrhage, which may terminate fatally, has been observed, as in a case reported by Weiss. Doubtless in this case the hæmorrhage was caused by gastric ulcer. Care must be taken not to confound with this the vomiting of blood which has flowed down from the posterior nares. On several occasions I have been greatly alarmed until the source of the hæmorrhage was discovered.

Dysentery, sometimes diphtheritic, occasionally exists as a complication or sequel. Gangrene of the intestinal mucous membrane may even take place.

Jaundice only rarely complicates typhoid fever. It may result from a catarrhal process or from parenchymatous changes in the liver which can attain such a degree that the characteristic symptoms of acute yellow atrophy appear. The liver is occasionally enlarged. Hepatic abscess is a rare sequel. Diphtheritic or ulcerative processes may occur in the gall-bladder.

Perforation of the intestine by an ulcer is the most dangerous complication which can arise. It occurs in about 2 to 3 per cent. of all cases. The 4680 cases tabulated by Fitz give a mortality from perforation somewhat higher than this—viz. 6.58 per cent. The accident forms, according to Murchison, about 11 per cent. of the causes of death in typhoid fever. In the 2000 Munich autopsies perforation constituted 5.7 per cent. of the causes of death. It is very frequently preceded by hæmorrhage. It takes place most often in the severer cases, especially in those in which other abdominal symptoms, as diarrhœa and tympanites, have been marked. At the same time, must ever be borne in mind the important fact that it sometimes occurs in the mildest of cases which have exhibited no abdominal symptoms. Fitz found it much more frequent in men than in women, and rarer in children than in adult life. It is commonest toward the end of the second week and in the third and fourth weeks, but it may occur later than this, and it has been met with as early as the eighth day. There are numerous instances on record in which perforation has taken place some weeks after convalescence had commenced, the patients being out of bed and even at work. Among the immediate causes of the accident may be mentioned the presence of hardened fæcal masses, undigested food, excessive tympanites, severe vomiting, the increased peristalsis caused by purgative medicines or by an enema, ascarides, straining at stool, sudden changes in position. A perforation which occurs early in the attack is probably due to the separation of a slough, while that which comes later is probably the result of an extension of the ulcerative process to the visceral peritoneum. The opening through the intestine is generally small, with clean-cut edges, and the slough may still be present and cover it, or may have entirely disappeared. The symptoms attending perforation come on abruptly. They consist of very severe abdominal pain, which develops in the right iliac fossa and rapidly spreads over the whole abdomen, and of profound collapse, the latter evidenced by feeble running pulse, cold sweat, subnormal temperature, feeble respiration, great thirst, suppression of urine, and frequent vomiting. Death may take place in a few hours. If it does not, the symptoms of acute diffuse peritonitis soon set in, the abdomen becoming more tympanitic and the liver dulness being obliterated. The latter symptom constitutes a valuable diagnostic sign. The abdomen grows excessively tender, the face wears an expression of intense suffering, the legs are drawn up, and the temperature rises again. Death takes place in two to four days, or sometimes after a longer time.

The opinion was formerly held that perforation was inevitably fatal, but there is abundant evidence that recovery may occur in rare instances. Thus

the perforation may at times produce only a localized peritonitis, terminating in abscess, which may be discharged by the bowel or externally, and recovery follow; or the bowel at the seat of a minute perforation may be so firmly glued by an adhesive inflammation to the wall of the abdomen or to another loop of intestine that little or no escape of intestinal contents can take place.

Peritonitis, local or diffuse, resulting from causes other than perforation, may complicate typhoid fever. This may be produced by the spreading of inflammation from the ulcerating mucous lining to the serous layer of the intestine, without perforation existing; or it may be the result of the rupture of a softened mesenteric gland or of the bursting into the peritoneum of an abscess of the spleen, liver, gall-bladder, urinary bladder, or abdominal wall; or it may follow causes entirely independent of the febrile disease. I have known death to occur from general peritonitis, with abundant purulent and plastic exudation, as early as the tenth day, without perforation and without any evidence to connect its origin with any particular ulcer in the intestine.

Venous thrombosis is the most frequent complication from the side of the circulatory system. It is oftenest met with in the femoral vein, where it is of very common occurrence, producing œdema and pain. It happens much oftener in the left leg than in the right, possibly due to the fact that the left iliac vein is crossed and pressed upon by the right iliac artery; not rarely the other leg is subsequently affected. It may be a complication, but is oftener a sequel, coming on after convalescence seems established. Its onset is marked by pain in the groin or thigh or calf. There is tenderness on pressure along the femoral vein, which can soon be felt to be swollen or hard. Pain is also complained of if pressure be made upon the calf. The swelling of the leg which follows is often considerable, and is more elastic and pits less readily than in ordinary œdema. It indicates that the lymph-channels, as well as the vein, are involved. The leg is heavy and entirely powerless. Irregular fever of moderate grade is kept up for some days, and may at first cause apprehension of a relapse. Recovery nearly always takes place, owing to the late period in the case when this sequel occurs. Convalescence is, however, protracted; the swelling subsides gradually as the collateral circulation is established, but some slight enlargement of the affected leg may remain permanently. In very rare instances the thrombus may become dislodged and be carried to the heart with fatal result, or septicæmia may ensue upon suppurative softening of the clot.

Obliteration of the larger or smaller arteries by embolism or thrombosis is an infrequent complication. Gangrene of the part from which the blood is cut off naturally follows. Arteries supplying any of the skeletal or visceral portions of the body may be involved, but the femoral artery is the one in which the condition most frequently develops.

Pericarditis and endocarditis are unusual complications, while myocarditis, with consequent dilatation of the cavities, is more frequent. Valvular disease is a rare sequel. Graves' disease has also been known to develop. A post-typhoid anæmia is occasionally observed, and the diminution of the percentage

FIG. 6.

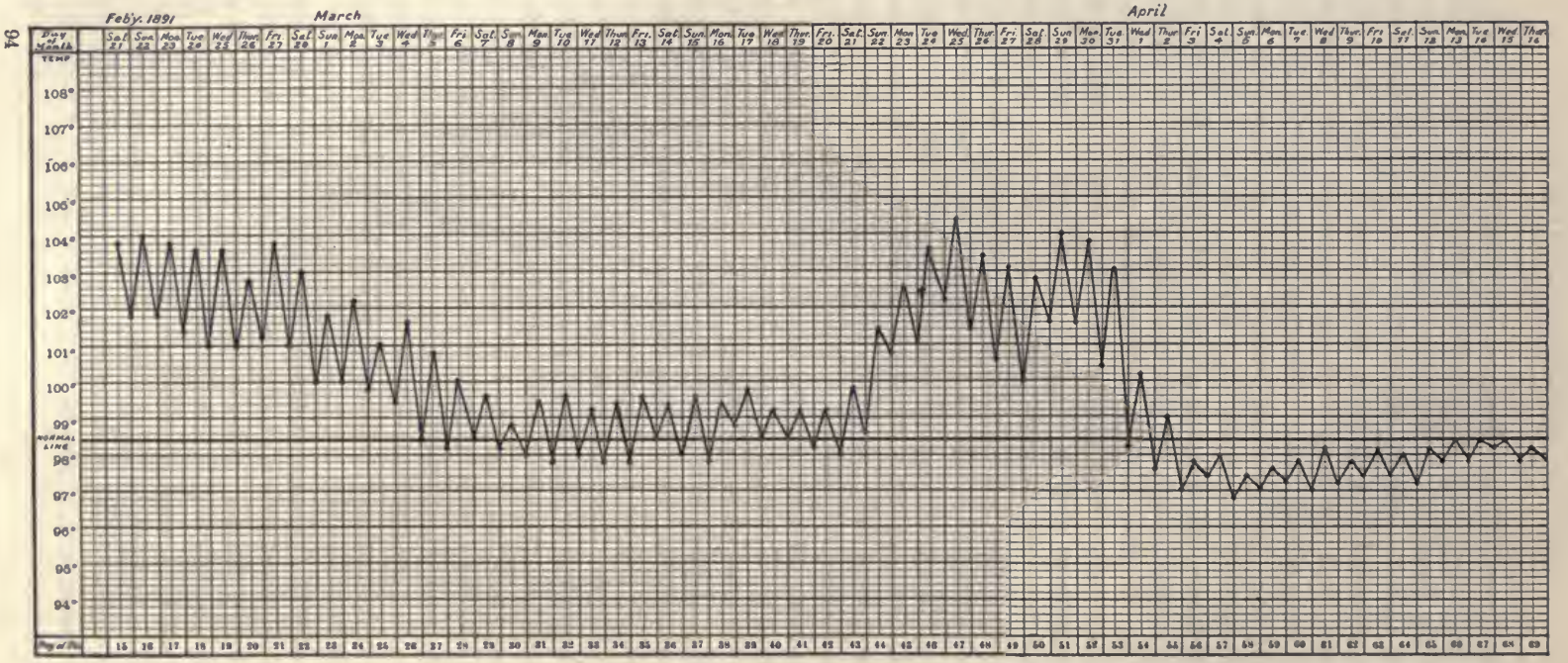


Chart showing the fever curve, beginning about the second week of the disease, with subsequent pseudo-relapse, due to femoral phlebitis.

of red blood-cells and of hæmoglobin may be very great. Ollivier reports the development of chlorosis as among the sequels. The disease may be complicated by the hæmorrhagic diathesis, though this tendency to bleed may exist only under the influence of and during the disease. This condition is not very rare, and the hæmorrhage may occur as petechiæ or as excessive epistaxis, or may take place from the gums, the stomach, or the kidneys.

Spontaneous rupture of the spleen is a very rare complication of typhoid fever. A more common, though still unusual, accident is the softening and rupture of a splenic infarct. Mention has already been made of the great enlargement of the mesenteric glands which sometimes takes place, and may lead to abscess. The other lymphatic glands are rarely involved.

In the domain of the respiratory system it may be noted that, although redness and swelling of the nasal mucous membrane are common during the disease, coryza is a rare sequel. Necrosis of the cartilages of the nose is also occasionally, but rarely, observed.

Laryngitis sometimes exists either as a complication or as a sequel. Laryngeal ulceration or perichondritis may develop, though this is generally considered rare. Œdema of the glottis is apt to be produced by the laryngeal implication, though it may occur independently of it. Hölscher reports tracheotomy done 15 times for perichondritis in 2000 fatal cases of typhoid fever.

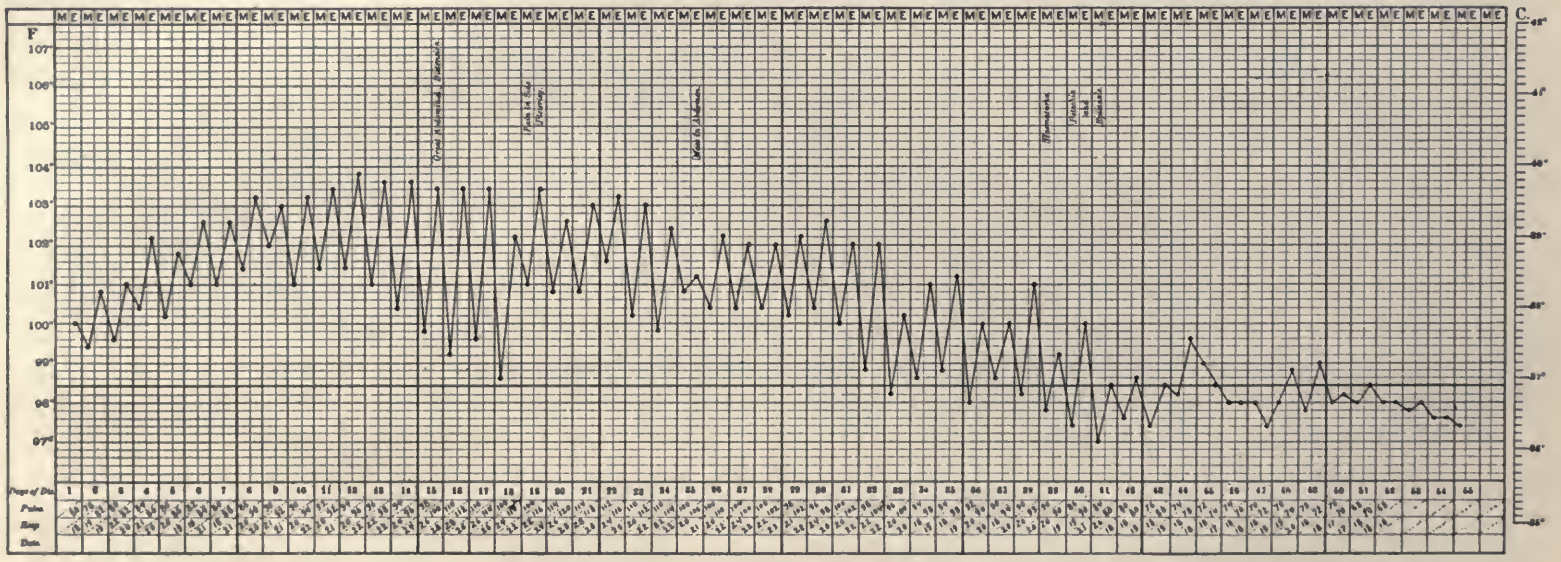
Bronchitis has been described among the symptoms. Both lobular pneumonia and collapse of portions of the lung may be consecutive to it.

Lobar pneumonia is a common complication. It occasionally develops early, even as an initial symptom, and in such cases the diagnosis of primary pneumonia might easily be wrongly made. (See Varieties of Typhoid Fever.) Much more frequently pneumonia develops in the second or third week, or even after convalescence has commenced. It is generally unattended by rusty expectoration or increase of cough, and may readily be overlooked. The temperature curve is apt to be highly irregular. In a fatal case which I saw in consultation with Dr. W. H. Warder, where pneumonia occurred from exposure in early convalescence, the temperature gradually rose for several days, and then assumed a paroxysmal type, with morning fall to 99° and evening rise to 106° for three days before death.

Pulmonary œdema and hypostatic congestion of the lungs are of very frequent occurrence in the later stages of the disease. They result from failure of the circulation and the constant recumbent position of the patient. Hæmorrhagic infarct sometimes develops, and gangrene or abscess may result from this or from lobular or lobar pneumonia.

Pleurisy with effusion is a serious but rather rare occurrence. When it arises acutely during the course of the disease it may be sero-plastic and terminate in gradual absorption, but when it develops slowly as a sequel it is nearly always purulent. The accompanying chart (see Fig. 7) of the temperature in the case referred to on page 66 is interesting as illustrating the extreme irregularities introduced by the occurrence of serious complications. Pneumothorax is a rare complication, and hæmoptysis is occasionally seen.

FIG. 7.



Temperature-chart of Typhoid Fever in a boy æt. 15. A typical curve after fourteenth day of disease, with excessive diurnal variations, due to grave complications: extreme tympany, pleurisy, hæmaturia. Recovery after desperate illness.

Acute miliary tuberculosis may be developed during or after typhoid fever. This, however, must be of extremely rare occurrence, and it is not improbable that some of the reported cases were errors of diagnosis by which tuberculosis was regarded as typhoid. The general opinion that persons recovering from typhoid are particularly liable to develop phthisis does not seem supported by adequate evidence.

Febrile albuminuria without casts is common in typhoid, as already stated, and does not materially add to the gravity of the case. Acute nephritis may develop, however, at the beginning or during the course of the disease, when the urine becomes scanty and contains albumin, casts, epithelium, or blood. The affection is a severe one, and the patient may die of uræmia. Undoubtedly, the typical typhoid state is often induced in part by this renal complication. The nephritis which comes on as a sequel after convalescence has commenced is attended by œdema and the usual symptoms of acute Bright's disease. It is often followed by recovery. Sugar in minute amount is occasionally found in the urine during the course of the fever, and diabetes apparently occurs as a rare sequel. Hæmaturia is a rare complication, and indicates a dangerous hæmorrhagic tendency.

Vesical catarrh is not infrequent, especially after cases where retention of urine was present and required catheterization. It may be slight or severe, transitory or obstinate and troublesome. More rarely pyelitis follows typhoid fever, and may even be attended with ulceration and membranous exudation. Among other rare sequels may be mentioned orchitis, inflammation of the ovaries, and gangrene of the genitals, which latter may occur in both sexes.

Menstruation is often irregular during the attack. It may occur prematurely and be profuse, or it may fail to appear. Amenorrhœa, lasting several months, is a frequent sequel.

Pregnancy may possibly give some degree of immunity from typhoid fever, but does not protect absolutely, as was formerly supposed. Abortion is very apt to take place, especially if the disease be contracted in the first half of pregnancy. It occurs oftenest during the later periods of the disease.

The existence of typhoid fever does not protect the system from possible invasion by other infections. Erysipelas may develop during the height of the disease, or more frequently as a sequel, but its occurrence is rare. Scarlatina has been repeatedly observed in those suffering with typhoid fever, and there are other reported instances in which the reverse has occurred. In fact, Murchison believes that scarlatina predisposes to the development of typhoid fever. It must not be forgotten that an erythema may appear early in typhoid and lead to error in diagnosis. Rubeola, variola, vaccinia, influenza, typhus fever, and pertussis may present themselves in combination with typhoid fever. Karlinsky reports a case complicated by anthrax. Diphtheria has been repeatedly observed in combination with typhoid fever, especially in children.

Malarial-typhoid (*Typho-malarial*) *Fever.*—The term "typho-malarial fever," first proposed by Woodward, is misleading and should be abandoned. It has been forced to designate two distinct classes of diseases, to neither of

which it is strictly appropriate. The first class consists of cases of severe malarial fever of the remittent type, in which the patient passes into a typhoid state. To these cases the term "typhoid remittent" is properly applicable, just as we say typhoid pneumonia or typhoid dysentery in the corresponding form of these diseases. The second class is a true combination in the system of the poisons of malarial and typhoid fevers, and this type of disease can be correctly styled "malarial typhoid." There is abundant evidence, both clinical and pathological, to show that this combination of the two diseases can exist, although it is doubtless true that most of the reported cases of so-called typho-malarial fever, bilious remittent fever, mountain fever, etc. were either purely malarial or purely typhoid in nature, but with some anomalous features, such as have already been described.

The symptoms of the combination of malarial and typhoid fevers are variable, depending upon which disease predominates. The attack is apt to begin as an intermittent fever, the paroxysms constantly growing more intense and prolonged, and the fever thus gradually approaching a continuous type. The remissions or intermissions are great, sudden, irregular, and attended by profuse sweating and decided adynamia. The evening elevations of temperature are more pronounced than in ordinary typhoid fever, and the headache is apt to be intense, while hebetude and delirium are usually less marked. Gastric and hepatic derangements are common, and there is painful enlargement of the liver and spleen. Quinine has no specific effect upon the disease. Microscopic examination of the blood should show the presence of the malarial organisms.

Varieties of Typhoid Fever.—Typhoid fever may exhibit variations either in its mode of onset or in the fully-developed disease. These may consist in the exaggeration of certain symptoms, or in a tendency to involvement of certain organs to an unusual degree, or in peculiarities in the general character or severity of the disease as a whole. The disorder is so complex that very many forms have been described. There may, in the first place, be decided variations in the mode of attack.

In the *onset with marked nervous symptoms* headache may early be exceedingly severe and resist all treatment. In some cases this is combined with stiffness of the neck, retraction of the head, photophobia, muscular twitching or even convulsions; and under such circumstances the disease is likely to be mistaken for meningitis.

In the *onset with marked pulmonary symptoms* bronchitis may be the first symptom noticed, and for several days the case may be regarded simply as one of severe bronchial catarrh. Occasionally the disease begins with chill and the symptoms of croupous pneumonia, and there may be nothing which justifies the diagnosis of any other disease than this until after a week or more, when the failure of the crisis to occur, the development of rose-colored spots and of intestinal symptoms, and the general typhoid condition of the patient may show that the pneumonia was but an early complication of a general infectious disease. In some cases, indeed, it may remain throughout impossible to deter-

mine whether we have to do with a typhoid pneumonia—*i. e.* with a pneumonia with typhoid symptoms—or with a true typhoid fever with initial and predominating pneumonic symptoms and localization.

Much less frequently similar doubt may be caused by the early and intense development of pleurisy at the onset of typhoid fever. The terms "pneumo-typhoid" and "pleuro-typhoid" have been used to indicate such cases, and the name "laryngo-typhoid" is given by Schuster to the even more rare cases where the onset of typhoid fever is masked by the great prominence of an initial laryngeal complication. The advantage of these special terms seems very doubtful.

In the *onset with marked gastro-intestinal symptoms* vomiting and diarrhœa may occur so early and be so resistant to treatment that the disease simulates corrosive poisoning. Chomel, indeed, must have met with epidemics presenting a large proportion of such cases, since he claimed that this was the most frequent mode of attack. In some instances jaundice has also been present, and such cases are not rarely regarded as bilious remittent fever.

In the *onset with marked renal symptoms* the condition of the urine and the other symptoms at first so closely resemble those of acute Bright's disease that there may even be danger of regarding these cases as instances of primary nephritis.

This early prominence of certain symptoms may disappear soon, or may continue into the fully-developed attack, thus determining the later type of the disease also.

Apart from the early predominance of one class of symptoms, variation of the whole course of the disease may constitute certain definite forms.

In the *abortive form* the attack begins often rather suddenly with chilliness. By the third or fourth day the temperature reaches 103° to 104° F. All the characteristic evidences of the disease are present: the spleen is enlarged and the gastric symptoms are often well marked. In my own experience diarrhœa and tympanites are less pronounced. The rose spots may develop early, even by the second to the fifth day, but it is always to be considered whether several days of unobserved indisposition have not preceded the apparent onset. Between the seventh and the fourteenth days all the symptoms rapidly improve. The temperature falls by rapid lysis, or a genuine crisis with copious perspiration may occur. (See Fig. 8.) Convalescence is rapid. According to Chantemesse, fresh crops of eruption may develop even after the fall of temperature, indicating that, although the fever has gone, the infectious process is not necessarily ended, so that watchful and rigid care should be enforced for some time longer.

It is obvious that the diagnosis of such attacks must rest chiefly on the detection of the characteristic eruption. No good reason appears why the abortive form should be less frequent in this country than elsewhere. It is not improbable that in many cases the course of the disease is really longer by several days than would appear, both by the existence of almost latent symptoms before the onset and by the persistence for some days of the infectious

process after the disappearance of the fever. Upon the whole, I incline to think that abortive typhoid fever is more common in America than is generally admitted. A more critical study should be made of each acute febrile case, and a lesson of greater caution in treatment be learned. The peculiarities of these abortive cases may depend upon a modified virulence of the virus, or more probably upon a greater resistance on the part of the tissues and system, with comparatively slight intestinal lesions and secondary fever.

The *mild form of typhoid* is of more importance on account of its frequent occurrence. As a rule, the course and duration correspond to those of the ordinary type, save that all the symptoms are on a subdued and moderate scale. Naturally, a considerable proportion of the short abortive cases above described are also included under this heading. The onset is usually gradual, though sometimes quite severe initial symptoms speedily subside and give place to a mild type of the fever. The temperature shows but moderate daily variation, and its maximum should not exceed 101° or 102° F., though a few brief rises to 103° or 104° F. may occur. The general condition of the patient is excellent, and the nervous, pulmonary, and abdominal symptoms are especially mild. Epistaxis often occurs, the spleen is enlarged, and the eruption appears as usual, and is almost as likely to be abundant as in more severe cases. Were it not for these characteristic symptoms, the case might be regarded as one of simple continued fever.

It must never be forgotten that even in cases of such mild type there is danger of the sudden development of serious complications. Profuse intestinal hæmorrhage, or even perforation, may occur when all the previous symptoms have been of such slight grade as scarcely to warrant the least anxiety.

Sporadic cases are more apt to be mild than those which form part of a decided local outbreak. It happens, however, that in some seasons, even when the disease is highly prevalent, a large proportion of all the cases assume this mild and favorable type. While, therefore, in individual instances the type may depend upon want of susceptibility, there must be at times associated a virus of less than usual energy. I would here repeat the caution that no small number of anomalous febrile disturbances might be found on careful study to be, in fact, typhoid fever of very mild or abortive form.

Closely associated with the preceding varieties is what is often described as the *afebrile form*. A severe epidemic of this nature occurred in the German army besieging Paris in 1870. The rose-colored spots were abundant, there were great prostration, slight abdominal symptoms, and violent delirium alternating with stupor. Though in some instances there was an elevation of temperature lasting not more than two weeks, in many others the temperature was normal, or even subnormal. All the cases which died exhibited the characteristic lesions.

Afebrile typhoid of a much less severe form is reported by Liebermeister as of frequent occurrence in Basle. The patients suffered from lassitude,

depression, headache, pains throughout the body, loss of appetite, coated and swollen tongue, slow pulse, constipation or diarrhœa, and, in many cases, enlargement of the spleen and rose-colored spots. They were often confined to bed for four weeks or longer; yet in most cases no elevation of temperature whatever was detected during the course of the attack. In some instances a fever of 100.5° F. was occasionally noted. In the afebrile cases which I have seen the type of the disease has been mild, and there has been a subnormal temperature at some period of each twenty-four hours, so that the diurnal variation was unduly great.

The *latent* or *ambulatory form* of typhoid fever, or "walking typhoid," is only a variety of the mild form of the disease. It is marked by an absence, during the early stages at least, of the decided sense of debility which leads the patient in ordinary cases to retire to bed soon after the invasion. In this form it is not unusual to find the patient walking about, or even attempting to pursue his occupation, until well into the second week. This may be the case although considerable fever, abundant eruption, and some diarrhœa may be present. Cases of this type are met with most commonly among the working-classes, and especially among males of phlegmatic temperament. I have, however, seen not a few instances among women engaged in domestic service. There is an almost total absence of nervous symptoms, and this, joined with the fact that such persons are rarely accustomed to note carefully or to attend promptly to slight disturbances of health, may help to explain the marked peculiarity of these cases. They are more generally met with in hospital practice; and it is a familiar thing in every large dispensary service to find patients applying with complaints of diarrhœa or cramps or dyspepsia who on examination are found to have a temperature of 102° or 103° F., with characteristic eruption, enlarged spleen, and bronchial catarrh. These patients, when put to bed, often develop symptoms of a more severe type, especially if they have travelled far. Sudden delirium, profuse intestinal hæmorrhage, or even perforation of the intestine, may be the first indication of the serious nature of the illness. I have known several cases in which the first complaint made by the patient was only after intestinal perforation had occurred, and when, after death in the course of twenty-four or forty-eight hours, examination showed advanced lesions corresponding to at least the condition at the close of the second week. The evil results which follow mental or bodily effort during the early days of typhoid fever are often conspicuously seen in these cases. It is probable that were rest and suitable care secured at the onset they would habitually run a mild course.

The *grave forms* of the disease may be characterized by the severity of the symptoms in general, dependent upon the intensity of the poison, or may be marked by the severity of certain groups of symptoms which attain an intensity sufficient to warrant their classification as complications. Where certain symptoms are thus especially prominent during the attack, the disease may be again divided into a variety of other minor forms, depending upon the nature of the prominently dangerous symptoms. A too minute sub-

division, however, only complicates the subject, and but a few varieties will therefore be briefly referred to.

The disease in the early stages of the grave form may exhibit no specially alarming features, the serious aspect of the case developing during the second week. On the other hand, the symptoms may from the beginning be especially urgent and violent. In most grave cases the fever runs very high and is attended by severe nervous symptoms. Delirium is active and continuous, and sleep is secured with difficulty, or there may be an early tendency to deep stupor. When delirium is marked, and with great muscular twitching or even convulsions, the ataxic or *cerebro-spinal* form is spoken of. Where prostration begins early and is intense, and is accompanied by great rapidity of the pulse, the *adynamic* form is present. The *hæmorrhagic* form —a very grave one—exhibits a special tendency to the occurrence of hæmorrhage from the various mucous membranes and into the subcutaneous cellular tissue, and indicates serious alteration of the blood.

In some grave cases the nervous system may be comparatively unaffected, while alarming abdominal symptoms appear, such as extreme distension or profuse and uncontrollable diarrhœa (*abdominal* form). In still other cases the respiratory symptoms are very pronounced from the start (*thoracic* form), and the intense bronchial congestion and catarrh pass into pneumonia with rapidly-failing heart-power; or the disease may begin with pneumonia, as already stated.

A variety of grave typhoid fever has been described as the *renal* form. In this, already referred to in discussing the variations in onset, the urine early exhibits albumin, blood, and casts. Cases have been observed which simulate acute nephritis, and in one instance reported by Thue even the autopsy did not render the diagnosis certain until a bacteriological examination had been made.

Other forms of typhoid fever, described by some writers, are the *gastric* or *bilious;* the *mucous,* some cases of which are probably identical with the last, while others are to be classed under abortive typhoid; *spleno-typhoid,* in which the spleen is greatly enlarged and the symptoms closely resemble those of relapsing fever; and the *sudoral* form, characterized by profuse sweating, particularly at such periods in the day that the probability of the disease being malarial is suggested.

Finally, there is what may be called the *malignant* form of typhoid fever, called also the *septicæmic* form. The quality of malignancy does not exhibit itself in this disease nearly so often as in some other infectious diseases, such as typhus, scarlet, and cerebro-spinal fevers. In severe local outbreaks of the disease, however, there may be a small proportion of cases which, from the very outset, are of a malignant character. The onset is violent, the fever rapidly rising to a high point and differing widely in this respect from the ordinary mode of development; symptoms of profound nervous disturbance, such as stupor, active delirium, or even convulsions, make their appearance early; the mouth and tongue quickly become coated with copious dark sordes; there is deep discoloration of the skin, forming at dependent parts, especially

where pressure is exerted, and marked hypostatic congestion of the lungs occurs. If life be prolonged until the appearance of the eruption, the spots may be unusually dark and petechiæ may develop. Such cases are generally fatal, and it is not uncommon for death to occur by the seventh or tenth day or even earlier.

It is important to observe, before leaving the subject, the influence of the period of life in determining the whole course of the disease.

Typhoid fever in infants and children formerly passed under the name of infantile remittent fever, on account of the erroneous belief that typhoid fever did not occur at so early an age. So far from this being the truth, the disease is very common in early life, though it is usually less severe and often of shorter duration. The temperature generally rises more rapidly and is more apt to assume a remittent type, and to fall by crisis. The eruption is often absent or slight, and epistaxis is rare. The pulse is more rapid, but not so dicrotic. Intestinal symptoms are wanting or slight, but vomiting is common, at least at the outset of the disease. Bronchitis is frequent, though catarrhal pneumonia is not a common complication. Nervous symptoms are variable: in some cases convulsions occur at the onset or may be developed by an indiscretion in diet or treatment, and delirium and stupor are marked. In other instances there is even but little hebetude throughout. Intestinal hæmorrhage or perforation is rare. I have, however, met with fatal general peritonitis without perforation in a child of five years. The mortality is, upon the whole, very small.

Typhoid fever in advanced life or after the age of fifty years becomes much more serious than at earlier periods. The onset is often insidious. The fever does not run high as a rule, but is prolonged, and during convalescence the temperature is liable to fall below normal, with a tendency to collapse. The eruption is less constant, as are also epistaxis and severe diarrhœa. The nervous symptoms assume the adynamic and ataxic types. Violent delirium is uncommon, but great prostration, tremor and subsultus, increasing dulness and stupor, retention of urine or involutary evacuations, are frequent. The tongue grows dry, hard, and brown, and it often becomes very difficult to nourish the patient. Hypostatic pneumonia and nephritis are frequent complications. It is often difficult to decide whether the case be actually one of typhoid fever or of pneumonia of a low grade with typhoid symptoms; of cerebral thrombosis or meningitis with development of similar symptoms; or of uræmia.

Relapse and Recurrence.—The term "relapse" is employed to designate a renewal and repetition of a morbid process occurring before health is re-established after the primary attack. It is to be clearly distinguished, therefore, on the one hand, from any mere sequel or accidental affection arising during convalescence, and, on the other, from a recurrence, which is a true second attack of the disease, separated from the original attack by a more or less considerable period of health.

One of the most remarkable features of typhoid fever is the frequency with

which a relapse of the disease occurs. It is difficult to determine in what proportion of cases this takes place. Murchison estimated it at as low as 3 per cent.; Gerhardt noted relapse in 6.3 per cent. of 4000 cases; Jaccoud and Schmidt found it in about 9 per cent. of their cases. It is evident that the tendency to relapse varies in different seasons and localities, so that the proportion may be as low as 1 per cent. or as high as 10 per cent. or 15 per cent., or even more.

It has already been seen that during the convalescence of typhoid fever sudden and brief elevations of temperature are likely to follow fatigue, excitement, or errors in diet. These are styled recrudescences. Anxiety is always felt when this takes place, lest it may be the beginning of a relapse, but the subsidence of the fever in a day or two allays alarm. So also fever attends some of the sequels which appear during convalescence, such as phlebitis or periostitis. A good example of this is seen in Fig. 6, where the second febrile period was connected with phlebitis. It would have been easy to overlook its true nature, and it is not improbable that many so-called relapses are really due to undiscovered sequelæ.

Relapses occur most frequently in the second or third week of convalescence, after an absence of fever for several days (one to twenty); but they may develop before the patient has become entirely apyretic, thus explaining some of the long-continued cases of typhoid fever.

To constitute a true relapse there should be an absence of all irritative sequelæ adequate to explain the fever; the elevation of temperature should present more or less clearly the step-like mode of ascent which marks the original onset; enlargement of the spleen should recur, and eruption may reappear. It is, indeed, true that the prodromal stage is usually wanting in relapses, and the temperature is apt to mount more rapidly and by longer steps than in primary attacks. So also the eruption appears at an earlier date, often being visible on the third or fourth day of the fever. I am inclined to think that the eruption is about as frequent, but apt to be less abundant, in relapses as in primary attacks. I have, however, seen it much more copious.

The fever curve in relapses is even more variable as to range and duration than in primary attacks. In general, there may be some correspondence between them in individual cases, but more commonly the fever neither runs so high nor so long as at first. It is not unusual for it to terminate by lysis at the end of from ten to fourteen days, though it may be even shorter, or in some cases may persist three full weeks. Nervous symptoms, especially headache, delirium, and tremor, often recur, and may appear early. Abdominal symptoms are usually less marked.

Upon the whole, relapses are less severe than the primary attacks. There is, however, no reliable rule in regard to this point, and I have seen cases in which the original attack was mild, but the relapse violent and fatal. Murchison states that of the 53 instances of relapse observed by him, one-third were more severe than the primary attack. The dangers of the relapse are the same as those of original attack, but less in degree as regards diarrhœa,

hæmorrhage, and perforation, while there is added risk of exhaustion, due to the already weakened state of the patient.

In rare instances a second or even a third relapse may occur after successive periods of apparent convalescence, so that the entire process may occupy months. These later relapses are, as a rule, but not invariably, milder and shorter in their turn than either the primary attack or the first relapse.

Hutchinson reports a case in which the third relapse occurred nearly four months after the patient was first taken ill; Chantemesse, one in which the whole course of the disease thus extended through five months; and I have attended a patient for one hundred and twenty days through a severe primary attack and three equally severe relapses to full recovery. In the bodies of those who have died in a relapse the intestine exhibits two sets of lesions—those of the first attack in process of cicatrization, together with fresh ulcers of the second attack, although these latter are less numerous and are situated higher in the ileum.

The cause of relapse is clearly a reinvasion of the blood by the bacilli. What, however, occasions the reinvasion is not understood. It has been supposed that a reinfection from without the body may be at fault, but this theory is certainly not tenable in more than a small proportion of cases. Doubtless the reinfection is more commonly from within by bacilli which have been thrown off in the sloughs from the ulcers, or, more probably, by those which have been deposited in foci somewhere in the internal organs. It is important to emphasize the fact that statistics have shown conclusively that relapses are no more frequent in cases which have been treated by cold baths after the method of Brand.

Recurrences or Subsequent Attacks.—The immunity afforded to the system by passing through an attack of typhoid fever is usually complete and lasting. Subsequent attacks do, however, occur, although rarely. A study of 600 cases made by Eichhorst showed the occurrence of a second attack 28 times, or in 4.7 per cent. A few instances of even three or four attacks in one person were included in the series of cases. I have attended several patients through two characteristic attacks of typhoid fever, and have had more than one apparently reliable account given me of three distinct attacks at intervals of several years. According to general observation, second attacks of typhoid are more common in men than in women, and are milder in type than the original one.

Diagnosis.—The whole question of the diagnosis of typhoid fever should be dominated by the view that this disease is much more likely to exist and to be overlooked than that other affections should be mistaken for it; and further, that in doubtful cases, whatever may be their nature, the patient will usually benefit by receiving from the beginning of his sickness the rigid care and treatment appropriate if it should prove to be typhoid.

After the first week of the disease the diagnosis becomes easy in typical cases. In this first week, however, and even after it in atypical cases, it may be very difficult. Still, a diagnosis possessing a high degree of probability

can usually be made early in the affection. The progressive lassitude, dulness, headache, anorexia, and gradually increasing fever with marked morning remissions, render the case very suspicious, especially if these symptoms are combined with epistaxis, diarrhœa, and enlargement of the spleen. If after the fever has lasted a week lenticular spots develop, the diagnosis becomes certain. Even without their presence, however, the continuance of fever of more or less characteristic type, the hebetude and developing nervous symptoms, the diarrhœa and abdominal distension, the epistaxis and bronchial catarrh, strongly confirm our suspicions, and, in the absence of any other demonstrable infection or local lesion to explain the symptoms, justify a working diagnosis of typhoid fever.

More difficult to recognize are the atypical cases which begin with or early develop an intense localization of the disease in certain organs. It has already been shown that typhoid fever in which the initial localization is in the lungs may closely simulate a primary pneumonia. In both conditions the cerebral symptoms may be marked, the fever may rise rapidly, and albuminuria may be present. In typhoid, however, the gravity of the general symptoms soon seems out of proportion to the extent of the local lesion; the spleen is more decidedly enlarged; epistaxis is more common, as is also bronchial catarrh on the side unaffected with pneumonia; abdominal distension and diarrhœa are more pronounced, and later the appearance of rose spots and the absence of herpes, and the failure of crisis to occur on the twelfth or fourteenth day, serve to establish the diagnosis.

The insidious development of *pneumonia* during the course of typhoid fever will often be overlooked unless daily examination of the chest be made systematically. It must be borne in mind, also, that when in the course of pneumonia, and especially in elderly people, the typhoid state ensues, there may be developed a group of symptoms indistinguishable from those of typhoid fever save by the absence of eruption and by the history of the case.

Typhoid fever beginning with marked nervous symptoms may readily be mistaken for *cerebro-spinal fever*. Such cases present rapid rise of temperature to a high point, severe headache and delirium, stiffness of the muscles of the neck and retraction of the head, muscular twitching, and even some degree of general muscular rigidity and soreness. The suggestion of meningitis may indeed be so forcible that a differential diagnosis is impossible for several days. Attention may be drawn to the following points: that in cerebro-spinal fever the onset is usually even more abrupt, the pain in the head more intense, and the stiffness of the neck and retraction of the head earlier and more marked; that vomiting is more common, while the abdomen is apt to be retracted and the bowels constipated; that the nervous symptoms persist and progress, instead of yielding, as those in typhoid may in some degree, to suitable treatment; that epistaxis is wanting and enlargement of the spleen less constant and marked, while herpes is very common. Of course the diagnosis between ordinary cases of these two diseases gives no difficulty,

and the above remarks apply only to the cerebro-spinal type of typhoid fever.

Simple continued fever may greatly resemble mild cases of typhoid fever. It is common to meet with patients who exhibit for a week or more a fever of continued type for which no satisfactory cause can be discovered, and the exact nature of which must often remain in doubt, even after convalescence. The more abrupt onset, the absence of characteristic temperature curve or eruption, the comparative infrequency of marked nervous or abdominal symptoms, of epistaxis, or of splenic enlargement, tend to exclude typhoid fever in the diagnosis.

Typhus fever is usually distinguished with ease from typhoid. Confusion may arise in rare instances, either from the presence of a profuse, dark-colored eruption in the latter disease, or the occurrence of diarrhœa in the former. As a rule, the character of the eruption will satisfactorily distinguish the two affections. That of typhus often appears as early as the fourth day, is copious, and consists of dusky-red, irregular spots which do not entirely disappear on pressure. In addition to this, the onset of typhus fever is sudden, the fever is more continuous in type, the pupils are contracted, petechiæ are common, abdominal symptoms of any sort are infrequent, and the disease is of shorter duration and more apt to terminate abruptly. A more full statement of the points of differential diagnosis will be found under the head of TYPHUS FEVER.

Relapsing fever can scarcely be mistaken for typhoid fever, since the whole history of the two diseases is so entirely different. Relapsing fever has a sudden onset, with continued high fever, which lasts a definite time and terminates by crisis; and this process repeats itself after an interval of a week. There are none of the symptoms of typhoid fever attendant upon the disease, while jaundice is more liable to occur, with pain in the upper portion of the abdomen.

Remittent malarial fever may simulate typhoid fever very closely. Diarrhœa, vomiting, epistaxis, splenic enlargement, and cerebral symptoms may exist alike in both. The locality and the history of the case should be considered. An absence of prodromes; a sudden onset; marked gastro-hepatic disturbance with bilious symptoms and even jaundice; the occurrence of herpes, but no rose-colored spots; and fever of markedly and regularly remittent type, attended with profuse sweating,—point to the malarial nature of the disease. The decided effect of a full dose of a cinchona salt, given as a therapeutic test, is an important help in diagnosis; and finally an examination may be made for malarial organisms in the blood.

It is necessary to bear in mind that *acute miliary tuberculosis*, which is happily of rare occurrence, may readily be mistaken for typhoid fever. In both affections there is a prodromal stage, with anorexia, progressively increasing fever, cough and bronchitis, headache, and delirium passing into stupor. But unless there is abdominal as well as meningeal tuberculosis there will be neither tympanites nor diarrhœa, as in typhoid fever, but rather constipation with retracted belly and cerebral vomiting. The temperature curve in tuber-

culosis is highly irregular; the pulse presents important variations at successive stages; respirations are hurried out of proportion to any demonstrable pulmonary lesion; strabismus, double vision, and local palsies may appear; eruption is wanting; epistaxis is rare; and splenic enlargement is less constant and marked than in typhoid.

Hughlings-Jackson states that an important diagnostic sign between typhoid fever and *tubercular meningitis* consists in the fact that the knee-jerk is never absent in the former, while in the latter it is variable—present one day, absent another, increased another. In this view he is sustained by Money. The diazo-reaction of the urine, once supposed to be characteristic of typhoid fever, occurs in tuberculosis as well. Leucocytosis is present in acute miliary tuberculosis, whereas in typhoid fever the number of leucocytes is often diminished. In all doubtful cases an ophthalmoscopic examination should be made. Although the failure to discover choroidal tubercles affords only negative evidence in favor of typhoid, their detection is of course proof positive of the tuberculous nature of the case.

Primary peritoneal tuberculosis, especially in children without precedent pulmonary lesion, may occasionally cause temporary hesitation in diagnosis, but the irregular fever, the absence of cerebral and bronchial symptoms, as well as of eruption, and the widely different course of the case will soon clear up the doubt.

Influenza may resemble typhoid fever in exhibiting great prostration with early bronchitis, and sometimes epistaxis, combined with sleeplessness, fever, and perhaps delirium. Diarrhœa also often occurs in it, and the typhoid state may develop. The disease is distinguished, however, by the shorter duration, absence of rose-colored spots, of abdominal symptoms other than diarrhœa, and of the characteristic temperature curve.

Scarlatina could only be confounded with typhoid fever in those cases of the latter disease in which the development of the characteristic eruption is preceded for several days by a scarlatinal efflorescence. Even in such there is little chance for error if the mode of onset and the symptoms in general be carefully studied.

Trichiniasis resembles typhoid fever in exhibiting vomiting, diarrhœa, fever, and, later, symptoms of the typhoid state. In no other respect are the two diseases alike. The muscular pain and œdema of trichiniasis are not seen in typhoid fever.

Those cases of typhoid fever which begin with marked mental symptoms may sometimes be mistaken for *insanity*. The same is true of cases first seen at the height of the disease, and of which no previous clinical history can be obtained. A systematic employment of the clinical thermometer and a careful observation of the symptoms will ensure the avoidance of any such error in diagnosis.

Gastro-intestinal catarrh at times produces a group of symptoms highly suggestive of typhoid fever. Either as the result of a profound impression made by unfavorable atmospheric influences upon a morbidly sensitive ali-

mentary tract, or of the ingestion of some non-specific toxic agent, an obstinate subacute catarrhal process is started which may for several weeks keep up irregular fever of moderate degree, coated tongue, anorexia, irritability of stomach and bowels, abdominal distress, marked debility, and mild nervous symptoms, such as headache and restlessness. In children the nervous symptoms may be more marked. Epistaxis is, however, uncommon; the spleen is not enlarged; bronchial symptoms are wanting; there is no characteristic eruption; and the course of the disease is wholly irregular. When the wide irregularities of a typical typhoid are recalled, it must be admitted that it may occasionally be impossible to arrive at a positive diagnosis; under which circumstances the patient should have the benefit of the doubt, and be treated as though in a mild typhoid fever.

Uræmia may develop gradually and pass into a typical typhoid state. I have met with this condition most frequently at or after middle life and in connection with chronic interstitial nephritis. The facial expression and mental state are curiously like those of typhoid fever; a low grade of fever with bronchial and gastro-intestinal catarrh is not unusual, so that I have repeatedly been asked to see such cases as instances of anomalous and protracted typhoid. The detection of arterio-sclerosis and cardiac hypertrophy and albuminuria with casts, the odor of the breath, the absence of eruption, epistaxis, and splenic enlargement, and the history and course of the case, will serve to establish a diagnosis.

Duration, Prognosis, Mortality.—The onset of typhoid fever is usually slow and insidious, so that it is difficult to determine the exact date of commencement or the total length of the attack. In many instances the duration can be only approximately estimated. More rarely the suddenness or severity of the early symptoms permits of a positive decision. The average duration of the attack is three to four weeks. Bartlett estimated it at 22 days in 255 cases, and Murchison at 24.3 days in 200 cases which recovered, and at 27.67 days in 112 cases which did not. When fever continues after the twenty-eighth day some complication may be suspected, yet the last stage of the disease is occasionally prolonged for several days beyond this date without discoverable cause. So slight a local irritation will then suffice, however, to maintain or to revive fever that such a cause may be strongly suspected. The extremely prolonged course pursued by cases where one or more relapses occur has already been fully alluded to.

Typhoid fever may, on the other hand, end considerably within the average period. In abortive cases it lasts no more than from ten days to two weeks. Indeed, some of the abortive mild cases run so short a course that the affection is recognized with difficulty.

The date of death in fatal cases is no less variable. In very grave cases the disease may prove fatal as early as the fifth or sixth day, and in the malignant form death may occur on the third, second, or even on the very first day. On the other hand, it may result from exhaustion or from some sequel or protracted complication long after the specific disease has itself ended. The fever

which may attend such cases is manifestly irritative or septic, and not due to specific typhoid infection. It has already been stated that death may occur in a relapse although the original attack has been a mild one.

In general it may be stated that the third week is the period of greatest mortality in typhoid fever. Death is comparatively rare before the fourteenth day, and, although less rare after the twenty-first day, is still not so frequent then as in the third week.

The immediate causes of death are numerous and varied. Toxæmia and cerebral exhaustion, associated with coma, with or without hyperpyrexia, cause death in many cases, especially from the beginning of the third week onward. In some instances uræmia, owing to a high grade of nephritis, plays a part in causing this condition. Hyperpyrexia, at whatever date it may develop, is often fatal unless promptly subdued. It speedily induces nervous exhaustion and cardiac failure, partly of nervous and partly of muscular origin. Intense asthenia is, as would be expected, a fruitful source of death in this disease. It may come on rather gradually and late in the disease as the result of continued high fever, of sleeplessness, of vomiting, of diarrhœa, or of repeated nasal or intestinal hæmorrhages. Or sudden collapse may occur from a single large hæmorrhage, from profuse diarrhœa, from the shock of perforation, or from direct cardiac failure. There are various ways in which cardiac failure may be induced. The mechanical effect of extreme tympanites, causing great upward displacement of the diaphragm, may co-operate. Advanced degeneration of the cardiac fibre, due to intense toxæmia and high fever, and possibly also acute changes in the cardiac or pneumogastric ganglia, serve to explain the extreme loss of contractile power or the violent disturbance of innervation (delirium cordis) which often precedes and hastens death. Sudden death may occur from cardiac or pulmonary embolism; from the entrance of gas into an intestinal vein; from convulsion, whether uræmic or not; from cardiac paresis, due to imprudent effort; from enormous hæmorrhage.

Severe bronchitis, pneumonia, pleurisy, or other complication may turn the scale against the patient. It is evident, therefore, that from the earliest day to the completion of convalescence there is ground for constant uncertainty and anxiety.

The exhausting effects of bed-sores, or of large centres of suppuration, as in the parotids, may prove fatal even after all the ordinary dangers of the disease have apparently been escaped.

The mortality of typhoid fever has been calculated almost exclusively from hospital statistics. It is evident that these are to some extent misleading, since many cases are admitted too late to be amenable to any treatment whatever, and the rest are only too apt to have undergone such exertion or exposure in the early days of the disease as to materially increase its dangers. It must be remembered also that the mortality of typhoid fever varies much in different epidemics and apparently in different localities. Study of the most extensive statistics available indicates that before the introduction of the Brand method of treatment by systematic cool baths the mortality of typhoid fever in

hospitals varied from 10 to 30 per cent., but most commonly ranged between 15 and 25 per cent. It is impossible to avoid drawing the conclusion, from recent statistics, that in those institutions where the Brand method has been used the mortality has been reduced abruptly, and without other ascertainable cause, to from 5 to 8 per cent. Undoubtedly, the modern antipyretic methods, even without the use of full baths, have been of vast service in the treatment of typhoid, especially in private practice, where as yet the Brand system has been used but rarely. No accurate figures are available on any large scale, but from numerous inquiries I incline to believe that the mortality of typhoid fever in private practice is not less than 10 per cent. It happens occasionally that a large series of cases will occur without a single death. I have myself treated 100 consecutive cases in private practice without a fatality, and I know of several series of 100 cases with a mortality of only 1 to 2 per cent.

The prognosis of typhoid fever is very difficult to estimate, and is influenced by general considerations and by special symptoms.

The disease is decidedly less fatal in children from infancy up to puberty. I have observed that in young persons who have been growing very rapidly the nervous symptoms and the asthenia are apt to be marked and the disease dangerous. The mortality increases rapidly after forty-five years of age. Sex exerts no definite influence. Most of the statistics show an excess of deaths among females of about 1 per cent.

Season does not appear to have any effect on the mortality. The varying reports are probably due to the different gravity of the outbreaks. Cases occurring during protracted spells of intense heat are undoubtedly more apt to be fatal.

The station in life is without influence on the prognosis. Quite as large a percentage of rich as of poor die. The personal constitution and habits are of some importance. I have repeatedly been impressed with the unhappy effect upon the course and result of typhoid fever produced by exertion or exposure during the early days of the attack. The curious fact that those who are in delicate health from previous disease or other causes do not suffer more in attacks of typhoid fever than those in vigorous health may be partly explained by the fact that the former yield to the early symptoms and place themselves promptly under treatment, while the latter are too apt to persist in their usual occupation until utterly exhausted. Murchison and others maintain that the strong and robust and those of large muscular development more readily succumb. It is well known that the corpulent are particularly liable to die from it. This is due to the fact that the fever runs unusually high in them, and that the high temperature induces degenerative changes in their tissues with unusual ease. In persons of intemperate habits or in those with gouty or renal affections the disease is more apt to terminate fatally. In those of a nervous temperament many of the symptoms are liable to be worse. The susceptibility of the system and the intensity of the virus have more to do with the gravity of the case than any other influences. It is a matter of general agreement that young

persons who have recently moved into large towns where more or less typhoid is always present are specially liable to the disease and in an aggravated form. On the other hand, most fatal outbreaks occur in isolated and healthy families or communities, owing to accidental infection of the locality. When typhoid fever attacks pregnant women abortion nearly always follows, and the danger of a fatal result is considerable. The existence of organic heart disease, emphysema, cirrhosis of the liver, or Bright's disease greatly increases the gravity of typhoid fever.

The prominence of certain symptoms has an important bearing upon the prognosis. The higher the temperature goes and the more persistently it remains elevated, the greater the danger to life becomes. This is, however, only a general rule. High temperature may often be borne well for a considerable time, provided severe nervous symptoms do not attend it. When the morning remissions are slight and brief the prognosis is worse. An inverted temperature curve, with the morning temperature higher than that of the evening, is also unfavorable. On the other hand, the earlier in the attack the morning fall begins to become steadily more marked, the more favorable is the prognosis. A sudden fall of temperature, if accompanied with a corresponding fall in pulse-rate and improvement in general symptoms, may denote the crisis of an abortive attack and be followed by convalescence. If, however, the sudden fall be attended with marked depression of strength, it may denote the approach of collapse, especially from copious hæmorrhage.

A temporary descent in the temperature curve and improvement in general condition during the second or third week, followed by a return of the fever and other symptoms in aggravated form, is an unfavorable occurrence, and the attack is apt to end fatally. I have repeatedly seen bitter disappointment result from this delusive lull in the symptoms. I am inclined to agree with Lacaze that the appearance of sudamina in the third week in severe cases is apt to be a favorable sign, and that the temperature often falls within a few days subsequently. Most writers, however, do not believe that sudamina possess any prognostic value.

A pulse of over 120—except in children or under excitement—is always a sign of cardiac weakness. This is particularly true if the pulse be at the same time feeble. Liebermeister's statistics show that the more rapid the pulse-rate the greater the mortality becomes. Of 12 patients in whom it attained a rapidity of over 150, 11 died. The character of the first sound of the heart is also of great prognostic importance. The more valvular its quality and the more feeble the cardiac impulse, the graver the prognosis. Naturally, the earlier the pulse and the heart-sounds show signs of weakness, the more unfavorable is it. Dicrotism is so characteristic of the pulse in typhoid fever that unless associated with great softness and weakness it is not especially significant of danger.

The early development of nervous symptoms is unfavorable. The presence of coma or of wild delirium is a grave indication. Low muttering delirium,

with tremor, occurring early in the attack, also is an indication that the case is a very severe one. According to Zenner, the degree of delirium is to some extent a measure of the gravity of the infection, though care must be taken to recognize those cases where the excited delirium is hysteroidal in nature and unattended by other symptoms of special danger. Coma vigil, carphologia, subsultus, rigidity, general convulsions, protracted hiccough, early incontinence or retention of urine, early incontinence of fæces, insomnia, great prostration early in the disease, great tympanites and abdominal pain, a dry brown tongue, severe diarrhœa, severe intestinal hæmorrhage, vomiting late in the attack, and the occurrence of peritonitis from any source or the development of any other complication, of course add to the seriousness of the disease to a greater or less extent.

Regarding the influence on prognosis caused by the association of other infectious fevers with typhoid fever, it is sufficient to say that the coexistence of malarial poison does not seem to add to the danger, but that most cases of the malarial form of typhoid are of favorable type.

Treatment.—*Prophylaxis.*—Typhoid fever is certainly to a large extent a preventable disease. Produced as it is by a specific germ, it is self-evident that the objects of prophylaxis are to destroy the germ wherever known to exist, and to adopt every precaution against its admission to the system.

In the care of each case of typhoid fever the fæcal discharges, which contain the virus in abundance, must be thoroughly disinfected and properly disposed of. Special reference is here made to the careful directions given for this purpose in the section on Disinfection in the article HYGIENE. The disinfected discharges should be emptied into privies or water-closets, but never upon the open ground. In rural districts they may be buried in the earth at points remote from the supply of drinking-water. Equal attention must be given to the disinfection of the body-linen of the sick, the bed-clothing, the mattresses, and the furniture of the sick-room.

While thus endeavoring to prevent extension of the disease, it is essential to make careful search for the source of infection in each individual case. The remarks under the head of Etiology in this article, as well as the article on HYGIENE, may be consulted with advantage. The drinking-water and the milk-supply offer themselves as the most probable sources of infection. In large cities it is for the most part impossible to follow up the investigation. In localized outbreaks, in small towns, or in rural districts, on the other hand, we know with what admirable results such examinations have been pursued. Grave defects, leading to contamination of these necessary articles of universal consumption, are detected whose correction will avert future trouble. If in any large community typhoid fever is habitually prevalent to a greater or less degree, it may be accepted as highly damaging evidence against the drainage, sewerage, water-supply, or milk-supply. During the existence of an attack of typhoid fever it is desirable that both the water and milk should be boiled before being ingested. Defects in drainage and in house-sewerage are less likely to lead to this than to some other infections. In well-sewered houses the

chief danger to health connected with the system occurs when the fixtures have been unused for two or three months and the traps and interior of the pipes have become dry, so as to give off dust-particles which are carried into the rooms. It is unwise to disturb existing arrangements about a house during the course of a case of typhoid. After its conclusion careful examination must be made of the entire drainage and sewerage systems. In large towns with public sewers this inquiry is necessarily limited to the internal fixtures and to the connection with the sewers. In rural districts a wider field of investigation must be covered.

Treatment of the Attack.—The general management of a case of typhoid fever involves many details, careful attention to which does much to determine the favorable result of the case. In this disease it is pre-eminently true that a good nurse without any doctor is better than the best doctor without a good nurse. A caretaker of intelligence, preferably a trained nurse, should be early placed in charge, in order that the careful written instructions of the physician may be accurately and systematically carried out.

The sick-room should be as large and airy as possible. When practicable it should have a sunny exposure. If there is a better room available than that in which the patient first takes to bed, a transfer should be made promptly before the more serious stages of the disease are reached. The course of the disease is so long, and the result so largely depends on the maintenance of vitality, that these questions assume great importance. The room should be kept scrupulously clean and well ventilated. It is, however, a grievous mistake to suppose that, on account of the infectious nature of the disease, strong draughts may safely be permitted. There are remissions in the fever with relaxation of the surface, and congestions or increased catarrhal irritation may readily be induced if the patient be not carefully protected. The use of screens is to be advised, both to guard against currents of air and to aid in softening the light in the room.

The bed should be neither too hard nor too soft. A feather mattress is to be avoided: a woven-wire mattress covered with one of hair forms one of the best beds. A rubber cloth should be spread beneath the sheet. Since much care is required to avoid the formation of bed-sores, the sheet must be kept smooth, and in the later stages of severe cases a water-bed may be used with great advantage.

Complete rest in bed is essential. From the first hour that the suspicion of typhoid fever arises the patient must be put to bed and kept there until the close of the case. The earlier this is done the better the prognosis. Not only in hospital practice, but in private, we meet many cases where the patient has persisted in his occupation or his pleasures, or where journeys have been undertaken, during the first week of the fever, and the effect upon the course and result of the disease is very bad.

The use of the bed-pan and urinal should be insisted upon from the start. Many patients find difficulty at first in emptying the bladder or bowels in the recumbent position, but as a rule they soon acquire the power. Cases are met

with, however, where the effort continues fruitless, and causes such excitement and annoyance that it is necessary to have the patient lifted on the commode at the side of the bed. I have seen this necessity arise most frequently with young women, and by the use of proper care no ill effects have followed in any case.

In cases marked with extreme restlessness and insomnia it sometimes does good to have a second bed prepared, and to move the patient to it for a portion of each day. A transfer to another chamber may exert a soothing and happy influence.

The diet is also of great importance. It should be, from the start, liquid and easily digestible. Milk generally answers the requirements better than any other article. In many cases it serves as the only article of food that need be given throughout the course of the disease, or at least constitutes the basis of the diet. It ensures the ingestion of considerable liquid; it is readily digested by most persons when taken as an exclusive diet; its favorable effect in gastro-intestinal catarrh, always present in typhoid, is well known; its relations to hepatic and renal activity are favorable. From one and a half to two quarts is the proper amount for an adult during twenty-four hours, provided it does not disagree. It should be given in divided quantities every two to three hours during the twenty-four hours. Care must be taken not to administer more milk than can be readily digested. This can be determined by watching for symptoms of gastric indigestion or for an increase of diarrhœa or the presence of curds and of fat-globules in the stools.

Great harm may be done by forcing excessive amounts of milk and other liquid nourishment upon fever patients, as though their debility demanded, and their inability to resist justified, its administration. It should not be necessary to insist on the fact that food must be digested in fever just as in health; that the secretions of the stomach and the tone of its muscular coat are much impaired, especially in typhoid; and that all ingesta in excess of the digestive power are doubly injurious.

The milk may be given raw or boiled, hot or cold, iced, peptonized, mixed with lime-water or aërated water, according to the case. It is a mistake, however, to consider milk the only suitable article of diet. It is, as has already been said, the most generally acceptable food in typhoid fever, and especially when a decided tendency to diarrhœa exists. But in some instances it tends to produce constipation, and may even form such hard masses of curd in the stomach and intestine that it becomes practically one of the most solid of foods. If these difficulties cannot be overcome by peptonizing it or by mixing it with lime-water, it may be necessary to substitute some other form of nourishment. Again, the repugnance of the patient to milk may necessitate a change of diet. In such cases buttermilk, whey, or koumyss may be of value. Broths or soups of mutton, beef, chicken, veal, oysters, or clams are often useful. They may contain a small quantity of barley or rice. White of egg mixed with water or with stimulants is valuable at times, as is beef-juice or some of the various preparations of beef on the market.

Whether the patient shall be aroused for nourishment during the night depends largely upon circumstances. If the case is mild, it may not be necessary to do so. If, on the other hand, there is little sound sleep, but the constant presence of somnolence, he will probably need the food, and awakening him will do no harm. If, again, the patient has been suffering from insomnia, and is enjoying the first refreshing sound sleep, it can scarcely ever be proper to arouse him either for food or medicine. The tact and skill of a nurse are in nothing better shown than in dealing with the sleep and the nourishment of fever patients.

The patient should be convalescent for at least ten days before a gradual return to solid food is commenced. I am aware that some excellent observers sanction the use of small quantities of semi-solid or solid food, but my own experience is wholly opposed to it. I have repeatedly observed unpleasant or even alarming symptoms so closely connected with the ingestion of even a minute amount of such food that it was impossible not to regard them as directly caused by it.

In addition to the liquid food, water should be freely given. The hebetude often blunts the perception of thirst, so that the patient may not ask for water, and yet will take it greedily if offered. It is important, therefore, that small amounts be given at short intervals. On the other hand, it is essential that the stomach should not be so flushed with cold water as to weaken digestion. Carbonated water or cracked ice is often an agreeable change. Very weak iced tea may be allowed in small quantities. Water acidulated with lemon-juice or with a few drops of dilute phosphoric acid is grateful, and may be given if there be no diarrhœa.

The question of administering alcohol comes up for discussion in every case of typhoid fever. It must be stated at the outset that in mild, uncomplicated cases, especially in young, healthy, and temperate subjects, stimulants are not needed so long as the disease is following its typical course. Here as elsewhere alcohol should be avoided when not absolutely demanded, both because it may irritate or disagree and because its use may tend to establish a habit. There is, however, such a dangerous tendency to exhaustion from various causes that in a majority of cases more or less alcohol is required sooner or later in the course of the disease.

The indications which call for its use are, in the first place, an inability to administer enough food. There are cases where, owing to repugnance or to nausea, it seems impossible to have enough food taken to support life until the disease abates. Under such circumstances small amounts of stimulants, such as brandy or whiskey in carbonated water or in milk, or dry champagne, not only are assimilated as food, but aid in maintaining the circulation till the crisis is past. There are also preparations which, like the various liquid peptonoids, are both nutritious and sufficiently stimulating. Again, the existence of high temperature nearly always makes it necessary to stimulate the patient. Nervous exhaustion and heart failure are urgently impending, and while proper antipyretic measures are the rational treatment, it is neces-

sary to use alcohol for immediate effect. The heart suffers so seriously in typhoid from failure of innervation, from changes in the muscular tissue, and from protracted reflex irritation that a weak, small, compressible, rapid pulse, with impaired cardiac impulse and systolic sound, is a frequent indication for alcohol. Other remedies may be, as we shall see, required, but alcohol cannot be dispensed with safely. The development of the typical typhoid state, with profound dulness, tremor, dry, brown tongue and sordes, weak pulse, and shallow, rapid breathing, whether associated with very high temperature or not, expresses so much nervous exhaustion that stimulation is called for.

It is necessary to give alcohol in the serious complications of typhoid, such as pneumonia, pleurisy, hæmorrhage, and severe bronchitis or diarrhœa. Patients over forty years of age, even of previously temperate habits, and younger ones who have been intemperate, had better receive small quantities of alcohol early; and the dose should be increased more or less rapidly as required. It will be seen, therefore, from the above indications, that although alcohol is not to be ordered as a mere matter of routine, it is called for in most cases, and we must be ready to give it as soon as, and in such amounts as, required.

The amount to be administered will vary with the needs of the case. Unless the symptoms are urgent it is well to begin with small and well-diluted doses. As the case advances, from 2 to 6 ounces of whiskey daily may be called a moderate amount; 8 to 12 ounces daily is not too much for severe adynamic or complicated cases; and even more than this, up to an ounce hourly, may be absolutely required for days in succession to tide a patient over a critical period.

When alcohol is ordered or when the amount given is increased, it must be considered a tentative measure, as in the case of any other remedy. I am convinced that under the routine practice of excessive stimulation in vogue until recently the symptoms of alcoholic over-action were often mistaken for advancing debility and regarded as an indication for still more free stimulation. If delirium grows less, the pulse stronger, and the tongue less dry under the use of alcohol, the remedy is doing good; but if these symptoms become aggravated, the question should be entertained whether too much alcohol is not being given.

Probably the most convenient and reliable form of stimulant is whiskey or brandy, its greater strength making the dose smaller and more manageable. It may be given in milk or, when this is not borne well, in water. Sometimes it is well to change for a time to champagne, sherry, claret, or other wine.

Undoubtedly, one of the most important indications, which must be met in the great majority of cases of typhoid fever, is that for reduction of temperature. It is true there are cases which run so mild a course, the fever not rising at any time above 102° F., that this question does not have to be considered. There are other and more rare cases where high temperature is carried without apparent inconvenience. I have seen the daily maximum at 105° F. for ten days in succession in the case of a young woman who had at no time either

delirium or cardiac weakness, and who made a rapid and complete recovery though no antipyretic treatment was used. This only shows that the pyrexia of typhoid fever is a highly complex condition, and that high temperature may occasionally persist, owing to some peculiar nervous disturbance, without the serious results usually consequent. Even when a temperature of 103° or 104° F. is apparently unattended with damage to the brain or heart, it must be watched with incessant anxiety, because alarming symptoms may appear most unexpectedly. To what point may fever be allowed to go safely in typhoid without interference, and have we any means at our disposal by which it can be surely and safely reduced if it threaten to overstep this limit? Our knowledge of the natural history of typhoid shows it to be a self-limited disease which tends to recovery in the great majority of cases, though the temperature reaches 102.5° or 103.5° F. more or less frequently in the course of average cases. But the normal mortality of the disease, if allowed to run its course simply with proper food and good care, is altogether too high to be satisfactory; and it is being more and more clearly made out that a large proportion of this mortality comes directly or indirectly from the baleful influence of the pyrexia. This statement has been confirmed conclusively by the remarkable results obtained in a large series of cases by resolutely keeping the temperature down below the lowest degree above mentioned (102.5° F.). The only way in which this can be done safely and effectually is by the external use of cold water, and hence to-day hydrotherapy is an almost constant feature in our treatment of typhoid.

There are various modes of applying it, which vary in their efficiency and value, including repeated spongings of the surface; the ice-cap to the head; the cold-water pack; cold affusion; Leiter's tubes; the graduated bath; the strict Brand method of cold-water bathing. In mild and even in ordinary cases sponging the entire surface of the body with cool water as often as the temperature in the mouth reaches 102° F., is distinctly valuable. A little vinegar or alcohol may be added to the water, which may be cold (50°–70° F.) or cool (70°–80° F.) according to the less or greater degree of fever; and the sponging may be kept up for ten minutes or more, and be repeated as often as every two hours. Friction and gentle kneading of the surface should be combined with it. The temperature may be temporarily reduced from 1° to 1.5° F. in this way. As a rule, it promptly rises again, but the process is agreeable and not fatiguing to the patient. A thin rubber bag or bladder filled with cracked ice may be applied to the head at the same time, and kept in place much longer. Even when the fever is not high, but nervous excitement is marked, this may be used with good effect.

The cold pack is a much more powerful antipyretic, and is applicable even when the temperature is 104° or 105° F. The bed should be protected by a rubber cloth, and the patient, with his body-clothing removed, wrapped in a sheet wet with cold water. The surface is then rubbed briskly through the sheet, and from time to time cold water is sprinkled freely over the sheet, so that it shall be kept wet and cold. By using ice-water, even hyperpyrexia,

104.5° F. or above, may be dealt with effectively in this way; but the process is tedious and troublesome. The recommendation to use friction during the pack must not be overlooked. Cold affusion possesses no advantage over the cold pack, and is rather more troublesome to carry out effectively. Both of these methods are inferior to the cold bath in certainty of action and durability of effect.

Leiter's tubes were originally made of flexible metal, but now much more conveniently of rubber. The most valuable forms are those for application to the abdomen and trunk and to the head. A vessel containing ice-water is placed at a slight elevation above the bed: one end of the tube is introduced, and, the flow having been started by syphon action, the water runs continuously through the coil, and escapes by the other end of the tube into a receptacle below the bed. One great merit of this simple apparatus is that it may be kept in operation for hours at a time with no attention save the occasional filling and emptying of the respective vessels. In my own experience I have not found these tubes adequate to cope with very high fever, but they are valuable adjuvants and are sufficient for many ordinary cases.

In the graduated bath the patient is placed in water of about 90° F., which is then cooled down to 70° or even lower. This form of bath must be continued longer and its results are less reliable than when the water is cold from the start. It has the advantage that the shock to the patient is not so great: it is therefore especially suited to children, to old persons, and to greatly debilitated cases.

All of these modes of supplying cold externally are useful and have their respective places, but from none of them can such results be obtained as from the systematic use of cold baths in precise accordance with the method advocated by Brand. His original publication in 1861 led Ziemssen, Liebermeister, and others to take up the subject, and gradually hydrotherapy in typhoid fever became generally recognized. Currie in the last century was the pioneer in this field. Hiram Carson in this country has for many years bravely advocated its claims, but the medical mind was not ready and the recommendations lacked strict scientific method. Herein is the immense credit of Brand, and it must be admitted that no such results have ever been achieved as are now reported by many observers who have followed his directions implicitly.

The Brand method consists in the systematic employment of general cold baths with frictions whenever the temperature of the patient reaches a certain elevation. A large bath-tub, movable on rollers, is kept half full of water of 65° to 70° F., and is rolled to the edge of the bed when needed. As often as the temperature, taken every three hours in the mouth or rectum, is over 102.2° F., the patient receives a bath lasting fifteen to twenty minutes. His clothes are removed, and he is covered with a sheet or arrayed in a thin muslin or linen garment specially adapted for the purpose. He is then carefully assisted from the bed to the edge of the tub, or, if not able to assist himself, is lifted bodily into the water by the attendants. If it seems advisable a small amount of stimulant is given. Some cold water is now poured over his head as he

gets into the bath in order to diminish the shock, and he is then submerged in the bath up to the neck. The head can rest conveniently upon a rubber cushion. During the whole period of immersion he should be briskly rubbed through the sheet and cold water poured on his head at intervals. This friction and affusion, together with a second dose of stimulant, aids largely to prevent or check the tendency to chill and cyanosis which otherwise develops at the end of about ten minutes. After the bath the wet linen is quickly removed, and the patient is placed in bed, wrapped in a sheet, and covered with a blanket without friction or any drying of the skin. A stimulant can be given after the bath, and the temperature, preferably in the rectum, should be taken. The accompanying chart (see Fig. 9) illustrates well the immense superiority of general cold baths over even the most through sponging.

Brand's last statistics are really remarkable. He tabulated 1223 cases treated according to his method by himself and a few other clinicians. Only 12 deaths occurred—a mortality of 1 per cent! All of the fatal cases were in individuals who did not come under treatment until after the fifth day of the disease. He claims as a result of thirty years' experience that every case of typhoid fever will recover in which his method of treatment is commenced before the fifth day.

Brand's method has been widely used in Germany for some years. In France it has not found general acceptance, although Glénard's results at Lyons have been excellent. In England the method has not yet been largely adopted. In America the results of all those who have given the treatment a fair trial have been most encouraging, although the number of cases is not yet sufficiently large to justify absolute conclusions. Hare of Australia reports 797 cases with a mortality of 7 per cent.

The percentages of recoveries in general have not been so good as in the remarkable statistics recently published by Brand. It must be remembered, however, that in many series of cases some of the patients have not been treated in strict accordance with his method nor from a sufficiently early day. The fact remains, in spite of this, that no other mode of treatment has ever yielded such good results on a large scale. Not only is the mortality lessened, but the whole course of the disease seems to be rendered milder. The mind remains more clear, without the development of stupor; sleeplessness and excitement are also less. The typical typhoid state is less frequently observed than under other plans of treatment. Nor are there any serious risks or disadvantages connected with it. It has been asserted that the treatment by cold baths increases the tendency to intestinal hæmorrhage, but this has not been substantiated. Liebermeister reports that of 861 cases treated without cold bathing, 8.4 per cent. suffered from such hæmorrhages, while they occurred in only 6.2 per cent. of 882 cases subjected to bathing. It has also been claimed that relapses and severe complications are more common under the cold-water treatment, but there is no reliable evidence in support of this assertion.

Experience has already shown that Brand's method is very generally applicable. Excellent results have been reported in cases occurring in infancy

FIG. 9.

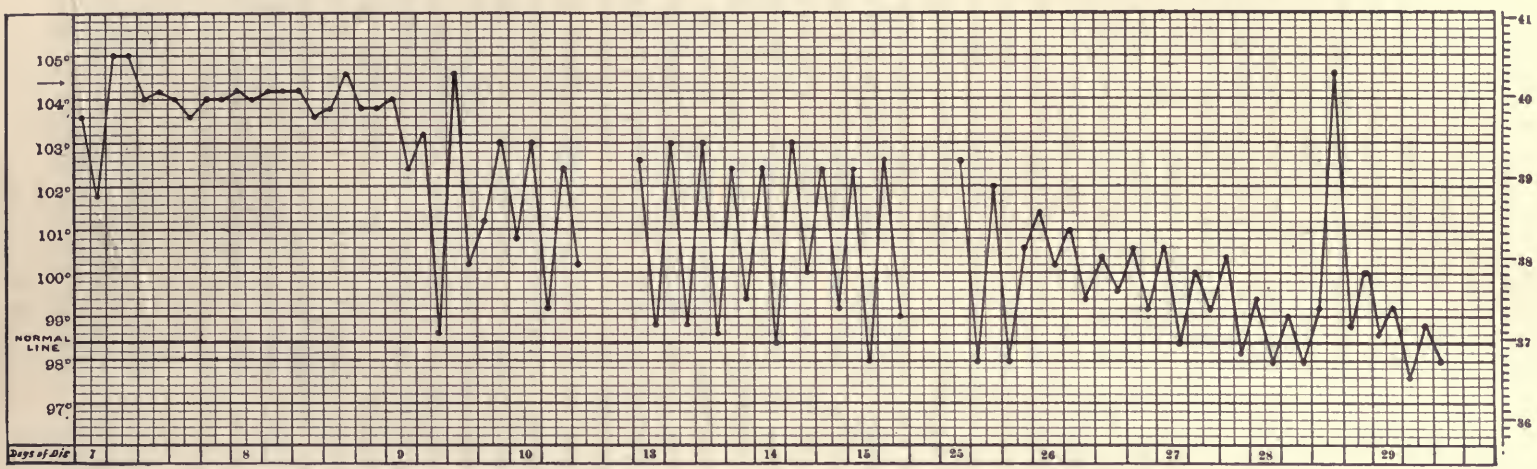


Chart showing relative effects of sponging and tub-bathing, the temperature during various parts of the disease, and a febrile recrudescence from dietary indiscretion during convalescence. The chart begins at about the seventh day of the disease. The case was treated with sponge-baths until the ninth day without influence on the fever, then by tub-bathing, with uniformly happy result. The second section of the chart shows the temperature during the thirteenth, fourteenth, and fifteenth days. The third section shows the effect of the last two baths, the decline of fever, and a post-febrile recrudescence.

and childhood. Not only are such complications as pneumonia and bronchitis not induced by cold baths, but it has been shown that their existence does not contraindicate this mode of treatment. Pregnancy likewise is no contra-indication.

Enough has been said to show that there has been placed in our hands by Brand a mode of treating typhoid fever of great simplicity and value. The question remains as to its limitations and as to when it should be insisted upon. In the first place, it is as yet a very difficult, and often an impossible, thing to secure its adoption in private practice. If it is to be employed, it should be with the rigid observance of every detail as above described. This certainly seems to many a harsh course of treatment to pursue, and the patients often complain bitterly of it. Not rarely, however, the relief obtained after a few baths is so great that the repugnance to it disappears.

The difficulty of securing a suitable movable bath-tub at short notice and on reasonable terms has been a serious obstacle. It will be found, however, that any leading druggist will cheerfully co-operate, so that a tub with proper attendants may be available at all hours and at reasonable rent. Dr. Wilmer R. Batt of Philadelphia has recently devised a portable tub which is all that can be desired in point of compactness and convenience. It is obvious that no community can with propriety be without this invaluable resource in the treatment of the numerous acute infectious diseases.

In cases which do not come under our treatment until a comparatively late period, as happens frequently in hospitals and less commonly in private practice, this method is less successful, as is urgently represented by Brand himself, than when adopted before the fifth day of the disease.

In mild cases the fever may not rise sufficiently high at any time to suggest any more powerful mode of applying cold water than by repeated sponging. If, however, the temperature rises to 102° in the axilla or 102.2° in the mouth or rectum, the Brand method should be adopted if practicable, or the use of Leiter's tubes, with the ice-cap, repeated sponging, and, if necessary, the cold pack, should be instituted. This is the more urgent in proportion as the temperature remains at or about the maximum for a greater number of hours. Let it be remembered that under such circumstances, even though serious cerebral or cardiac symptoms may not be present, they are liable to appear abruptly and unexpectedly, so that the case will speedily assume a highly dangerous position.

In extremely nervous cases or in elderly or much debilitated subjects the milder forms of hydrotherapy are preferable. The actual existence of intestinal hæmorrhage or of peritonitis precludes cold-water bathing.

In addition to the external use of cold water we have other antipyretic remedies that may be used in conjunction with hydrotherapy, or even, in certain cases, to its exclusion. A warning word must be spoken as to many of them, for serious harm is often done by the excessive use of drugs which possess the power of reducing temperature, under the mistaken notion that this result alone is sufficient proof of their value in the case. It must never be

forgotten that the mere lowering of temperature by such means is not necessarily an improvement in the febrile process, and that the drugs which are powerful enough to effect it are sure to possess other activities which may be harmful. In short, it is to be borne in mind that it is the patient, and not the fever alone, we are called on to treat. Happily, a reaction has set in against the practice, which grew out of the recognition of the dangers of pyrexia and the possession of drugs of great antipyretic power, of hammering down, and of trying to keep down, the temperature by large, and if necessary by larger, doses of such remedies.

Antipyrine, phenacetin, and acetanilid are the most powerful and reliable drugs of this class. Not only do they reduce fever temperature remarkably, but they are usually well borne by the stomach and they often exert a decidedly tranquillizing action. Upon their first introduction the natural mistake was made of giving them in doses far too large and frequent, so that serious depression of strength, and even fatal collapse, followed in many instances. Patients with typhoid fever are often peculiarly susceptible to the action of these substances. This is true throughout the course of the disease, but particularly so in the later stages, when great variations in temperature naturally occur. It is not so much that these drugs are directly depressing to the heart, for they rarely cause cardiac symptoms when given in afebrile conditions. Even here, however, I have observed not a few cases of extreme susceptibility to their depressing action. But it appears that in fever they affect the nerve-centres, so that an artificial crisis is produced, and, as in all crises, danger of severe depression, and even of collapse, arises. With this danger clearly in mind the proper cautious use of these remedies is of great service in certain cases. They are not required in the mild form with moderate fever: the question as to their use arises when the temperature reaches or passes 103° F. If hydrotherapy is to be used systematically, only occasional doses, if any, of these powerful antipyretics will be required. The amount given should always be small. Five grains of antipyrine and less of phenacetin or acetanilid is as large a dose as should be given. If no effect is produced it may be repeated in the course of an hour. The object should not be to cause a great fall in temperature: it is enough if a reduction of one or one and a half degrees is secured. It sometimes happens that this reduction lasts a considerable time, so that only a few doses at long intervals are required; and it is in such instances that the happy effects of the remedy are conspicuous. If the temperature promptly rises again to the former point, I am totally opposed to pushing the use of any remedy of this class. Ehrlich and others have tested the plan of keeping the fever constantly low by the continuous administration of small doses of these drugs, but the results were not satisfactory either as to the duration or the mortality of the cases so treated.

The use of quinine in typhoid fever has been excessive, and yet it is of service in many cases as a tonic rather than as an antipyretic. There are so many more powerful and reliable means of reducing temperature than by colossal doses of quinine (20 to 40 grains in the evening, as advised by Lieber-

meister, so that the full effect of the drug may be exerted at the time of the usual morning remission) that it is now rarely used for this purpose. If in any case with high fever hydrotherapy cannot be used, if antipyrine or its analogues would be too depressing, and if the stomach be not irritable, antipyretic doses of quinine could be given. It is, however, in moderate doses, 4 to 8 grains in the twenty-four hours, that it is of most general utility. It may be given in soft, freshly-made pills, in capsules, or in solution with mineral acids. If there be the least reason to suspect that it irritates the stomach or favors diarrhœa, its administration in larger amount by suppository or enema should at once be substituted. In ordinary cases which are pursuing a normal course it need not be given until marked debility begins to show itself.

The preparations of salicylic acid will often exert a powerful antipyretic action, but in adequate doses they affect the head as unpleasantly as does quinine, are apt to disorder the stomach, and are probably depressing to the heart.

In the moderate fever of mild or ordinary cases aconite in small doses may be given safely and with pleasant effect. One drop of the tincture of the root, with or without a small amount of citrate of potassium, solution of acetate of ammonium, or spirit of nitrous ether, may be given every hour or two for five or six doses from noon onward.

Digitalis is a drug about whose value in fever I have much doubt. I am clear it should not be given in large doses for its antipyretic effect. The only indications for its use are to be found in the state of the heart's action and the pulse.

Having considered the general care of the patient, the diet, and the indications for the use of stimulants and for the control of the fever, we have met the questions which arise in every case of typhoid fever. We have seen that it is only in the mild cases that an expectant plan of treatment can be pursued, because the scientific use of antipyretic measures constitutes a definite treatment, and we have now learned that by this method far better results are secured than by allowing the disease to run its normal course. It constantly happens, however, that special indications present themselves which call for additional medication. It cannot be too strongly urged, however, that no single dose of medicine should be ordered unless with a definite and well-recognized purpose. The symptoms are numerous and complex, so that many suggestions for medication offer themselves: the patient is dull and acquiescent, so that remedies are taken for the most part without opposition; care must be constantly observed lest by degrees drug is added to drug until opportunities for rest are curtailed and the digestive power of the stomach is damaged.

The catarrhal and ulcerative lesions of the gastro-intestinal mucous membrane are constant in greater or less degree. It is true they are part of a specific process, and therefore much less amenable to treatment than if idiopathic. There is much evidence, however, that these lesions can be favorably affected by suitable remedies if administered from a very early period of the disease. It is obvious that if this can be done the secretions will be improved,

digestion will be assisted, and intestinal asepsis promoted indirectly. It is doubtful, indeed, whether any of the so-called antiseptics which have been recommended in typhoid fever can be given in sufficient amount to disinfect the whole mass of intestinal contents; and it is not improbable that their surface action may account for a considerable part of whatever good they do. But, upon the whole, it seems desirable that a remedy of this class shall be given in all cases of the disease, due care being taken to select one which is adapted to the condition of the stomach and bowels. The large number of such remedies recommended is of itself sufficient to prove that no one is the most available in all instances. The list from which choice may be made is a large one, so that the special indication of each may be met.

Among them may be mentioned calomel, nitrate of silver, the mineral acids, turpentine, naphthalin, iodine and carbolic acid, chlorine-water, thymol, salol, iodoform. It is of course understood that only one remedy of this class should be used at one time.

My own decided preference has for years been for nitrate of silver, which I give in every case from the first hour that the nature of the disease is suspected. It is given purely for its surface action, just as it would be used in a case of idiopathic gastro-intestinal catarrh. It is administered in conjunction with appropriate antipyretic treatment, and it is usually compatible with any other remedy required for special indications. Its use is continued throughout the entire course of the case, and as much as twenty-five grains may be given to an adult without the least fear of causing discoloration of the skin. In case symptoms arise which suggest another remedy of this class, the change should be made promptly. If the stomach is irritable, the following solution may be used:

℞. Argenti nitratis, gr. ij;
Aquæ destillat., f℥iij *vel* iv.
M. Ft. sol.
Sig. A teaspoonful on an empty stomach every four or six hours.. One or two drops of deodorized tincture of opium may be added to each dose.

Usually silver is best given in pill form, according to this formula:

℞. Argenti nitratis, gr. vj;
Ext. opii,
Ext. belladonnæ, āā. gr. ij;
Mannæ, q. s.
Misce et div. in pil. xxiv.
Sig. A pill three times daily soon after food.

If diarrhœa develop, the belladonna may be omitted and the opium be increased; if constipation be present, the opium may be dropped and ext. nuc. vomicæ gr. $\frac{1}{6}$ be added to each pill.

My own belief is that this remedy so administered is a safe and useful part of the regular treatment of typhoid fever, and that when begun early and continued judiciously it helps in some degree to prevent the development of serious symptoms and intestinal complications. This is not only my own experience, but that of many of my colleagues who have adopted this treatment. Many ridicule the notion that any good can be done by such small doses of so unstable a salt; but the clinical evidence, both in this disease and in gastro-intestinal catarrhs, deserves consideration, and we know that we are dealing with a mucous membrane in a state of such morbid irritability that the ingestion of even a minute amount of unsuitable food may induce violent symptoms.

The mineral acids, muriatic, nitro-muriatic, sulphurous, and phosphoric, are all valuable, both for surface action and as antiseptics. They are most useful when the tongue has a heavy yellow coating, but is not dry; when the abdomen is not greatly distended and the bowels are quiet; and when there is considerable thirst. They should be given in moderate doses, freely diluted, at intervals of three or four hours.

Turpentine is, in my judgment, of unquestionable value in certain cases. Not only is it a powerful antiseptic and a good stimulant, but its action on the mucous membrane in properly selected cases is excellent. The symptoms which call for its use in preference to the other remedies of this class are a dry, brown tongue with tendency to sordes; abdominal distension without much diarrhœa; considerable bronchial catarrh, and cardiac and muscular weakness. I venture to believe that no one can have given this remedy in suitable form and dose under the above conditions without sometimes observing, as I have done repeatedly, such prompt and positive improvement in all the symptoms as could only be attributed to its action. It may be used according to this formula:

℞. Ol. terebinthinæ, f℥iij;
Pulv. acaciæ,
Sacchari, āā. q. s.;
Sp. lavandulæ comp., f℥iij;
Aquæ, q. s. ad f℥vj.
Ft. mist. sec. artem.
Sig. One to two teaspoonfuls in a little water every three hours.

If the stomach be irritable, it is well to reduce the dose of turpentine and to add to each dose one or two drops of deodorized laudanum.

In place of the oil of turpentine a capsule containing from two to four grains of white turpentine may be given every three hours.

Calomel has often been used as an intestinal antiseptic, and it has been claimed, without any sufficient grounds, that the disease may be aborted by it when given in small doses day after day. The only purpose for which it is to be recommended is, however, as a sedative to the stomach and as one of the

mildest and most manageable of laxatives; so that if, either at the beginning of the case or at any time during its course, a laxative is indicated, fractional doses of calomel (gr. $\frac{1}{10}$ every two hours) may be used until a single movement is secured. The view that the condition of the intestine or the course of the disease may be influenced favorably by purgative doses of calomel or of salines is totally opposed to my own experience and opinion.

A mixture of two parts of tincture of iodine with one part of carbolic acid has also been recommended to produce intestinal antisepsis, and has found some acceptance. One to three drops of the mixture may be given three times a day. Yeo recommends chlorine-water and quinine for the same purpose. Murchison years ago, and Schönlein before him, spoke highly of chlorine in this disease. Naphthalin has been recommended by Rosenthal and others following him. Very excellent results were reported by Wolf, the disease seeming to run an abortive course in 16 out of 100 patients. The dose should be from 15 to 60 grains a day. During its administration the urine becomes very dark in color, but this is claimed to be a matter of no significance.

The employment of thymol has been urged very strongly by certain writers. Henry is even convinced that the typical symptoms of typhoid fever will rarely develop if the drug be administered during the first week of the attack. It should be given in pills freshly made with medicinal soap. One or two pills, each containing $2\frac{1}{2}$ grains, may be given every three hours. Sufficient water should be swallowed after each pill to ensure its passage out of the œsophagus, as otherwise disagreeable burning may result. Salol has been advocated by a number of writers, and undoubtedly possesses decided power in producing intestinal antisepsis. Other derivatives of salicin have likewise been recommended for the purpose, as have also iodoform, the sulphites, sulphocarbolate of zinc, α-naphthol, β-naphthol, and corrosive sublimate.

Treatment of Special Symptoms and Complications.—As would be expected, some of the special symptoms or complications in typhoid fever usually require treatment directed particularly toward them. Fortunately, it will be found that in proportion as rigid rest and diet are insisted upon, and as proper measures for the control of fever and for the treatment of the intestinal condition are adopted at the outset of the disease, the subsequent development of grave symptoms or of serious complications will be correspondingly infrequent.

Headache is best managed by strict quiet and darkening of the room. Cold may be applied to the head, or menthol, chloroform liniment, camphor, or the like may be used locally. If these fail, potassium bromide may afford relief, or small doses of antipyrine (gr. v) or of the effervescing granulated antipyrine and sodium salicylate (ʒij, containing 4 grains of each) or of phenacetin (gr. iij) may be given. Finally, a suppository of ext. opium gr. $\frac{1}{2}$, with quinine, 5 or 8 grains, may be used if the pain be not allayed by other means.

Insomnia sometimes demands active treatment in order to prevent nervous exhaustion. Codeia (gr. $\frac{1}{4}$ to $\frac{1}{2}$), sulphonal (gr. x or xv), potassium bromide (gr. xv) with chloral hydrate (gr. vj), have proved the most useful in my

experience. An enema of deodorized laudanum or a suppository of the extracts of opium and hyoscyamus may be required, or even a hypodermic injection of morphine (gr. $\frac{1}{8}$ or $\frac{1}{6}$) with a minute dose of hydrobromate of hyoscine (gr. $\frac{1}{120}$ or $\frac{1}{90}$).

Delirium and somnolence are so often associated with high fever that hydrotherapy, applied as already described, is generally essential and most valuable in their treatment. Suppositories containing asafœtida and quinine, gr. x each, may be given two or three times daily. If the delirium be active or violent, small doses of hyoscine (gr. $\frac{1}{100}$ to $\frac{1}{80}$) may act admirably. Large doses may prove depressing. Codeia is often useful, quieting delirious excitement without inducing stupor. Potassium bromide with elixir of valerianate of ammonium is of value, especially in cases of hysteroidal type. When the delirium tends to be of the low muttering type, camphor or musk may be of service. The latter is of very uncertain action. To be of any benefit it must be of the best quality and be given in large doses, and unless it acts promptly it is useless to continue it. Carbonate of ammonium is much less used now than formerly, but may occasionally be given with advantage in this condition in the dose of gr. v every two or three hours in the form of emulsion. Ice to the head is here also useful. A hot mustard foot-bath may induce relaxation and quiet. Blistering the nape of the neck, or even the shaved scalp, has been recommended, and frequently practised when the cerebral symptoms are aggravated. It never seems to me proper to use this painful measure, and I fear it usually does more harm than good.

Patients with typhoid fever always require close watching, but when delirium is present they must not be left alone for an instant. They frequently endeavor to leave the bed, and will even struggle violently to do so. Before resorting to mechanical restraint all the resources of kind and skilled nursing must be exhausted. It may be judicious to let the patient rise to the sitting posture or sit on the edge of the bed for a few minutes, after which his delirious restlessness may be for the time allayed, and he will sink back on the bed in a more quiet state. I have even been led in rare cases to allow the patient to rise to his feet and to be placed in an easy-chair close to the bed, and have observed good results to follow. If restraint be unavoidable, it is best applied by passing a sheet over the body and fastening it under the bed.

Vomiting at the outset of the disease should be treated by withholding food and medicine, save fractional doses of calomel, alone or combined with bismuth, or minute doses of nitrate of silver, of creasote, or of dilute hydrocyanic acid. If the stomach be very irritable, a mild emetic may be useful. When vomiting occurs later, it usually shows that the food or medicine disagrees. The milk should at once be reduced in amount, and be diluted with lime-water or carbonated water, or milk may be temporarily suspended, and small amounts of dry champagne, liquid peptonoids, or light broths be administered. Very hot water taken in spoonful doses, small pieces of ice swallowed, or some of the remedies above mentioned may be tried. Even if vomiting occur only once a day or every other day, it shows that the stomach

is in a state of continuous irritation, and a change of food or of medicine, or of both, is required.

Diarrhœa is one of the symptoms which most frequently demands attention. Even if the bowels are quiet, so that movements must be encouraged, a little carelessness in feeding or medication is apt to bring on looseness. Actual constipation is comparatively rare, though more common than usual in some epidemics. If the bowels are quiet, therefore, it is better to use a simple enema every other day. If there is looseness it is necessary to decide by the number and size of the movements whether anything shall be done to check it. As a rule, it is not necessary to interfere if the movements do not exceed two in number daily, and are not very large and liquid. The stools should be inspected in order to determine whether undigested food is being passed, and if so, the diet should be modified and restricted before recourse is had to remedies to check the looseness. Beef tea or other strong meat broths, for instance, may increase the tendency to looseness; and when this exists milk properly diluted and in suitable amounts should be depended on exclusively in most cases. The pills of nitrate of silver with opium are very efficient in controlling diarrhœa. Small doses of acetate of lead with opium are also valuable. The following may be used with advantage:

℞. Bismuthi subnitratis, ʒiij;
Pepsin. saccharat., ʒij;
Morphinæ sulph., gr. j.
M. et div. in chart. xx.
Sig. One powder every three or four hours.

It is, as a rule, judicious to suspend other medication while using any of these astringents; but if the latter are not effective, or if it seems essential to continue other remedies by the mouth, the diarrhœa should be controlled by enemata of deodorized laudanum in starch-water or by such suppositories as—

℞. Pulv. iodoformi, gr. xxx;
Acid. tannici, ʒj;
Ext. opii aq., gr. v;
Ol. theobromæ, q. s.
M. Ft. mist. et div. suppos. xx.
Sig. Use as required.

Or,

℞. Plumbi acetatis, ʒj;
Ext. opii, gr. v;
Ol. theobromæ, q. s.
M. et div. in suppos. xx.
Sig. Use as required.

On the other hand, constipation may demand treatment. We have seen that the existence of ulceration, even so deep as eventually to cause perfora-

tion, is consistent with a torpid and constipated state of the bowel throughout the course of the disease. Modification of the diet may effect a change. Occasional short courses of small doses of calomel or of a mild saline may be required, but, on the whole, small enemata are to be preferred to laxatives. I have found the simple white-wheat gluten suppositories sufficient in some cases, or glycerin in the form of suppository or of enema will be more active.

Tympanites may become so extreme as to cause great distress and to add materially to the danger of the case by pushing up the diaphragm and embarrassing the heart and lungs. It is most serious when paretic in nature and due to degenerative changes and weakness of the muscles of the abdominal and intestinal walls and of the diaphragm. In other cases it is due to excessive development of gases in the intestine from decomposition of the food and morbid secretions. Under the latter circumstances more rigid restriction of the food, small, frequently repeated doses of such antiseptic remedies as turpentine, thymol, or naphthalin, mild laxative doses of calomel or of castor oil with turpentine in emulsion, and the external use of turpentine stupes, are to be considered. When the distension seems largely due to weakness of the muscles, an increase in the amount of stimulus may relieve it; and with this may be joined full doses of strychnine by the mouth or injected hypodermically into the abdominal walls. Enemata containing turpentine or asafœtida are also valuable, or, as a last resort, a soft rectal tube may be introduced very carefully as far as possible into the colon.

Abdominal pain often accompanies great tympanites. It usually is only the result of the excessive distension, but occasionally it is so acute and intense as to indicate the existence of localized peritonitis. It is indeed not improbable that in many cases of extreme tympanites there are patches of plastic peritonitis which further aid in inducing paresis of the intestinal walls. In the case of the lad referred to on page 66 the fact that the extraordinary and prolonged distension was thus caused was shown by the subsequent presence of a large indurated mass, doubtless composed of enlarged glands, agglutinated coils of intestines, and plastic exudation, which required many months to disappear.

Peritonitis, unless of circumscribed character, is a very fatal complication, whether it result from perforation of the bowel or from extension of inflammation from the base of deep ulcers. It is to be treated by the most absolute rest, on a water-bed if possible; by opiates by the mouth or hypodermically, in such doses as to maintain a proper constitutional effect; by cold to the abdomen; and by the smallest possible amount of food and drink, and at first only cracked ice and dilute stimulants. Strychnine may be given with morphine hypodermically. If collapse is threatened, external heat and ether hypodermically are to be tried. The whole effort must be to maintain life and to keep the bowels quiet, so that if perforation have occurred the development of adhesions may be favored. The same treatment is appropriate if peritonitis has arisen without perforation; and it is always presumable if recovery ensues that such has been the case, though the possibility of recovery after perforation is not

to be absolutely denied. It is questionable whether laparotomy is ever justifiable in typhoid fever, and whether the patient's chances for life are not better without it. The statistics of the operation during the course of the disease are most unfavorable; and it is only when, during convalescence, peritonitis suddenly develops with the symptoms of perforation that I should sanction its performance.

Intestinal hæmorrhage presents most difficult questions in treatment. If it be small in amount, without apparent tendency to recur, and the general symptoms show no sign of depression or shock, it is unnecessary to pay any attention to it. Harm may be done by instituting at once active treatment with irritating and depressing astringents. Cold to the abdomen may, however, always be applied with safety and advantage in the form of Leiter's tubes or the ice-bag. In every case of intestinal hæmorrhage absolute rest must be insisted upon, a folded cloth being substituted for the bed-pan, so that even the lifting of the nates may be avoided. If the hæmorrhage be larger, if the blood be dark as though coming from high up in the intestines, and if there be any evidence of depression or shock, in addition to the external use of cold small pieces of ice may be swallowed and a small enema of cold, or even of iced, water may be used. Hypodermic injections of ergotin in large doses form in my judgment the most reliable hæmostatic treatment. Acetate of lead and opium or aromatic sulphuric or gallic acid may be given by the mouth. I have known very large doses of oil of turpentine (f℥j in emulsion in thirty-six hours) given with marked success in a case under the care of Dr. Batt, where six large hæmorrhages on the twenty-first and twenty-second days had induced most alarming collapse, with a fall of temperature from 105° to 97°. Stimulants should be given freely if collapse threatens, but ether and strychnine hypodermically are the most prompt and powerful remedies to induce reaction. Recovery may occur from an apparently moribund condition, so that we should never relax our efforts in this condition so long as life is still present.

If the loss of blood has been great, transfusion of blood and intravenous injections of a warm solution of sodium chloride have been used with occasional success. When a hæmorrhagic diathesis develops, as it may do late in the course of grave cases, the tincture of the chloride of iron and turpentine have seemed to me most useful.

Epistaxis profuse enough to demand interference is unusual. In exhausted conditions, however, even small losses of blood are dangerous. The best treatment is by ice to the nose, forehead, and nape of the neck; ergot, turpentine, or oil of erigeron internally; insufflations of alum or tannic acid; and, finally, as a last resort, by plugging the nares.

Heart failure is chiefly to be guarded against by systematic reduction of the temperature and by the use of alcoholic stimulants and of strychnine in full doses. In an emergency either alcohol or ether may be given hypodermically. Tincture of digitalis in small doses, 5 or 6 drops every two or three hours, or tincture of strophanthus in equal amounts, may be useful; but I have seen no

good result from large doses of these remedies in the heart failure of fever. On the contrary, harm may result from pushing their administration freely. Nitro-glycerin (gr. $\frac{1}{100}$ to $\frac{1}{50}$) or caffeine (gr. ij, repeated at short intervals) is more valuable. Either may be given by the mouth or hypodermically, as may also camphor dissolved in olive oil. I would lay especial stress on the systematic use of strychnine in moderate doses when there is a tendency to cardiac or respiratory failure, and in large and oft-repeated doses hypodermically when such failure is imminent or actual. Reference may be made to the case reported on page 88 as showing to what extent and with what happy result its use may be pushed.

Hypostatic congestion of the lungs, extensive bronchial catarrh and pulmonary œdema, and hypostatic pneumonia are all apt to be associated with high temperature and a tendency to cardiac and respiratory failure. We have seen that hydrotherapy neither tends to induce these complications nor is contraindicated by their existence. Frequent changing of the position of the patient is useful in prophylaxis and in treatment. Dry cups may be applied repeatedly to the chest posteriorly, and I have even used a few wet cups with good effect. Frequent deep inspirations or inhalations of oxygen are also of value. Finally, strychnine and quinine in solution with mineral acids (as in the following formula) may be given with great advantage:

℞. Quininæ sulph.,	ʒj;
Strychninæ,	gr. j;
Acid. muriatic. diluti,	fʒiij;
Tr. cardamomi comp.,	fʒiij;
Aquæ puræ,	q. s. ad fʒv.—M.

Ft. sol. et filt.

Sig. A teaspoonful in water every four or six hours.

Carbonate of ammonium in emulsion (gr. 3 to 5 every two or three hours) or turpentine is often useful.

Bed-sores are to be avoided by carefully keeping the surface clean and dry, by preventing too continuous pressure on prominent parts, and by bathing the skin at these parts with some such astringent wash as alum in alcohol. If the case is protracted and severe, a water-bed will be of great service in aiding to prevent bed-sores, as well as in their treatment if unfortunately this complication has developed. The best local applications are intended either to protect the ulcerated surface, as soap plaster, or to exert an antiseptic and healing action, as in the case of ointment of iodoform or boracic acid, or powder of iodoform and bismuth, or the following, recommended by Dr. Beverly Robinson:

℞. Ichthyol,	ʒj;
Flexible collodion,	fʒj.—M.

Sig. Use locally.

Thrombosis of the femoral veins should be treated by the daily application of tincture of iodine along the line of the vessel, by enveloping the leg in raw cotton from the ankle to the groin, holding this in position by a light bandage, and by rest and elevation of the member. In the early stage of the case great care should be used in manipulating the part, lest a fragment of the clot be detached. Later inunctions may be used of dilute mercurial ointment or of the following:

℞. Ichthyol,
Lanolini, āā. ʒij;
Ung. iodini comp., ℥ss;
Petrolati, q. s. ad ℥iss.—M.

When all tenderness has disappeared and the swelling has subsided, so that it is safe to let the patient leave bed, a long stocking of elastic webbing should be worn, and in many cases the use of this must be continued for a number of months.

It is well to allude again to the importance of examining the region of the bladder morning and evening during the continuance of the typhoid state to guard against retention of urine.

Treatment of Convalescence.—The management of the case during convalescence has already been partially discussed. Although the appetite returns and the patient clamors for food, the greatest care must be observed in this respect. No solid food should be allowed for at least ten days after all fever disappears, as a slight indiscretion in diet may readily cause the return of the fever. It is not necessary, however, to persist in a strictly milk diet.

Continued rest is of equal importance, as the danger of perforation is by no means past. It should be maintained for at least a week after all fever has ceased. It is to be borne in mind, however, that in some cases a slight evening rise of temperature persistently continues without apparent cause, and that this may sometimes be brought to a stop by allowing the patient to leave the bed.

Caution must be exercised in permitting even slight excitement during convalescence. Visits of friends are very apt to be followed by rise of temperature. Tonics, such as iron, quinine, strychnine, and cod-liver oil, are useful at this period. Stimulants, too, may often be given advantageously. It is well, however, to exercise caution in their use, lest an intemperate habit be acquired. A change of air is also to be highly recommended.

TYPHUS FEVER.

By WILLIAM PEPPER.

Definition.—Typhus fever is an acute infectious and highly contagious fever, endemic and epidemic, presumably due to a micro-organism as yet undetected, attended with no characteristic lesions of the solids, but with grave alterations of the blood, and marked clinically by an abrupt onset, a maculated and petechial eruption, and continued high fever for twelve or fourteen days, terminating usually by crisis.

Synonyms.—Exanthematic typhus is the name applied to this disease by the Germans, in contradistinction to abdominal typhus, by which is meant typhoid fever. This is also used at times by the French. Petechial typhus; Spotted or Putrid fever; Hospital, Jail, Camp, or Ship fever, are among the best known and most appropriate of the older names. They indicate marked clinical features of the disease or else the conditions under which it has often developed.

History and Etiology.—While the ignorance or neglect of sanitation made every war more fatal by its diseases than by its battles, while vessels were often floating pest-houses, and jails enclosed as much physical as moral filth, outbreaks of typhus were common. From the earliest accurate account of the disease, given in 1546 by Fracastorius, when he described the Verona epidemics of 1506 and 1508, there are numerous reports of violent and widespread outbreaks in all parts of Europe. It is indeed difficult to believe that prior to the above dates the same thing did not occur. During the past fifty years, although typhus has frequently appeared, it seems possible to note the effect of sanitary regulations in tending to restrict the spread of the epidemics.

That the poison is constantly present in its favorite habitat is shown by the occasional occurrence of sporadic cases in crowded cities. It cannot, however, be said that the disease is endemic except in comparatively few places, such as certain parts of Great Britain, especially London, Dublin, and Glasgow; in Brittany in France; in the provinces of the Danube and the Baltic, etc. In America it has appeared for the most part in the form of limited outbreaks in seaboard towns, following the importation of the virus from abroad by fomites or by actual cases of the disease. Independent outbreaks of the disease have, however, occurred in a few instances. An epidemic occurred in New England in 1807, and one in Philadelphia in 1812, after which the elder Wood states that occasional cases developed in the slums of that city until 1820–21. Gerhard gave an admirable account of the epidemic he studied in

the same city in 1836, and subsequent outbreaks have been described by Flint, DaCosta, Loomis, and others. Although doubt has been cast on the diagnosis in many of the 1723 cases reported to the Surgeon-General's Office during the Civil War, 1861–65, typhus apparently occurred at various points, and chiefly, I believe, among returned prisoners of war. In the winter of 1864–65 I had the opportunity of studying a circumscribed but severe epidemic, coincident with an epidemic of variola, in a crowded and unhealthy portion of Philadelphia, where I then served as district dispensary physician. As far as I could determine, both diseases had been introduced by returned soldiers and deserters. In 1866 I again saw much of the disease in the medical wards of the Pennsylvania Hospital, while resident physician in that institution; and during seventeen years of service as visiting physician to the Philadelphia Hospital, from 1867 to 1884, there were several outbreaks of typhus, during which a considerable number of cases were admitted there. In 1880 a fatal outbreak occurred at the town of Blackwood, New Jersey. I had the opportunity of studying it, and it has been well reported by Dr. Branin. The disease was introduced to the almshouse there by a sailor who came from a lodging-house in Philadelphia where there were several cases of imported typhus. The sailor developed the disease after his arrival at the almshouse. It spread at once among the inmates. At first the cases were of mild type and distinguished with difficulty from typhoid. Severe weather came on, the sanitary condition of the almshouse became very bad, and the disease assumed a grave type. In the early part of the outbreak all the cases recovered; later the mortality was almost 50 per cent.; in all there were 103 cases, with 33 deaths.

The causes which predispose to typhus fever are famine, destitution, overcrowding, and filth. Whatever tends to reduce vitality and lessen resisting power, such as intemperance, overwork, depressing passions, renders the system more sensitive to the virus. Attacks occur, therefore, chiefly among the lower classes and in the overcrowded and dirty sections of large cities.

Age exerts no special influence, and the disease may occur in infants and in old persons. Naturally, the larger proportion of cases in all epidemics will be found among young adults and the middle-aged, as more subjects are exposed to the causes of the disease at these periods of life. Sex is equally without definite influence, and so is occupation, unless, as in the case of physicians, nurses, and clergymen, it may bring them in contact with patients suffering with this contagious malady. Laundresses who are called on to wash the linen of typhus-fever patients are also peculiarly liable to be affected. Epidemics may occur at all seasons and in most varied localities, but they are most frequent, as already stated, in seaboard towns and during the winter, when the ventilation and cleanliness of houses are apt to be most defective.

The *exciting cause* of typhus is exclusively the specific virus. The extreme contagiousness of this disease is so fully established that the evidence need not be recited. Murchison formulates the conclusions as follows: When typhus appears in a house or a locality it usually spreads with great rapidity; the

number of cases in the house or in the circumscribed locality is in direct proportion to the relation between the well and the sick; individuals living in localities where the disease is unknown acquire it on visiting typhus patients in a distant locality; the disease is often imported by infected persons into previously healthy localities; and, finally, the contagiousness of typhus is established by the success of prophylactic measures, and especially by the isolation or removal of the earliest cases.

The virus may acquire intense energy. It is not necessary that there should be actual contact with the sick, and yet the distance through which the poison can exert its influence is limited. Brief visits to a single case may be made with impunity, but if several cases are confined in one room the air becomes so infected that those who enter are apt to contract the disease though they may not go within several feet of the sick. A large proportion of persons unprotected by a previous attack contract the disease when first exposed to it. As would be expected, therefore, many physicians and nurses lose their lives during large epidemics. There is, however, great difference in the susceptibility of different individuals and of the same individual at different times. Thus, one of my nurses, who had passed unscathed through previous epidemics of typhus, and in the severe outbreak here in the winter of 1864–65 had been most faithful and devoted in his care of many cases in the fever ward, escaped until May 1, when he suddenly developed a malignant attack, and died in four days. The disease is contagious throughout its entire course, and it is difficult to say if it be more so at one period than another.

Although it is evidently difficult in such cases to exclude other sources of infection, it seems that typhus may be contracted from the corpses of those who have died of that disease. I shall never forget the sudden impression made upon me as I dissected the body of a subject dead of malignant typhus in 1866. It seemed as though a thick, strong vapor rose from the open surfaces and struck me in the face. Within ten minutes I was too giddy and weak to walk; a chill occurred within an hour, a high fever followed immediately, and an attack of moderate severity ensued. I had, however, been in almost daily contact with typhus patients for a year previously.

The avenue by which the infection gains access to the system is not definitely known. There is no evidence to show that it is by contamination of drinking-water or other ingesta. Analogy is opposed to the view that it is through the skin. It seems probable that it is by way of the inspired air that the disease is contracted. The poison attaches itself tenaciously to clothing and bedding, and the fever may be thus communicated by fomites at considerable intervals of time and space. An attack of typhus protects strongly and usually permanently against subsequent attack. Both relapses and recurrences are extremely rare.

No typhus microbe has as yet been discovered. Hlava in 1888, at Prague, found a streptococcus which he was disposed to believe peculiar to the disease. But he found it only in 20 out of 33 cases, and Cornil, Thoinot, and others are not disposed to regard it as specific. Thoinot gives fresh experi-

ments to confirm the view that the blood of typhus patients will not convey the disease to animals by inoculation.

The effect of overcrowding, defective ventilation, and filth in increasing the virulence of the poison is so decided that the question has naturally arisen if under such influences it may not arise *de novo*. This view has been espoused by some high authorities, but if the poison is, as is probable, associated with a microbe, all analogy is opposed to its spontaneous generation. A more plausible suggestion seems to be that the microbe, which may be widely distributed, and under ordinary circumstances possessed of but moderate pathogenic properties, acquires, when cultivated in contact with the foul effluvia of human beings overcrowded, filthy, and degraded in vitality, such virulent properties as make it the effective cause of typhus fever.

Morbid Anatomy.—There are no lesions of the solids peculiar to typhus fever. As a rule, rigor mortis is not marked. Putrefaction occurs rapidly after death. The petechial eruption persists after death, and large purple patches are present on the dependent portions of the body.

The blood is profoundly altered. It is dark and fluid; the lining of the aorta is deeply stained by imbibition; such clots as are present are soft and dark like currant jelly. Ecchymoses may be seen on all the serous membranes, and especially on the pericardium. The muscles are dark and, notably in the case of the heart, have undergone granular degeneration. Extravasations of blood are occasionally noted in the substance of the muscles, more frequently in the recti than elsewhere. The liver is softened and somewhat swollen. The spleen is enlarged, though usually not so much so as in typhoid: its pulp is greatly softened, even to diffluence in many cases. In some of the cases I observed it was from three to five times its normal size and extremely soft. The kidneys are swollen and enlarged, and may show the changes of infectious nephritis. Congestion and catarrh of the larynx and bronchial tubes are common. Hypostatic congestion of the lungs is very frequently, and pneumonia not rarely, met with: the latter may be either croupous or catarrhal. Pleurisy, either sero-plastic or purulent, is an occasional complication. On the whole, the lesions of the respiratory organs are much less constant and pronounced than in typhoid fever.

The gastro-intestinal tract presents no characteristic lesions. Congestion and occasionally ecchymoses of the mucous membrane of the stomach may be noted. Peyer's patches may be slightly swollen and present the shaven-beard appearance, but not in a degree greater than is occasionally found in all violent infections. The solitary glands also are sometimes unduly prominent. The Breslau epidemic, in which Lebert reports the occurrence of small ulcers of the solitary and agminated glands, was certainly exceptional. In the Philadelphia epidemic of 1864–65, where diarrhœa occurred in fully one-third the cases, some enlargement of Peyer's patches and of the solitary glands was found repeatedly, but no ulceration was reported. A tendency to general hyperplasia of the lymphoid tissue is present, but in a much less marked degree than in relapsing or typhoid fever. The cerebral meninges are usually congested and

the sinuses filled with dark blood. A moderate amount of subarachnoid and ventricular effusion of serum may exist, but not to a greater degree than in acute diseases unattended with the intense nervous disturbances of typhus. Organic lesions of the nervous centres are conspicuously absent as a rule.

General Clinical Description.—The incubation of typhus fever varies from a few hours to two weeks or even longer, according to the virulence of the infection and the susceptibility of the individual. Twelve days may be regarded as a safe average. Prodromes are more often absent than present. There may be a feeling of general indisposition, with weakness, vertigo, and loss of appetite, for two or three days. The invasion of the disease is as a rule abrupt, with sudden vertigo, rigor or actual chill, extreme weakness, and rapid rise of temperature. The patient is forced to take to bed at once in most cases. Headache, pains in the back and limbs, and soreness of the flesh appear speedily. Nausea and vomiting are not rare. The tongue is moist at first, with but slight coating. The abdomen is not distended, and constipation is usual. Epistaxis is rare. The expression is heavy and like that of one intoxicated. The face is flushed uniformly, and the eyes are congested. Delirium may occur almost at once, and serious nervous symptoms speedily ensue. Prostration may appear early, and is so marked as to be highly characteristic.

The fever rises so rapidly that a temperature of 104° or 105° may occur on the second or third day, and this may be the highest point attained during the attack. The daily variations are not marked. The sense of heat imparted to the hand even exceeds the degree actually present. (See Fig. 10.)

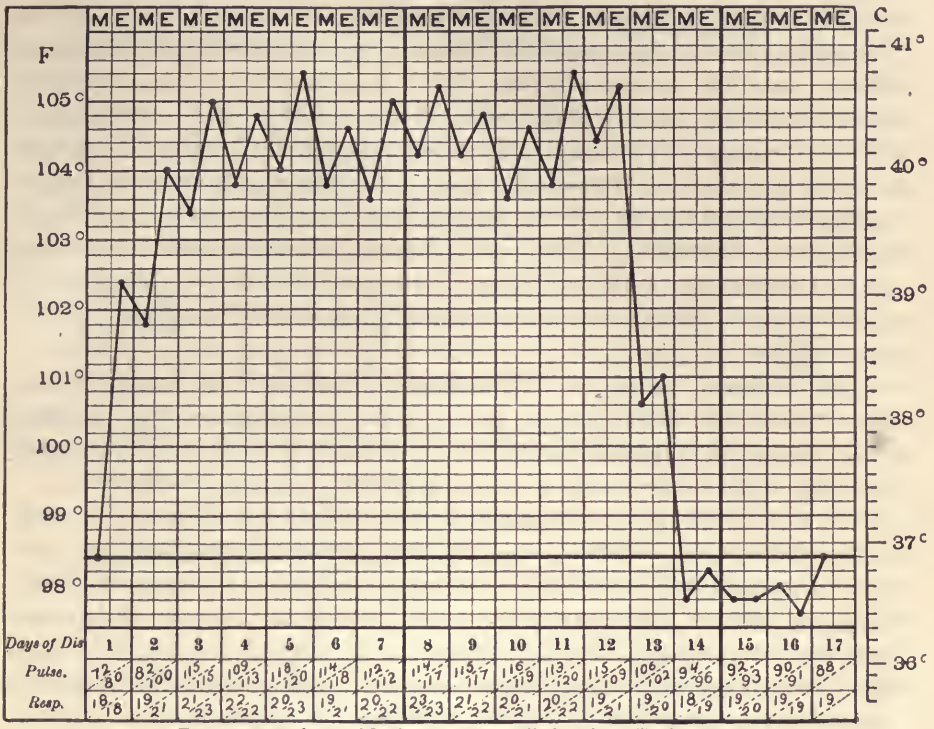
The pulse is rapid from the first. Not rarely it reaches 110 or 120 by the third day, and this rapidity increases as the disease advances. Even if full and strong for a day or two, the pulse speedily grows small, soft, and compressible, and the heart's action is found to fail rapidly in force. The respirations are hurried, in accordance with the height of fever and the acceleration of the pulse. If any pulmonary complication develops, the disturbance of pulse and breathing may become extreme.

On the third or fourth day the typhus eruption makes its appearance in the form of numerous irregularly-rounded spots, of a dull-red color, barely elevated above the skin. These disappear on pressure at first, but soon it is found that the centre persists on pressure, and later the entire spot is converted into a petechia. It is preceded or accompanied by an irregular congestion of the derm which causes a subcuticular mottling.

By the end of the first week the disease has reached its height. The stupor from which the disease takes its name is pronounced. The decubitus is dorsal, and the patient must from time to time be turned on either side. There is a deep dusky flush of the face, and the expression is profoundly dull. It is often impossible to rouse the patient to answer. Delirium may be low and muttering or wild, excited, and noisy. Despite the stupor, sleep may be almost absent. The prostration and muscular weakness are extreme. Tremor, difficulty in protruding the tongue, retention of urine, slipping down in the bed, and inability to turn are often present. The severe headache of the earlier

days has subsided, or, if it continues, may be associated with muscular rigidity and retraction of the head. The fever continues uniformly high, with a dry

FIG. 10.



Temperature-chart of Patient, æt. 19, suffering from Typhus Fever.

burning skin. The pulse is small, weak, and rapid; the impulse and first sound of the heart are almost effaced. The tongue is dry and brown, tremulous, and protruded with great difficulty if at all. Liquid may still be taken freely, and digestive disturbances are for the most part wanting. Some enlargement of the liver and spleen is present. The urine is scanty, concentrated, highly colored, and often albuminous. No second crop of eruption has appeared; the spots have become to a large extent petechial, and after the ninth or tenth day begin to change color and fade. The alarming or even desperate condition of the patient grows aggravated as the limit of the disease approaches. A sudden critical improvement occurring about the end of the second week, with a rapid fall in fever and abatement of all symptoms, ushers in a speedy and uninterrupted convalescence. Or, on the other hand, complications arise and may determine a fatal issue to the case, or the symptoms of infection and of cerebral or cardiac failure progress, and death occurs from the middle to the end of the second week.

Special Symptoms.—The appearance of the patient is highly characteristic. It is true that in some cases of typhoid fever and in some of cerebro-spinal fever a similar facies appears, but much more constantly in typhus is there a uniform deep dusky flush of the face, with a glazed appearance of the

skin, a finely-injected eye with contracted pupil, and an expression occasionally wild and fierce, but usually veiled, heavy, and profoundly dull.

The nervous symptoms of typhus fever are most severe and characteristic. Muscular debility is extreme from the very first, so that it is uncommon to have patients walk about for more than a few hours after the invasion, although cases are occasionally met with of such mild type as to present themselves at the out-door department of a hospital with the rash already out. It is commonly associated with great vertigo, which makes the patient still more helpless, and throughout the case this latter symptom may be distressing, causing a frequent feeling that the bed is sinking or swimming away. The prostration increases as the disease advances. It shows itself in the tendency to heart failure, in the retention of urine, in difficulty in protruding the tongue or in swallowing, in inability to turn in bed, in a high degree of tremor and subsultus.

Pain is often severe in the early stage. Headache is its most constant form: it may be intense, and in cases of the cerebro-spinal type is associated with rigidity of the muscles of the neck and retraction of the head. Cutaneous hyperæsthesia and muscular soreness may also be marked.

It is only in very exceptional cases that delirium is absent, though it varies much in its degree. As a rule, it appears early and continues throughout the case. It may be mild and muttering or noisy and wild. Hallucinations may occur, and patients have given me apparently coherent accounts of the most improbable occurrences. In one instance a woman of excellent character accused herself of lewd thoughts and conduct in her past life, and repeated these accusations daily through a considerable part of the attack, though it was apparent on her recovery that no recollection of such remarks was retained. It required decisive explanations to avoid unpleasant social results, so emphatic and plausible had been her accounts. Stupor is equally common, and varies from hebetude to profound coma. Only in very mild cases is there absence of the peculiar mental confusion and heaviness with besotted expression of countenance from which the disease gets its name. Oscillation of the eyeballs is an unfavorable symptom occasionally met with. Convulsions may occur at the onset in children without necessarily grave significance: occurring in adults toward the close of the second week, they are evidences of intense toxæmia, often with renal complication, and are ominous. In spite of the stupor, true refreshing sleep is apt to be wanting, and insomnia may persist to a dangerous extent. Careful attention to this point is always demanded, as fatal exhaustion may be induced by prolonged watchfulness.

The fever runs a course of about two weeks. It is ushered in by a rigor which may be slight or may amount to a hard chill. The latter is far more frequent than in typhoid. The initial rise of temperature is sudden and high, and 103° is often, and 104° occasionally, reached by the evening of the first day. Wunderlich reports 104.9° in one case at that period, and Lebert 106.4° on the second evening. The maximum of the case is reached from the third to the fifth day. It varies from 103° in mild cases to 105° in severe cases, and

even to 106° in the grave form. In one case at Blackwood, N. J., it was 109° on the fourth day, and yet the patient ultimately recovered. The remissions, which usually occur in the mornings, are apt to be but slight; the high fever persists with great uniformity. During the second week the fever rarely exceeds the maximum of the first week unless inflammatory complications occur, or in fatal cases, where death is often preceded by a sudden rise to 106°, or even to 109°. About the twelfth day in favorable cases a crisis occurs, marked by an abrupt fall in temperature, and often by some critical discharge. Thoinot states that there is never a sudden fall in the temperature, but this is totally opposed to general experience. I have seen it drop five degrees in twenty-four hours, and even more abrupt and extensive falls are recorded. The temperature often becomes subnormal and remains so for several days.

The symptoms furnished by the digestive system in typhus are usually unimportant. The tongue may remain moist and but slightly coated in mild cases, but more commonly it becomes dry and brown or even blackish and cracked. It is tremulous, and in bad cases it is difficult or impossible to protrude it. Sordes form abundantly on the teeth and lips.

The appetite is variable. Usually there is anorexia, but thirst is preserved. Occasionally relish for food is not entirely lost at any time during the case. When stupor is marked no request for nourishment or water may be made, though they will be swallowed willingly when offered. Nausea may exist with the intense vertigo at the onset, but both it and vomiting are rare symptoms during the disease. Uræmia or cerebral irritation may induce vomiting toward the close of the case. The abdomen is not distended, though, on the other hand, retraction is rare. The condition of the bowels varies in different epidemics. Constipation is the rule, and I have often known it difficult to secure satisfactory movements with simple enemas. A typical typhoid state develops in some cases, with meteorism and looseness of the bowels, but even then neither ochre-colored stools nor intestinal hæmorrhages occur. In some epidemics, as at Philadelphia in 1864–65, diarrhœa may be present in fully one-third the cases.

The liver is occasionally somewhat swollen, the spleen much more frequently so, but to a less degree and with less constancy than in typhoid fever. Tenderness on pressure is apt to exist over the liver or spleen if enlarged.

A rapid pulse is nearly always present. Its rate corresponds with the height of fever and with the degree of disturbance of the cardiac ganglia and muscle. In mild cases with moderate fever it may at no time exceed 96 or 100. Occasionally a disproportionate slowness may be noted, and if this be unaccompanied by alarming uræmic or cerebral symptoms, it is favorable, as indicating a large reserve of cardiac power. But usually the pulse-rate rises rapidly from the onset and varies between 110 and 120 in cases of ordinary severity. In children, in sensitive females, and in grave cases a pulse of 136 to 160 is not uncommon. Sudden slowing of the pulse without fall in temperature or improvement in nervous symptoms is of serious significance. When the critical fall of temperature occurs, about the close of the second week, the

pulse-rate should fall decidedly, though not so rapidly. Abnormal slowness to 60, or even to 48, is not rare during convalescence. In one case in the Blackwood epidemic the pulse on the twentieth day was 24, with respirations 16 and temperature 98°; recovery followed, and by the thirty-second day the pulse had attained its normal rate. Excitement or exertion will rapidly send the pulse-rate up again, however, and this undue mobility of the heart may last for weeks. When the muscular substance or nervous ganglia of the heart are seriously involved the pulse may be so rapid, small, and irregular as to be uncountable; the pulse taken at the wrist may differ widely from the count of the cardiac impulses at the præcordia, and, if life be spared, pronounced weakness and irritability of the heart may persist long after recovery is otherwise complete.

The character of the heart's action and pulse is even more important than the rate. In mild cases and in the young and strong the pulse may retain fulness and force; but there is pronounced tendency to failure of cardiac power, and as a rule the pulse grows soft, small, and compressible in two or three days, and from that onward its weakness increases. For some time before the crisis, in very grave cases which nevertheless recovered, I have been unable to detect the pulse at the wrist. Dicrotism is less common than in typhoid.

The cardiac impulse soon grows weak and diffuse; it becomes impossible to count the apex-beats by palpation; the first sound is altered in character, becoming short, clear, and valvular, and ultimately almost inaudible, owing to increasing impairment of the ventricular contractions. A blurred or murmurish character of the first sound is also often present, but actual endocarditis is rare. These changes indicate, and to some extent measure, the dyscrasia of the blood, the degeneration of the cardiac muscle, and the failure of innervation.

The pulse-respiration ratio is fairly preserved, so that the breathing is usually 30 to 40 in the minute. I have, however, observed the respiration at 50 and at 40, with a pulse of 104 and of 88, respectively, and without demonstrable pulmonary lesion in either case. Respiration becomes much more rapid in case of pulmonary complications, not rarely reaching 50, or even 60. As the development of such complications is often insidious, cautious explorations of the chest should be made daily. Weak respiratory murmur, with fine expansion crepitus on deep breathing heard over the lower lobe behind, may often be found as evidence merely of hypostatic congestion and imperfect expansion. But even when pneumonia exists the percussion-dulness may be only relative and bronchial respiration be imperfectly developed. Cough may lack force, and the muco-sanguinolent expectoration be scanty and raised with difficulty. The increased lividity and cyanosis, elevated temperature, and cardiac failure confirm the suspicion aroused by the physical signs.

Bronchitis in any serious degree is greatly less frequent than in typhoid. Sonorous and sibilant râles scattered over the chest are common, and indicate congestion and slight catarrh. The character of respiration varies greatly.

If there be much pulmonary congestion, it is shallow and superior costal in type. If the cerebral symptoms and the toxæmia are profound, it is irregular, jerking, or even stertorous. Under these circumstances its frequency may fall below the normal. An inverted type of respiration, due to pneumogastric paresis, is a fatal symptom. The expired air is heavy and offensive. Little is known of its composition save that it often contains an excess of ammonia.

Hiccough is not rare in grave cases. It is important to be aware that epistaxis is of quite frequent occurrence in some epidemics. It was noted in twelve out of one series of thirty cases under my observation.

The urine is scanty and highly febrile. It is highly colored, of strong, offensive odor, and apt to become ammoniacal. The urea and uric acid are increased, while the chlorides are greatly diminished or absent. Albumin is usually present in all but mild cases. The amount is not great, nor does it it add materially to the gravity of the case unless infectious nephritis be present, when albumin is more abundant, with granular or epithelial tube-casts. The proportion of cases in which nephritis occurs varies much in different epidemics; its existence adds decidedly to the gravity of the case. If the patient survives the fever, the nephritis rarely persists. At the time of the crisis copious discharges of urine, at first loaded with urates and then very light colored and of low specific gravity, sometimes occur. As already stated, retention of urine is of frequent occurrence. The urine may be passed if the patient's attention is drawn to it; but the region of the bladder must be examined regularly, and the catheter be used if required.

The cutaneous symptoms demand careful study. The skin imparts a peculiar sense of pungent heat, aptly styled calor mordax. It exhales an odor which, combined with that of the breath, may be recognized as characteristic after a few experiences. There is but little tendency to moisture, so that sudamina are of rare occurrence in typhus as contrasted with typhoid fever. A copious sweat may attend the critical fall in the temperature. Cold, clammy sweats of the head and extremities, with continued high central temperature, often presage fatal cardiac failure. Herpetic eruptions are of rare occurrence.

The eruption of typhus consists of a combination of subcuticular mottling with the characteristic macules. The mottling is not essential or constant, though of common occurrence. It may appear as early as the first day. The spots or macules appear, as a rule, on the third or fourth day; they may be postponed for several days later, as to the seventh or even tenth day. They come out in a single crop, appearing first on the trunk, then on the extremities, and less constantly or copiously on the face. The spots are irregularly rounded in form, barely if at all elevated above the surface, and vary in size from that of the point of a pin to two or three lines in diameter. Both the number and the color of the spots bear relation to the severity of the case. As a rule, the more copious and darker-colored the eruption the deeper the infection. In mild cases the spots may be pinkish or rose-colored and disappear readily on pressure, so as to resemble those of typhoid, and may continue thus until they fade. Usually they are of a

deeper and duller red, more resembling the eruption of measles, and though they disappear on pressure at first, there is in bad cases a gradual conversion of the macules into petechiæ. In some cases the spots fade rapidly in the course of three or four days, but when petechial they last throughout the case, fading away gradually toward the crisis or remaining visible after death. In addition to these small petechiæ, there may be ecchymotic patches at various points of the surface or on the conjunctivæ. A fine branny desquamation occasionally, but not generally, follows the subsidence of the eruption. It may affect the entire surface, even the hands and the soles of the feet desquamating.

Varieties.—For practical purposes it is enough to recognize the mild, the ordinary, the grave, and the malignant varieties of typhus. In some epidemics a considerable proportion of mild cases occur, and more rarely walking and also abortive cases, corresponding to those respective forms of typhoid, are met with.

The grave form embraces cases with serious complications, such as pneumonia or nephritis, and also those cases whose gravity is due to the undue prominence of certain groups of symptoms. Under the latter heading must be classed the ataxic type, with a high degree of irregular nervous disturbance, and the adynamic type, with extreme prostration and tendency to heart failure. Murchison describes an ataxo-adynamic type which combines the features of both. The cerebro-spinal variety, of special interest owing to its resemblance to cerebro-spinal fever, is a form of the ataxic type.

Malignant typhus, also called typhus siderans, may prove fatal in twelve to twenty-four hours. The virus acts with concentrated intensity; immediate dissolution of the blood occurs; petechiæ may appear in large numbers, and large ecchymotic patches form on dependent portions of the body. Hyperpyrexia is developed in a few hours; the extremities may be cold and livid, while the rectal temperature is 106° or 107°; the pulse becomes running and thready, and delirium and stupor appear early. Death may ensue so speedily as to leave the diagnosis in doubt unless the evidence of infection is clear.

Complications and Sequelæ.—Epidemics of typhus vary greatly as to the frequency with which complications occur. It is an error to think them rare, and as they often develop insidiously, there is great danger of overlooking serious complications unless continual watchfulness be exercised.

Hypostatic congestion of the lungs, bronchitis, and pneumonia are of frequent occurrence. The pneumonia may be either croupous or catarrhal, is apt to run an irregular course, adds greatly to the danger of the case, and occasionally terminates in gangrene or runs into a chronic form.

Several loose dark stools occur daily in a considerable proportion of cases in some epidemics, but severe diarrhœa is rare. Intestinal hæmorrhage does not occur, save in the grave hæmorrhagic type.

A small amount of albumin is present in the urine of most cases of typhus fever, but does not necessarily imply the existence of nephritis. Careful microscopical examination will, moreover, show tube-casts and renal epithelium in

a considerable proportion of severe cases. They may be found even in urine which is amber-colored with but very slight deposit. I am not prepared to say that a slight degree of catarrhal nephritis adds alarmingly to the danger of the case; certainly most of my own cases where it was present recovered. It is always a source of anxiety, however, and if the urine be scanty and the albumin abundant uræmic symptoms are often added to the existing nervous disturbances and the danger is greatly enhanced. If the patient survives the fever, chronic nephritis rarely persists as a sequel unless the patient is allowed to expose or exert himself at too early a period of convalescence.

Epistaxis is occasionally met with, even when no pronounced hæmorrhagic tendency exists. Hæmatemesis is much more rare. In certain hæmorrhagic cases blood escapes from almost all surfaces, in addition to numerous subcutaneous ecchymoses.

Parotitis is both more frequent and more dangerous than in typhoid. Both glands may be affected simultaneously, though more commonly but one, or first one and then the other. Suppuration usually ensues, and the gland breaks down and is discharged in small necrosed fragments. I have seen death result from parotitis arising after all danger from the original attack of fever seemed over, extensive infiltration and burrowing having caused fatal exhaustion. If the pus be not evacuated promptly, it is apt to discharge by the ear, the cartilaginous meatus being separated from the bone. I have rarely known deafness to persist. There may be inflammatory swelling of other glands, analogous to the buboes of the plague.

Meningitic or other intracranial lesions are, as already stated, rare. Palsies of a single member, or even paraplegia, may occur among the sequels. Neuritis is in most instances the cause.

Jaundice, erysipelas, cancrum oris, and abscesses in the subcutaneous tissues or in the joints are occasional complications.

The muscular tissue of the heart is affected in typhus with granular degeneration whenever high fever is present, but in some cases this lesion is so extreme as to be the chief cause of fatal heart failure and collapse. Endo- and pericarditis are rare.

The hair falls out after typhus, though probably not so frequently as after typhoid. Permanent baldness is not to be feared. The nails present transverse ridges, as after other severe acute affections.

Prognosis.—The duration of typhus fever is, on an average, about two weeks. Short, abortive cases are occasionally met with, in which the crisis occurs as early as the eighth or tenth day. On the other hand, the fever may be prolonged to the eighteenth or twenty-first day, and if serious sequelæ have developed the sickness may be greatly protracted.

The mortality varies in different epidemics between 10 and 35 per cent. The type of the disease must be considered in estimating the prognosis, as well as the symptoms of the individual case. Children rarely die; young adults have many chances in their favor; beyond the age of thirty the prognosis grows more grave, and after middle life the mortality may reach 50 per

cent. Sex exerts no influence. The previous condition and habits of the patient, and especially as regards privation and intemperance, are of great importance: the disease is terribly fatal among drunkards. The negro race seems to succumb readily to typhus as well as to relapsing fever.

Intensity of the nervous symptoms; persistent hyperpyrexia; extreme prostration and rapid, feeble pulse, with threatened heart failure; scanty, highly albuminous urine; vomiting or diarrhœa; copious dark-colored eruption, soon becoming petechial; pulmonary complications,—these are most unfavorable elements in prognosis. Typhus is noted, however, for the almost miraculous recoveries which take place when patients seem moribund, so that our efforts must never be relaxed as long as a spark of life remains.

By far the larger proportion of deaths occur from the ninth to the twelfth day. In very grave or in malignant cases the system is overwhelmed by the toxæmia even as early as from the first to the fifth day. During the second week toxæmia, exhaustion, and heart failure are the common causes of death. When a fatal result occurs after the close of the second week, it is usually from some complication or sequel, especially pneumonia.

True relapses are excessively rare in typhus, and second attacks or recurrences, though not unknown, are likewise very rare. Patients who are convalescing from other infectious diseases seem highly susceptible to the poison of typhus, and the disease is very dangerous when contracted under such circumstances. In 1865, when attending a large number of cases of small-pox and of typhus, I saw several instances where each of these diseases developed in patients convalescing from the other. In at least two instances I could not avoid the conclusion that I had been the medium of communicating typhus to patients recovering from variola, although I did not myself contract typhus until the following year.

Diagnosis.—Sporadic cases of typhus or the early cases of an epidemic may be mistaken for cerebro-spinal fever, for typhoid, or for measles. This is partly because typhus is a much more rare disease than any of the others mentioned, so that the observer is off his guard. But even in the midst of a well-recognized epidemic occasional cases present themselves where the diagnosis is difficult.

In 1864 both typhus and cerebro-spinal fever were prevalent in Philadelphia. Errors of diagnosis were frequently made. The onset in both diseases is abrupt. Fever rises rapidly to a high point; delirium is early and may be violent. Headache, backache, and pains in the limbs occur, and hyperæsthesia is often present. In typhus, though not nearly so often as in meningitis, there may be painful rigidity of the muscles of the nucha with retraction of the head. The headache is, however, usually more intense and persistent in cerebro-spinal fever; muscular soreness and rigidity, and especially the retraction of the head, are more pronounced; intolerance of light and sound is present; vomiting is much more common; the evidences of prostration are less marked, and especially is the failure of heart-power later and less constant; herpetic eruptions are common, while the characteristic eruption of typhus is wholly wanting.

In ordinary cases of typhoid fever there is little or no danger of mistaking it for typhus. The mode of onset, the absence of chill, the gradual, step-like rise of temperature, the more gradual development of nervous symptoms, the bronchial and abdominal symptoms, the marked enlargement of the spleen, the occurrence of epistaxis, and the postponement of the eruption until the seventh day, serve to establish the diagnosis. But it must be remembered that in typhoid the onset is sometimes abrupt and the rise of temperature to a high point early and rapid; that intense headache, pains in the limbs, and hyperæsthesia may be present; the delirium be early and active, with marked tendency to stupor; the abdominal symptoms be absent; and the eruption be more or less uniformly converted into petechiæ and accompanied with subcuticular mottling. On the other hand, in typhus the symptoms may be mild, the eruption postponed till the sixth day, and then be scanty, light-colored, and disappear wholly on pressure; the bowels disposed to be loose and the symptoms of prostration be largely wanting. In short, there are few outbreaks of typhus in which some cases are not met which demand cautious and critical study before the diagnosis can be established.

The eruption of typhus comes out at about the same time as does that of measles, and at first may resemble it considerably. But in measles the eruptive stage is preceded and attended by marked catarrhal symptoms; the rash comes out first on the face; the spots form groups with crescentic borders, and rarely become petechial.

The bubo plague is so strictly limited to certain Oriental countries by modern quarantine that the question of its differential diagnosis from typhus can rarely arise. The symptoms of the onset are not dissimilar, but the very rapid development in the plague of profound typhoid symptoms; the early appearance of buboes, carbuncles, and extensive petechiæ; the pronounced tendency to collapse, with sudden fall of temperature; the absence of the characteristic eruption,—serve to distinguish this frightfully fatal disease from typhus.

It is unnecessary to repeat here, with reference to the possibility of mistaking typhus for uræmia, what has been elsewhere said on this point in regard to typhoid.

Treatment.—The highly contagious nature of typhus fever renders imperative the prompt isolation of each case. The infected house should be vacated and thoroughly cleansed and disinfected. If a case has occurred in a large community, the public health authorities are to be summoned to take charge of the locality, with a view to the adoption of such radical sanitary measures as may prevent any spread of the disease. Patients suffering with typhus fever should not be admitted to general hospitals if it can be avoided, but should be accommodated in special hospitals for infectious diseases. If the climatic conditions are favorable and the cases are numerous, they are best treated in isolated tents.

The hygiene of the sick-room as regards nursing, rigid cleanliness, ventilation, disinfection of all clothing, demands specially close attention. Although

there are no grave lesions of the alimentary tract, and though there may be some maintenance of appetite, it is on the whole safer that the diet should be liquid throughout the course of the disease. Milk is the best basis, and to it may be added strong animal broths. Junket, thin arrowroot, light custard, and raw egg may be cautiously tried, and continued if found to agree. Tea or coffee, either black or with hot milk, may be taken with relish, and may be very useful, especially where there is a tendency to ataxic symptoms or to stupor. Nourishment should be given in comparatively small quantities at short intervals: four to six ounces of milk or its equivalent every two hours may be taken as a fair standard. Water should be offered frequently, and the patient may be encouraged to drink it freely. Alcohol is indicated in nearly all cases by the prostration and the tendency to heart failure. It is specially well borne in childhood and in advanced life. Cases of moderate severity in vigorous young adults often do well without it. The same rules are to be observed as to administration and as to deciding for or against its beneficial action as were laid down in the article on Typhoid Fever. Upon the whole, it is needed in typhus earlier, more constantly, and more freely than in typhoid. It is usually well to begin with small amounts much diluted, but the remedy must be unhesitatingly pushed if the symptoms call for it. I find among my notes the records of two cases where one and a half ounces of strong brandy were given every hour, day and night, for ninety-two and ninety-six hours respectively, with the manifest effect of saving life.

The presence of marked ataxic or adynamic nervous symptoms, a copious and dark eruption with abundant petechiæ, a small, weak, and rapid pulse with failing cardiac impulse and first sound, are the positive indications for stimulation: the effect of the stimulus upon the symptoms is the guide as to the proper amount to administer, and the fact that the disease runs a short, self-limited course justifies the freest use of stimuli to carry the patient along till the critical hour is reached.

The management of the fever should be upon the same general lines as in typhoid. The use of cold baths, systematically employed after the Brand method, should be insisted upon in all cases where the fever rises to 103° in the axilla or 103½° in the rectum. While the temperature remains below that point dependence may be placed upon repeated sponging with cold water or an occasional pack. Should the surroundings of the case render bathing impracticable, it will of course be necessary to rely on repeated, thorough cold-water packs or affusion as originally used by Currie. It will, however, soon be possible to secure portable bath-tubs by means of which hydrotherapy can be carried out in private houses of every class. It is of the utmost importance that the temperature should be controlled from the very first day. If this be judiciously and firmly done, the development of the gravest nervous symptoms and of alarming heart failure may often be averted. The most frequent cause of failure of hydrotherapy is its postponement until heart, brain, and blood have been too seriously damaged by the continuance of high temperature.

Antipyrine and analogous antipyretics must be used with extreme caution. A sudden rise of temperature may be met and modified by one or two medium doses, but in typhus, even more than in typhoid, there is a tendency to continuous high temperature, and anything like the continuous use of these antipyretics is absolutely forbidden.

The mineral acids, especially nitro-muriatic and phosphoric, may be used freely with advantage. There is no good ground for believing that they exert any specific effect on the virus of the disease, but their general and local action is tonic. Besides, when properly diluted they make a pleasant acidulated drink, so that the patient is encouraged to take water freely. Dilute chlorine-water may be used in the same way.

Headache may often be relieved by applications of cold to the head, but if intense and persistent it may require the use of sedatives. Small doses of morphine and atropine may be given safely by hypodermic injection, or opium may be used by the rectum or by the mouth.

When active delirium is present, with sleeplessness and severe headache, an opiate combined with cannabis Indica or with hyoscine hydrobromate is to be tried; under these circumstances Graves advised tartar emetic in conjunction with opium. Chloral hydrate, in doses of 12 to 15 grains by the mouth or of 20 grains by enema, has given good results. If insomnia, with or without headache, is associated with marked prostration and ataxia, remedies such as camphor, valerian, or asafœtida are of use, and may have a small amount of opium associated with them. Full doses of quinine and asafœtida, 10 grains each, in the form of suppository, given morning and evening, exert a supporting and quieting effect.

If irritability of the stomach and vomiting are present, they must be relieved by simple sedative remedies and restriction of food, with substitution, if necessary, of nutritious enemata. Constipation may call for the use of glycerin suppositories or of simple enemata, or for the internal administration of fractional doses of calomel or mild saline aperients. A careful watch must be maintained against complications.

Pulmonary congestion or catarrh, if moderate in degree, may be relieved by dry cups or by counter-irritation applied to the back of the chest. If more severe, or if pneumonia has developed, increased stimulation, ammonium carbonate, and strychnine should be directed. I have used turpentine with much apparent advantage when the typhoid state became fully developed, with great nervous prostration, feeble circulation, and marked pulmonary congestion. Strychnine should be used in the same manner and to meet the same indications as in typhoid fever. More reliance is to be placed on it and alcohol than on digitalis in the treatment of threatened heart failure.

Convalescence is retarded by few sequels, and relapses do not occur. Care should, however, be observed both as to diet and exercise. The amount of stimulant should be reduced as rapidly as possible, and a bitter tonic with iron may be substituted with advantage.

RELAPSING FEVER.

By WILLIAM PEPPER.

Definition.—Relapsing fever is an acute infectious and contagious epidemic disease, characterized by its division into successive stages of exacerbation and intermission, by various uniform alterations in the viscera, and by the constant presence in the blood of a specific micro-organism—the spirillum of Obermeyer.

Synonyms.—It has many synonyms, the chief of which are—Rückfalls typhus, Febris recidiva vel recurrens, Fièvre à rechutes, Bilious typhoid fever, Hunger-pest, and Spirillum fever.

History.—While it is certainly a fact that the disease existed prior to that date, the first clear account of it was written in 1739. Since then numerous outbreaks have from time to time occurred in various parts of the world. Its first appearance in America of which we have any certain knowledge occurred in 1844, when it was imported by the passengers on an emigrant-ship. After this a few cases were observed in this country, and in 1869 an epidemic of the disease prevailed in Philadelphia. I had the opportunity, in conjunction with my colleague, the late Edward Rhoads, to study several hundred cases which were admitted to our wards at the Philadelphia Hospital. Since that time other epidemics have occurred, the last of any considerable size having been located in Russia during the years 1885 and 1886.

Etiology.—The etiology of relapsing fever is not as yet entirely clear, but for reasons that will be more fully stated below it is certain that the spirillum of Obermeyer plays an important, if not the chief, part. Aside from this immediate cause, we have numerous factors important in their influence upon the existence and spread of the disease.

Chief among the predisposing causes, although not essential, is the presence of the combination of filth and starvation. The former of these is usually associated with overcrowding, itself a powerful predisposing cause aside from its importance in effecting the spread of the disease and widening the limits of the affected area; while all three factors—filth, famine, and overcrowding—make a combination pre-eminently calculated to reduce the vital force of both individual and community, thereby offering favorable conditions for the onset and spread of this as of any other general disease. That filth, overcrowding, and starvation are not necessary factors is shown by the fact that those in the entirely opposite condition may be, and often are, attacked.

A still more essential and ever-necessary factor is contagion. This may take place either by direct contact of the well with the sick, or the contagious

principle may be carried by fomites, as is well evidenced by the frequent occurrence of the disease among laundresses. The infecting material may be transported from the ill without the bearer contracting the disease, although communicating it to others.

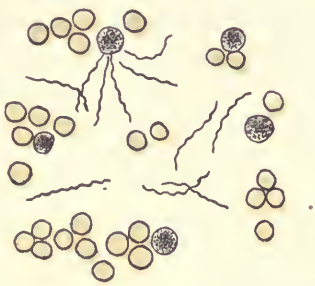
Neither age nor sex has any manifest bearing upon the etiology of this disease, although, as would be expected from the greater exposure to infection, the male sex and the active or middle period of life furnish the greater number of cases.

Race would seem to have no predisposing or protective influence, save only in so far as the hygienic surroundings and physical condition of different nations may alter the relative resistance to contagion. The negro shows, possibly, somewhat greater susceptibility to the poison than do other races. This liability is not strongly marked—not more so than we would expect when we remember the susceptibility of this class to various other diseases of a somewhat similar character.

Season has no evident etiological relation to the onset of the disease, nor do climatic conditions favor or limit its power, save for the wide variation in habits of life and surroundings among the dwellers in different climates.

By far the most important etiological factor is, however, the minute spiral organism discovered by Obermeyer in 1873—the spirillum Obermeieri. This has been found so constantly in the blood of patients suffering from this disease that suspicion pointed strongly toward it as the cause before its successful cultivation on artificial media and inoculation into animals. The microorganisms are long, extremely delicate, fibre-like bodies of spiral shape, in length measuring about six or seven times the diameter of a red blood-cell. (See Fig. 11.) They move freely about in the field of the microscope, causing

Fig. 11.



Recurrent Spirals in the Blood (after Jaksch).

disturbance of the blood-cells. This spiral motion takes place in the direction of the length of the organism. Dried preparations of the blood may be readily stained by the ordinary aniline colors in order to show the parasite. Not only has it been found in the blood obtained directly, but the organism is also present in the menstrual blood, in that coughed up, and in that passed by the urethra; but has never been found in the urine, saliva, milk, sweat, or in lymph from vesicles.

From the researches of numerous observers it is proved beyond peradventure that during the febrile access the spirilla are very numerous in the blood, while during the apyretic stage, or after the subsidence of an attack wherein but one pyretic period occurs, the organisms are found to be either totally absent or present in but very inconsiderable number. The question of the habitat of the spirilla during the apyretic intervals is one of deep interest, and not yet fully determined, although the experiments of Metschnikoff upon apes, in which the disease had been produced by inoculation, would indicate that the organisms retired to the spleen with the subsidence of pyrexia. Sarnow and v. Jaksch found in the blood, examined just prior to an exacerbation, numerous highly refracting forms resembling diplococci. These immediately upon the beginning of the attack developed into short, thick rods, from which the spirilla were formed. These bodies may possibly be the spores of the parasite.

As mentioned above, the pathogenic organism has been injected into apes with the result of producing the disease in the animals so inoculated. Accidentally, by cuts at autopsies and other means, the disease has been contracted by man in a similar manner, except for the fact that in these cases not only the micro-organisms, but also other material, were brought into the body. It can, therefore, be asserted as proven that this disease is produced by a specific micro-organism constantly present in large numbers in the blood during the periods of pyrexia, disappearing from the blood with crisis, capable of being cultivated upon artificial media, and of producing the disease when inoculated in pure culture.

Morbid Anatomy.—The external surface of the body shows no characteristic changes, although jaundice is seen where that symptom was present during life. I noticed in the cases at the Philadelphia Hospital that where the fatal result came during the pyretic period the cadaver retained its heat for a remarkably long time. Emaciation is not marked or is wholly absent, depending somewhat upon the duration of the illness.

Upon section, the muscles are frequently found to be jaundiced, but only where icterus was one of the symptoms before death. The voluntary muscles are often flabby, and under the microscope are found to have undergone granular degeneration. Interfascicular hæmorrhages may be present.

The blood shows one other peculiarity besides the presence of the micro-organism: numbers of granular cells are seen among the proper blood-cells. These may possibly be accounted for by the stripping off of the endothelial cells of the intima of blood-vessels, as was observed most markedly by Puschkareff in his observations upon the pathology of the disease in Russia.

The pericardium may show no abnormal change, or we may find pericarditis or subpericardial hæmorrhagic extravasation.

The heart in fatal cases is usually found seriously affected, the muscular tissue being of a grayish color and softened, while under the microscope the muscle-fibres are found to have undergone a similar change in greater or less degree. As would be expected, this granular change is most marked in cases of long duration.

There are no valvular lesions save those due to a preceding illness. Beneath the endocardium there may often be found hæmorrhagic effusions.

Pleurisy is frequently present, usually in combination with pneumonia. Subpleural ecchymoses are common. In the upper air-passages there has been noted the presence of catarrhal inflammation, while in some cases a diphtheritic exudate has been found in the pharynx and larynx. Œdema of the glottis may be present. Lobar pneumonia is a frequent lesion found upon post-mortem examination of fatal cases of relapsing fever, and in the lungs, as in many other portions of the body, hæmorrhagic infarctions are by no means rare. In one case in my own experience an area of gangrene of the lung occurred as the termination of a complicating lobar pneumonia.

True metastatic purulent foci may be present. The bronchial glands show no special alterations save those due to any pulmonary condition that happens to be present.

The peritoneum may show signs of local inflammation (chiefly in the splenic region), or there may be a general peritonitis, as in cases of rupture of the spleen.

In neither the stomach nor the intestinal tract are there any characteristic lesions, although submucous ecchymoses and extravasations are frequently discovered. The special glandular apparatus of the gastric and intestinal mucous membrane shows no change, the solitary and agminated glands of the intestine presenting less swelling and congestion than is usually found in other infectious diseases. The abdominal lymphatic glands show no pronounced morbid changes.

The spleen is constantly and characteristically altered, with more definite and specific changes than any other organ exhibits. It is always large, usually adherent to the diaphragm, and almost always partly covered by fresh fibrinous exudation. The size of the spleen is quite variable, the limits in the series of cases observed by me being 10 and 44½ ounces. The capsule often presents a mottled appearance, or may actually have in its substance large purple ecchymotic areas. In a few cases rupture of the capsule has been found. The splenic pulp is usually more or less softened and swollen, and shows enlarged Malpighian bodies. The latter may vary somewhat in character with the stage of the disease at which death occurs. In the early stages they are enlarged and of a greenish-yellow color, giving to the cut surface very much the appearance of shad-roe. Later in the disease this enlargement still further increases, until, by coalescence or aggregation of neighboring corpuscles, large masses may be formed. Hæmorrhagic infarction of the spleen is very frequent, the infarcts being, as a rule, venous, and frequently breaking down into purulent, softened areas.

These enlarged Malpighian corpuscles are found, upon microscopical examination, to be composed of large numbers of small lymphoid cells which have undergone cloudy swelling, or, later in the disease, marked fatty degeneration. The splenic pulp is found to consist of large numbers of lymphoid cell-elements in a more or less pronounced state of granular degeneration, free red-blood corpuscles, and fibrous tissue.

The liver is, in the vast majority of cases, enlarged, frequently much congested, at times pale and mottled in appearance. Ecchymotic areas beneath the capsule and extending for a short distance into the liver-tissue proper are met with, as in the case of the spleen. The hepatic substance is usually softened, but may, on the other hand, be found to be more firm than normal. The parenchymatous cells of the liver are commonly in a state of cloudy swelling or of fatty infiltration. The capillaries of the organ are stuffed with blood during the febrile stage, but regain almost, if not quite, their normal size during the apyretic period. In the cases accompanied by jaundice the hepatic cells contain brownish granules. Various changes in the hepatic interstitial tissue are found, but these usually depend upon previously-existing disease, and are not constant. The smaller biliary canals within the liver are at times found to be entirely patulous, but their epithelial cells may present a swollen, granular appearance, with scarcely visible nuclei, or in the cases of most intense jaundice the lumen of the ducts may be entirely occluded. The larger bile-ducts present no changes of sufficient gravity to account for the icterus that is so often present in this disease.

The gall-bladder is, as a rule, found to be filled with dark bile, but that the latter is capable of passing through the ducts is shown by the presence of bile-pigment in the contents of the duodenum and in the fæces.

The pancreas and suprarenal capsules show no peculiar alteration.

The kidneys are usually found to be moderately enlarged, at times of a mottled appearance or with actual hæmorrhagic extravasations beneath the capsule. Hæmorrhagic infarcts are met with in a small proportion of cases, and at times puriform collections are found near the periphery. In a large proportion of cases true parenchymatous nephritis is found, but in how many this is merely an acute engrafting upon former chronic process it is difficult to determine. According to W. Puschkareff, the kidneys always show the appearance of a parenchymatous affection, and a not highly pronounced acute glomerulo-nephritis is also a constant accompaniment of relapsing fever. Bloody extravasation into Bowman's capsule has been noted by some authors.

As already incidentally mentioned, there has been found a peculiar swelling and stripping up of the endothelial cells of the blood-vessels in some organs, notably in the spleen. The cause of this process it is difficult to assign, but it may be that one of its results is the large number of infarcts found so widely distributed in some of the cases. The bone-marrow shows peculiar alterations, in that the lymphoid elements are markedly increased in number and degenerated, so that in the ends of the long bones there are at times found cavities with puriform contents.

Symptomatology.—The incubation period extends over from five to eight days, during which the individual may suffer from vague pains and slight malaise. At the end of the time mentioned there is an abrupt onset, with chill, and aching pain in the head, back, and limbs. The chill may be preceded by obstinate vomiting or vertigo. There is marked physical depres-

sion, with distress, and, possibly, tenderness in the epigastric region. The temperature rapidly rises after the occurrence of the chill, or even during its continuance, while the muscular pains continue to be very severe. The rise is usually very abrupt, reaching in some cases 105° or 106° F. within the first twenty-four hours. With the rise in temperature the pulse becomes very rapid, averaging about 110 beats per minute, and is full and bounding. While the attack of pyrexia is in progress the face is usually flushed, and in some cases may present a bronzed appearance, or icterus may be present and hide the appearances just noted. There is no eruption characteristic of the disease, but sudamina are very numerous, and in some epidemics purpuric spots have been noted. The conjunctivæ are usually, but not invariably, clear, except in cases presenting jaundice. The tongue is coated white over the dorsum, with clean red borders and triangular area at the tip.

During this stage the cephalalgia, which is usually either frontal or general, persists. Delirium, except in alcoholic subjects, is but rarely present, but extreme wakefulness is a very common and annoying symptom, and is overcome by drugs only with extreme difficulty. Convulsions are rare, but may occur in cases, even though not accompanied by albuminuria.

Besides the muscular pains above mentioned, there is great hyperæsthesia, with marked tenderness over the position of the nerve-trunks and endings. Another common symptom connected with the nervous system is a peculiar tingling of the extremities. Occasionally motor palsy is observed, but its occurrence is rare.

Thirst is usually intense, while there is extreme repugnance toward the taking of nourishment. Nausea and vomiting are prominent symptoms, the matters vomited at times containing blood. The state of the bowels is very variable, although some constipation is usually present. Tympanites is not a marked or by any means constant symptom. Abdominal pain is one of the most prominent subjective symptoms, and is usually situated in the epigastric and splenic regions. There is also, as a rule, decided tenderness on pressure. The areas of hepatic and splenic dulness are invariably increased during this pyrexial stage, the latter being more markedly enlarged than the former.

There is usually some annoying cough, and epistaxis may be quite obstinate. Examination of the chest during this stage may be negative, but there are usually present the signs of acute bronchitis or even of pulmonary congestion, with some impairment of resonance at the bases. There is frequently to be heard a hæmic murmur over the cardiac region, but no other murmurs are developed as a result of the disease.

The urine is concentrated and high-colored, bile-stained in the cases with icterus, and may contain blood. As before stated, where hæmaturia is present the spirilla may be found in the urine.

The condition above described persists, the temperature varying but little from day to day, until the crisis, which usually occurs in about six or seven days. Just preceding this event there is frequently a marked rise in the body-temperature to a point even higher than that previously attained. The crisis

is signalized by a rapid fall in the temperature, a less-marked fall in the pulse-rate, a cessation of many of the most distressing symptoms, and, as a rule, the occurrence of some critical discharge—a profuse outpouring of sweat, a free flow of urine, a copious stool or a series of bowel movements, epistaxis, or, more rarely, metrorrhagia. During the occurrence of the crisis the face becomes pale unless icterus mask all pallor. The crisis may extend over as much as several hours, the temperature in that time falling 6°, 8°, or even 14° F.

The patient then enters upon a period of apyrexia, the intermission. During this stage most of the more distressing symptoms are absent: the temperature remains subnormal for a day or two before regaining the level of health; the pulse-rate diminishes, but not to an extent commensurate with the fall in temperature; the pulse loses its bounding character, but becomes easily excited; the cephalalgia becomes less intense, although the muscular pain and soreness continue to be severe. During this time, it is to be remembered, but few or no spirilla are to be found in the blood.

The disease in some cases ceases after one attack, the patient's condition merging from that of the post-critical period into that of convalescence; but usually after an apyretic interval of six or seven days (the extreme limits being two and twenty) a relapse occurs resembling in its onset the first attack described above. The relapse differs from the primary pyretic period in but few particulars. The patient's general condition is not so favorable, owing to the fact that the attack occurs in a system already weakened by fever; but, fortunately, the second attack is not, as a rule, accompanied by such high fever and such intense cephalalgia, nor is it of such long duration as was that with which the illness began.

The first relapse (second pyrexial period) continues, on an average for from three to four days, the extremes being a few hours and seven days. With the beginning of this second pyrexial attack the spirilla reappear in the blood, to again disappear with the second crisis. In the great majority of cases the morbid process terminates after the first relapse, but two, three, four, or even so many as eight, relapses may occur. The duration of the disease may thus extend to eighteen or twenty days, all told, where a single relapse has occurred, up to ninety or even more days in cases with multiple relapses.

During an attack such as has been described certain other symptoms and conditions, more or less deserving of the name of complications, may occur. These demand a more detailed examination.

Delirium, that at times, though infrequently, occurs, may be of different kinds. Although the temperature may remain at a great height, the mental condition is much clearer than is usually observed in cases of either typhus or typhoid fever, in which the thermometer indicates so high a degree. There may, however, appear in alcoholic subjects a delirium that is active and almost maniacal. On the other hand, there may be present a low, muttering delirium in the cases that assume the so-called typhoid character.

Sometimes in the first intermission—or, more rarely, at other times—there

occurs a sudden rise of temperature without any appreciable cause. This may in some cases be due to the influence of embolism of some important organ. During the period succeeding crisis, when the temperature should maintain a normal or even subnormal course, we may have a continuance of febrile movement. This is usually due not to the continuance of the influence of the specific poison, but to the continuing irritation of some organ or tissue secondarily involved. The local peritonitis in the splenic region may well be sufficient to maintain a considerable elevation of temperature.

Complications and Sequelæ.—Of complications, lobar pneumonia stands well to the front as being the most frequent cause of death. In the St. Petersburg epidemic of 1885–86, Puschkareff found this lesion present in 18 out of 47 cases examined. While, however, this complication is one of the most frequent immediate causes of death, its presence does not necessitate a fatal prognosis. With hepatization of the lung-tissue there is usually associated plastic pleurisy, and at times pericarditis. Gangrene of the lung may terminate the course of a complicating lobar pneumonia. So numerous are the examples of pulmonary congestion that that condition scarce merits the name of a complication, as it seems to be a part of the *ensemble* of a severe case of relapsing fever, just as it is in typhoid fever and other diseases of asthenic type.

In some epidemics grave catarrhal laryngitis has been a frequent complication, while cases with a diphtheritic deposit in the upper air-passages have been recorded. Epistaxis may be sufficiently severe to require plugging of the nares, and may vastly increase the anæmia so prone to occur in the ordinary course of the disease.

Pericarditis is not a frequent complication, but is met with occasionally, being usually an accompaniment of lobar pneumonia.

Cardiac thrombosis is frequently the immediate cause of death, being due, in part at least, to the extreme weakness of the degenerated heart-muscles. Sudden cardiac failure is quite often seen, cases dying after some apparently trivial exertion necessitated by change of posture. While cardiac thrombosis is frequently seen, the same process in the veins is observed much less frequently in this disease than in typhoid fever.

Brief allusion has been made to the tendency to the occurrence of embolism in various organs. Whatever may be the cause of this liability, its frequency is remarkable. Almost all of the chief organs of the body may be affected, giving rise to the symptoms peculiar to that condition when occurring in other morbid conditions. Superficial gangrene, probably a result of embolism, has been seen in the extremities and affecting the tip of the nose and ears.

The digestive tract is not especially prone to offer a field for complications in this disease. Suppurative parotitis is a condition that may occur, as in one of the cases occurring in the epidemic observed by the author. It occurs in a varying number of cases in different epidemics. Hiccough is a frequent and unfavorable symptom, being not only productive of much discomfort, but also exhausting strength and preventing natural rest. Hæmatemesis is not a very

rare complication, and is of very unfavorable import, three out of four cases in which it occurred in our series of cases being fatal.

Although diarrhœa is not so frequent as in typhoid fever, it occurs in a considerable proportion of cases, and may be sufficiently profuse to bring about a fatal result. Melæna may occur to a varying extent, and dysentery was, as might be expected, a notable complication in some of the epidemics occurring in India. General peritonitis is rarely present save as a result of splenic rupture: when present a fatal result may be predicted with certainty.

Splenic abscess occurs with sufficient frequency to cause us to be on our guard lest it may be the lesion present in those cases where the temperature of what would naturally be the period of apyrexia remains above normal. Rupture of a splenic abscess may be the cause of a generalized purulent peritonitis. The occurrence and significance of perisplenitis has already been mentioned.

The urinary system is the seat of varying morbid conditions, some of which are of great importance in determining the result. Albuminuria is present in a very large number of cases, and is not necessarily a cause of very serious alarm. When, however, the excretion of albumin is accompanied by the presence of tube-casts, the prognosis is very grave. The affection of the kidneys may vary from simple congestion to the lighting up of an old chronic process or the production of an actual acute nephritis, which may be hæmorrhagic in character. Complete suppression of urine is at times present. Hæmaturia may be profuse and exhausting: it is a grave complication, and is often followed by a fatal issue. Glycosuria has been observed during the course of some cases.

Profuse hæmorrhage from the uterus may occur, and it is recorded that in one case observed by Wolberg the menstrual accession seemed to be brought on by the general disease. Abortion usually happens when the disease attacks pregnant females.

Purulent otitis media or purulent rhinitis may present itself during some part of the course of the disease.

Various local palsies occur with peculiar frequency during or after attacks of relapsing fever. The lower extremities, shoulders, arms, or forearms may be affected. Precisely what condition is the underlying cause of these palsies it is sometimes difficult to determine; but in most cases, and more certainly in those with coincident anæsthesia, a perineuritis may be assumed as the pathological lesion.

An extremely frequent complication is collapse. This may be due to cardiac weakness from degeneration of the heart-muscle, to cardiac thrombosis, to rupture of the spleen, or to internal or external hæmorrhage. All of these conditions have been more particularly mentioned above.

Following an attack we may have a variety of more or less important pathological conditions. A frequent sequel is intense and persistent cephalalgia, or severe rheumatoid pains with or without swelling of the joints may persist. In some cases imbecility has been known to follow upon an attack

of this disease. Intense anæmia is by no means a rare sequel, while diabetes mellitus and acute miliary tuberculosis are among the rarer results.

A frequent sequel is a peculiar ophthalmia that is subdued with difficulty and is of long duration. This is most frequently seen in individuals whose nutrition was impaired before their attack of relapsing fever. Optic neuritis and atrophy are among the rarer sequelæ.

Diagnosis.—It would seem at first sight that the existence of the specific spirillum in the blood would be sufficient to prevent all chance of confounding this with any other disease. This would be true were it possible or customary to examine the blood of every patient, and were it always an easy matter to discover this organism when such an examination was made. In the earlier cases of an epidemic that is so rare a visitant to any one locality as is the one now under consideration it is not probable that a correct diagnosis will be made until either a case has been observed that has gone through a relapse or a clear case of contagion has been remarked.

To enumerate again the prominent symptoms: a sudden onset with chill, preceded by few or no prodromes; enlargement of liver and spleen; a flushed face; rapid, bounding pulse; rapid rise of temperature without marked nervous disturbance; intense rheumatoid pains; cephalalgia and obstinate insomnia; tingling of the extremities; tenderness and pain in the epigastric and hypochondriac regions; nausea and vomiting; hæmorrhages from various surfaces; frequent jaundice; crisis, followed by a period of normal or subnormal temperature. These go to make up a picture too characteristic to be mistaken where we are induced to bear in mind the existence of this affection.

The diseases with which it is most apt to be confounded are typhus fever, typhoid fever, malaria, and rheumatic fever.

In typhus the onset, although quite abrupt, is usually much less so than in relapsing fever. The temperature rises less suddenly, but, instead of the insomnia, persistent headache, rheumatoid pains, and freedom from the cerebral symptoms of hyperpyrexia which mark relapsing fever, there appear delirium, deepening stupor, subsultus, and rapid loss of cardiac power. To these must be added the appearance of the characteristic eruption on the fourth day and the absence of the spirillum from the blood.

In typhoid fever we have gradual and progressive rise of temperature, with early epistaxis, diarrhœa, increasing muscular weakness, tendency to hebetude, tympany, local tenderness in the right iliac fossa, and upon the seventh or eighth day the characteristic eruption.

To distinguish relapsing fever from malarial poisoning is less difficult if we bear in mind the place of residence or business, and note the presence or absence of periodicity, the presence in the blood of peculiar organisms in each disease, and, finally, the ready control of the malarial manifestations by quinine. From yellow fever the history of the case as to residence would be, as a rule, sufficient to prevent error.

In rheumatic fever without arthritis we may have rapid rise of temperature, with tendency to hyperpyrexia, and severe diffuse pains closely like those

noted in relapsing fever; but the acid sweats, the frequent cardiac complications, the absence of marked enlargement of the spleen and liver, of jaundice, and of the spirillum in the blood will establish the diagnosis. In no disease is hyperpyrexia more surely attended with grave cerebral symptoms than in rheumatism; and this and the absence of any critical fall in the temperature are further points of distinction.

Prognosis.—Although this disease presents such alarming symptoms, and in spite of the large number of complications that may occur, the prognosis is usually favorable. The sudden and extreme elevation of temperature, with the intense muscular pains, furnishes a picture that would seem to point to a disease of much greater mortality than the one under consideration. The actual mortality varies much in different epidemics, being chiefly governed by the proportion of the bilious typhoid form as compared with the ordinary and uncomplicated variety. The death-rate varies from 2 or 3 to even 50 per cent.

The more unfavorable symptoms are—prolonged pyrexia after the pyretic period should have been completed; pneumonia or intense pulmonary congestion; active maniacal or muttering delirium; the typhoid state; convulsions, with or without albuminuria, nephritis, and hæmaturia.

Treatment.—The special points for consideration in regard to the treatment of this disease are the prevention of contagion, the reduction of hyperpyrexia, the combating of the pain, insomnia, asthenia, and various complications, and the prevention of the relapse.

In regard to prophylaxis but little need be said, as, aside from actual contact of the person with one ill of the disease or with his emanations, the predisposing causes are such as are decidedly unhygienic from a general as well as from a special point of view. The fact that the disease may be carried by fomites should, however, be constantly borne in mind, and all our efforts should be directed toward lessening the risk so produced to as great an extent as in our power.

The patient should be isolated, and all clothing, whether of body or bed, should be promptly burnt or plunged into boiling water or strong bichloride-of-mercury solution immediately after having been discarded, and before it has been handled by more people than are actually necessary. During the time of an epidemic such hotbeds of contagion as are plentiful in the slums of all large cities should be dealt with as radically and thoroughly as possible.

Absolute confinement in bed and avoidance of exertion are essential elements of treatment, and should be insisted on not only during the febrile stage, but during the intermission as well. The diet must consist of liquids, such as milk, koumyss, pancreatized milk, broths, etc.

It must be admitted that we have no specific remedy for this disease, and that it is at present beyond our power either to check the growth of, or to destroy, the specific parasite that is its apparent cause. Not only has quinine no specific influence in controlling the manifestations of this disease, such as it exerts over the periodic symptoms of malaria, but it fails even to markedly

affect the pyrexia, while in some cases it adds to the discomfort in the head and may also excite or increase gastric disturbance. It is therefore to be used carefully if at all, and never in the large doses that have been tried in times past.

The other great antiperiodic drug, arsenic, seems to have no more controlling influence than has quinine. The use of oil of eucalyptus or eucalyptol has not, to my knowledge, been tried. All efforts to avert or postpone the occurrence of the relapses have hitherto proved ineffectual.

The most important indication of reduction of the high temperature should undoubtedly be met promptly by hydrotherapy. No adequate reports are yet at hand to show the effect of the systematic use of cold baths in relieving the symptoms and modifying the cause of this disease. But in spite of the remarkable tolerance of the high temperature, there is every reason to hope that its prompt reduction may prove the most satisfactory and effective method of treatment in this curious affection.

Antipyrine, antifebrin, and thallin were used in the late Russian epidemic, but, according to the reported observations, were without benefit, and even produced such great prostration or nausea and vomiting that their use had to be discontinued.

For the rheumatoid pains and insomnia morphine or other opiate will give the greatest relief, while its sedative action upon the gastro-intestinal tract is a further advantage. Hypodermic injections of morphine and atropine, combined with the free use externally of anodyne liniments, will be found of great value. Salicin, salicylic acid, and salicylate of sodium have been fully tried; but have not been found to exert any good influence upon the rheumatoid pains.

In a disease such as this, where asthenia is much to be dreaded, all depressing remedies must be carefully avoided. For this reason, and also because of the tendency to gastric disturbance, chloral and the bromides must be tried with great caution. In the above-mentioned epidemic in Philadelphia but little or no benefit was obtained from these drugs as nervous sedatives and hypnotics.

Aside from care in diet, the gastric irritability may require the use of some more direct medication, as by small doses of calomel, subnitrate of bismuth, or nitrate of silver.

Stimulants in the form of whiskey or brandy are required in almost every case, and should be freely given in accordance with the amount of prostration. Ammonia, digitalis, compound spirit of ether, or strychnine may be used as adjuvants to the alcoholic stimulants.

As the jaundice is in large part of hæmic origin, no special treatment can be advised, but the condition of the stomach and duodenum must be considered and carefully treated. The hiccough may be either relieved or checked by the administration of chloroform, this drug also controlling the peculiar periodical chills that occur in some cases. In obstinate cases hypodermic injections of morphine and atropine into the tissues around the base of the chest may be tried.

Complications must be met as they arise, but during the whole course of treatment the need for support of the vital forces must ever be kept in mind.

CEREBRO-SPINAL FEVER.

By WILLIAM PEPPER.

Definition.—Cerebro-spinal fever is a specific, infectious, pandemic disease, slightly if at all contagious, probably microbic in origin, occurring sporadically or in epidemics, characterized anatomically by inflammation of the meninges of the brain and spinal cord, and clinically by irregular nervous symptoms pointing to profound disturbance of the cerebro-spinal functions, the most prominent of which are intense pain in the head and often in the trunk and extremities, hyperæsthesia, contraction of the muscles of the nucha and back, vomiting, irregular fever, delirium, and, in severe cases, coma.

Name and Synonyms.—No satisfactory title has yet been suggested for this disease. Upon the whole, cerebro-spinal fever seems preferable. It is open to the objection of implying that the fever is dependent upon the meningeal lesions, whereas it is an infectious disorder of the general system, and the meningitis is only one of its manifestations. For the same reason we refuse to accept the name "enteric fever" in place of typhoid fever. But, in the first place, although the intestinal lesions in typhoid fever are of great importance, they do not dominate the symptomatology of that disease nearly to the same extent as does the cerebro-spinal meningitis the symptoms and course of the disease we are now considering. Further, it cannot be said that any of the clinical conditions in cerebro-spinal fever suggest for it a descriptive name so characteristic as, for instance, typhoid is of the fever which is almost universally known by this term. Again, there are weighty objections against all other names suggested. The disease is so often sporadic that I fear the term "epidemic cerebro-spinal meningitis" has not rarely led to a failure to recognize the nature of isolated cases. "Infectious cerebro-spinal meningitis" is a name I have thought of proposing, and it may have some advantages, but it does not mention the acute febrile nature of the disease, and it must be remembered that there are other forms of acute infectious meningitis. Other names which have been more or less widely used, such as spotted fever, petechial fever, malignant purpuric fever, have become wholly obsolete. Pending the suggestion of a better name, it seems desirable to unite in the use of the title "cerebro-spinal fever," since the possession of a simple, clear, generally-accepted name certainly favors the clinical recognition of a disease and an appreciation of its nature.

History.—There seems to be no reason to believe that the disease was clearly recognized before the early part of the present century. Whether or not it

existed before cannot now be determined, although some authors claim that there is evidence of its having occurred even in ancient times. It seems difficult to doubt its occasional occurrence, as the specific cause has probably not come into existence of recent years only. Viesseux in 1805 appears to have been the first to give a clear description of an epidemic which occurred in Geneva, and in which 33 persons died. In the following year the disease made its appearance at Medfield, Mass. From this date up to 1816 local epidemics were observed in various countries of Europe and in several parts of the United States. It then disappeared entirely until 1822–23, when cases were reported from Vesoul, France, and from Middletown, Conn., and after this, up to 1837, from a few other localities. From 1837 the disease began to spread throughout France, and for years prevailed extensively there. Since that date, also, epidemics have appeared suddenly, and often simultaneously, in different parts of the world widely separated from each other, and where there has not been the slightest possibility of transportation. They lasted a variable time and were more or less widely spread. Sometimes the disease was for years unheard of in one country while prevailing in another. From 1850 to 1854 it was unheard of anywhere. Since 1860 epidemics have occurred in nearly every civilized country.

In the United States it has at times been very prevalent and very fatal. In 1864, 400 persons, out of a population of 6000, died of it at Carbondale, Pa. It affected both the Union and Confederate armies during the Civil War, and was at times very malignant. Although 782 deaths from cerebro-spinal fever were reported in New York City in 1872, the disease appears on the whole to have been more limited there, both in extent and duration, than in Philadelphia, where it has been endemic since 1863, and at times severe. The tabular statement of the number of deaths in Philadelphia yearly from 1863 to 1883, published by Stillé, I have completed up to the year 1892:

Deaths in Philadelphia from Cerebro-spinal Meningitis from 1863 to 1891, inclusive.

1863 49	1873 246	1883 50
1864 384	1874 82	1884 124
1865 192	1875 83	1885 87
1866 92	1876 85	1886 75
1867 109	1877 56	1887 45
1868 55	1878 90	1888 50
1869 37	1879 62	1889 37
1870 36	1880 78	1890 25
1871 49	1881 90	1891 23
1872 133	1882 51	Total, 2575

I had the opportunity of studying the severe Philadelphia epidemic of 1863–65 under my father, the late Dr. William Pepper, and the late Dr. William Gerhard. I made a number of autopsies under their direction and verified the nature of the cases. Having been familiar thus early with the disease, I can confirm from subsequent experience the truth of Dr. Stillé's state-

ment, that it has lingered in this locality longer than has been reported of any other place in this country from which information has been obtained.

Etiology.—Of the predisposing causes, climate seems to have a decided influence, for, although cerebro-spinal fever has occurred in all portions of the temperate zone, it is unknown in the tropics. It is most prevalent in the northern regions of the temperate zone. Season, too, is an important factor, as the prevalence of the affection is much greater in cold weather. Not only do by far the greater number of epidemics occur in the winter-time, but those developing then are more severe and extended. The nature of the locality with regard to moisture, elevation, sea, mountain, city or country, is generally considered to be without predisposing influence. With regard to moisture, however, this opinion is not undisputed. Wollf, who carefully analyzed 132 cases which had been treated in the Hamburg hospital, came to the conclusion that moisture of the earth and air is a decidedly predisposing factor. Very few of his cases occurred during July and August, the dry months of the year. As in regard to all infectious diseases, it may be said that bad hygienic conditions, as exposure, overcrowding, excessive bodily or mental exertion, insufficient food, and the like, exercise a predisposing influence. But it appears that the effect of these conditions is much less marked as regards the occurrence of cerebro-spinal fever than of other diseases of this class. I must state, however, that in the majority of the cases I have seen there has been some marked defect in the sanitary condition of the dwelling or in the physical condition of the individual. In the other cases healthy subjects under admirable sanitary conditions were attacked violently. It has often been noticed that soldiers crowded in barracks and the occupants of tenement-houses suffer most severely. On the other hand, in some epidemics large cities, which apparently afforded the most favorable conditions for severe attacks, escaped entirely, and the disease has devastated cleanly villages or occurred in isolated outbreaks. Race is not a predisposing factor. Sex is also probably without influence, and that more males than females are attacked is doubtless due to the fact that the former are more exposed to privation, crowding, and other predisposing causes. Age is a very important factor. Statisticians agree that the disease is far more prevalent among children, and is also more fatal among them. J. L. Smith found, from the reports of the New York Board of Health, that infants under one year of age furnished the largest proportion of fatal cases. Different epidemics have, however, differed widely as to the relative proportion of adults and children attacked.

The question whether cerebro-spinal fever can be acquired by direct contact with or proximity to a patient suffering with this disease is an important one. It is, however, almost universally admitted that it is either not directly contagious at all or so to a very slight extent. Not only do the first cases of epidemics develop without there existing the slightest possibility of the disease having been acquired by contact with other cases, but the majority of cases occur singly in families, and where several cases do occur in a household it is never possible to trace any fixed period of incubation between them which

might indicate that they had acquired the disease the one from the other. Nurses and physicians in attendance are attacked with the greatest rarity. Nor does the evidence justify the opinion that it can be transmitted by the secretions. On the other hand, there is abundant proof of the existence of a specific poison which may attach to certain houses or localities so as to render them infectious. An impressive instance is recorded by Hirsch, in which a woman who had nursed a patient with cerebro-spinal fever returned to her home in another village, and there sickened and died. Mourners at the funeral came from another township, and three of these died from the disease soon after. Moreover, there are cases on record which indicate very strongly that the disease may be contracted by contact with infected garments, if not by direct contagion in rare cases. In one instance, reported by J. L. Smith, a mother was attacked by cerebro-spinal fever two days after washing the clothes worn by her son, who had died with it, and a few days later her infant also sickened and both died. One of the most remarkable published cases with which I am acquainted is that reported by Kohlmann. A servant-girl died with typical symptoms of cerebro-spinal fever. Her clothes were lent by her family to different neighbors. A man in one house, who had received a coat, was attacked by the disease some months later, and several weeks afterward his son was stricken down, and in one week more his daughter. A woman who had visited the last case for not more than ten minutes, and who had held the head of the patient while the throat was being examined by the physician, suffered from the disease in a mild form eight days later. Another coat was lent to a young boy in another house in a different part of the city. He took the disease and died, while his mother also died, probably of the same affection, although it began in her case as a croupous pneumonia.

Instances such as these, together with other peculiarities of the affection, warn us not to be dogmatic as to the possibilities of the transmission of the disease when the poison is virulent and the system unusually susceptible.

Recognizing, then, the existence of a specific virus as the true cause of this disease, it must be stated that its exact nature is as yet unknown. It is generally believed to be microbic. A micro-organism identical with or indistinguishable from the pneumococcus has repeatedly been found in the meningeal exudation. Many investigators claim that this is the only microbe which occurs in the meninges in this disease. It certainly is the one oftenest discovered, but in many cases other organisms as well have been described, so that it would seem possible that the disorder may be capable of being produced by different species of microbes. Foa and Uffreduzzi made some interesting studies of the pneumococcus found in the meninges of several cases, and observed that it retained its vitality both after exposure to cold and after drying.

The inference has been suggested that this disease may be caused by several different species of micro-organisms, of which the lance-shaped coccus, similar to or identical with the pneumococcus, is the most common; but I do not consider that the bacteriological studies of this question are sufficiently advanced to justify this opinion. Whatever the exact nature of the specific

micro-organism, assuming its existence, it evidently is of widely diffused occurrence, and probably has its growth favored by moderate temperature and by moisture; when scant in numbers or feeble in virulence it is inoperative or affects only susceptible systems, thus producing isolated cases of the disease; whereas when local and atmospheric conditions favor it acquires such concentrated intensity as to act uniformly and violently upon those who receive it into their systems. We have no actual knowledge of the mode in which the virus gains entrance to the economy.

Morbid Anatomy.—Emaciation is very great in the bodies of those who have been long sick. In rapid cases no such change is exhibited to any degree. Rigor mortis is marked. Decomposition often commences early. Ecchymoses usually are extensive. The remains of different eruptions, particularly of petechiæ and herpes, are often found on the skin. Abscesses are sometimes met with in the subcutaneous connective tissue. The muscles, especially those along the vertebral column, are dry and dark reddish-brown or sometimes pale. They often exhibit a waxy degeneration or an extensive fatty degeneration in the form of fine granules. Abscesses are at times seen in the intermuscular connective tissue. The articulations sometimes contain sero-pus.

The heart is often flabby, and the muscle-substance exhibits the same condition as do the voluntary muscles. The pericardium is sometimes inflamed and shows ecchymoses. Endocarditis is rare. The blood is usually of a dark color and fluid, or any clots present are dark and soft. Bubbles of gas are occasionally found in the blood of the heart and arteries. In cases where fever has not been high, and especially in the early stages of the disease, the blood may coagulate readily, and firm whitish clots may be found in the heart or vessels.

The lungs often exhibit congestion, œdema, bronchitis, atelectasis, or pneumonia. Ecchymoses or evidences of purulent inflammation are sometimes seen on the pleura.

The condition of the spleen is of special interest, as bearing on the true nature of the disease. Its size seems proportionate to the degree and duration of the pyrexia. It is rarely as much enlarged as is common in typhus, and in cases where the inflammatory lesions are marked the spleen may yet be small and merely congested.

The liver is congested; the stomach and intestines generally show no change except occasional congestion. Sometimes the lymphatic tissue of the intestine is hypertrophied and rarely ulcerated. The kidneys are usually congested, and may exhibit the lesions of nephritis. The mucous membrane of the bladder may be ecchymosed.

Naturally the most striking lesions in cerebro-spinal fever are those of the nervous system. The calvarium is very hyperæmic. In acute cases the sinuses, arteries, and veins of the brain are found engorged. The meninges are exceedingly hyperæmic, generally throughout. Death may occur before the lesions have advanced beyond this stage. The dura often shows scattered punctiform hæmorrhages, and its surface is dry, hyperæmic, and more or less adherent to

the arachnoid. The arachnoid itself is often normal, but in other cases it is dense and cloudy, especially along the vessels and in the depressions. Serum, or rarely pus, sometimes occupies the space between the arachnoid and the dura. In cases which have run a prolonged course a serous, fibrinous, or purulent exudate takes place within the meshes of the pia. This infiltration is generally widespread, but is especially well marked in the depressions. It may be limited to scattered patches or follow the course of the vessels. Pus is particularly liable to be found in the Sylvian fissure, at the optic chiasm, the anterior surface of the pons, and the surface of the cerebellum; but the convexity is also commonly involved, and I have seen the entire surface bathed with a thick layer of pus. The pia is often so adherent to the brain in places that it cannot be separated without tearing the latter. The brain-substance in section exhibits numerous *puncta vasculosa*. Areas of softening are sometimes seen, and occasionally the whole brain is softened. Abscess has been reported. Rarely the brain is œdematous. The choroid plexus is congested and infiltrated. The walls of the ventricles are softened. In long-standing cases the effusion into the ventricles of serous, turbid, or even purulent fluid may become very extensive, and flattening of the convolutions with atrophy of the brain-substance may result from the pressure. Sometimes chronic hydrocephalus ensues.

The condition of the spinal membranes is analogous to that of those of the brain. The dura is often dark and hyperæmic, and extravasated blood sometimes separates it from the vertebral canal. The arachnoid is often cloudy and infiltrated, and serum or pus may distend its cavity. The pia is hyperæmic, thickened, and adherent to the cord, and a serous, fibrinous, or purulent exudate occupies its meshes. This exudation may be almost universal, but is far more commonly situated chiefly at the posterior aspect of the cord. The cord itself is hyperæmic, infiltrated with serum, and sometimes softened. Frommüller reports a case in which the central canal was dilated and filled with pus.

The organs of special sense may exhibit lesions. Choroiditis with detachment of the retina has been reported. Ulceration of the cornea sometimes occurs. Purulent inflammation of the labyrinth and tympanic cavity has been observed.

As might be expected, both cranial and special nerves are often involved in the morbid process. The auditory and optic nerves are especially liable to suffer. The exudation extends along their lymph-sheaths, and the roots of the special nerves are often bathed in pus. Not only does this occur, but it is not infrequent for perineuritis or neuritis to spread along the nerve-trunks, leading to troublesome sequels or even grave lesions of nutrition in the parts to which they are distributed.

Symptomatology.—The symptoms of cerebro-spinal fever vary so greatly in different epidemics, and even in different cases in the same epidemic, that it is exceedingly difficult to present a general description of them. Numerous and elaborate classifications of the forms of the disease have been made, but to repeat them at length would only add to the confusion already existing. The

simpler the classification the better, and the following seems to me both natural and convenient for purposes of study:

1. *The Ordinary Form.*—The period of incubation is entirely unknown. Prodromata are not encountered with any regularity, and when present last a few hours to a few days, and consist of depression, loss of appetite, headache, vertigo, pain in the back, and other symptoms of an entirely indefinite nature. Ziemssen states that there is sometimes an interval of several hours, just before the onset of the disease, in which all prodromes disappear. In the great majority of cases prodromes are absent, and the disease is ushered in with great suddenness and severity, quite distinct from the beginning of an ordinary meningitis. The attack nearly always begins between noon and midnight. There is a chill, often violent, with intense headache, repeated vomiting, moderate fever, and generally a strong, full pulse. The face is usually pale and livid, and denotes great suffering. In children the attack sometimes begins with convulsions. In a recent sporadic case in a boy of thirteen years the attack began with intense pain in the epigastrium, with threatened collapse.

In a very short time, generally by the second day in the majority of cases, pain and stiffness of the muscles of the back of the neck develop, and constitute one of the most characteristic symptoms. The headache grows worse and there is sensitiveness to light and noise, and often irritability and restlessness. The pain and stiffness extend along the muscles of the spine, and even into the limbs as well, where the suffering may be very intense. In severe cases retraction of the head and orthotonos, or even opisthotonos, soon develop. As a result of the tonic spasm in the muscles of the extremities, the forearms are flexed upon the arms and the legs upon the thighs. There may be tremor or clonic spasm in the muscles of the arms, legs, eyelids, or face. Strabismus is frequent. The pupils are dilated, contracted, or unequal, or do not react well to light. General epileptiform convulsions with unconsciousness are sometimes observed, but more often in children than in adults. Local paralyses occasionally occur in various parts, as in the muscles of the face, of the eye, or in a single group of muscles of an extremity or of the trunk.

With these motor symptoms are seen also disturbances of sensation. The intense pain has already been mentioned. There may be great sensitiveness over the spine, and a remarkable hyperæsthesia of the entire surface of the body and of the joints. Vertigo sometimes persists, and is distressing even when the patient is lying quietly in bed. Ringing in the ears, with great sensitiveness to sound, is succeeded by deafness. Photophobia is almost constantly present, and there may be double vision and even temporary blindness. Delirium occurs very early, varying from a simple wandering to a true maniacal form, and often alternating with stupor.

The tongue is coated and often remains moist, though in severe cases it may become dry and brown. Vomiting usually subsides as the disease advances, but may persist and be distressing. Taste and appetite are lost. The bowels are usually constipated, and the abdomen may be decidedly retracted.

The amount of urine passed is variable, but is apt to be increased, and albumin is occasionally present. The spleen is often somewhat enlarged, as already stated.

Even during the first few days of the disease the skin is liable to exhibit eruptions. Herpes facialis is a very common form, and a petechial rash is quite frequent. Other eruptions likewise occur in some cases. The fever is irregular and presents no typical curve. It is generally moderate in degree, though occasionally it is high. The pulse is likewise variable; sometimes slow, and again very rapid. Respiration, too, varies, but is not often much accelerated. Cheyne-Stokes or sighing breathing is sometimes encountered.

The disease exhibits a variable course, but generally reaches its height between the third and the sixth day. It has been claimed by Frey and others that a remission in the symptoms sometimes occurs about the third day, although it lasts but a short time. I have on several occasions noted this in such marked degree as to rouse hope that error in diagnosis had been made, but the characteristic symptoms quickly resumed their course of development. The duration of the disease may be from a few hours to several months. If the case tends toward recovery, the symptoms become less marked after five or six days, the spasms grow less, the mind becomes clearer, and the depression, headache, and general pain ameliorate. Convalescence is fairly established in one or two weeks, although often not until after a much longer time, and it is very apt to be interfered with by complications and sequelæ.

If, on the contrary, the case is destined to end fatally, the symptoms of nervous excitement pass into those of exhaustion; delirium changes into a state of coma; prostration grows extreme, the pulse rapid, the temperature high, and there is paralysis of the sphincters with involuntary discharge of urine and fæces. Sometimes the course of fatal as of non-fatal cases is greatly prolonged, lasting weeks or even months.

2. *The Malignant Form.*—This form has also gone under the title of fulminant (*méningite foudroyante, meningitis siderans*), apoplectic, rapid, explosive, etc. It may occur sporadically in rare instances, and with variable frequency in all epidemics, but especially at their commencement. The patient, previously in perfect health, is stricken by the disease with the greatest suddenness, and rapidly passes into a condition of collapse. There is usually a violent chill, intense headache or drowsiness, great prostration, and a feeble pulse, which may be slow at first, but which soon grows rapid. There is little or no fever—the temperature may, indeed, be subnormal—and there may be coldness and clamminess of the skin, with cyanosis. Respiration is slow and labored. The urine is scanty and full of albumin. There is contraction of the muscles of the back of the neck, spasm in other muscles, or even general clonic convulsions. Delirium appears almost from the first, and rapidly passes into coma. A purpuric rash usually develops, and often quickly vesicates or sloughs.

These cases are almost invariably fatal, and generally so within a few hours. A case is reported by Gordon in which death occurred in five hours,

and in the Philadelphia epidemic of 1863 I saw cases which proved fatal in seven, ten, and fourteen hours, respectively. Rarely the fatal ending may not take place until the third day. The lightning-like suddenness of the onset and the malignancy of the symptoms surpass any description which can be given of them. If reaction is established and the case is prolonged, as happens in rare instances, it is only to exhibit a course of long duration, with great violence of symptoms, numerous dangerous complications, and ultimately with crippling sequels.

3. *The Mild Form.*—This form exhibits throughout symptoms of very little severity. Many cases scarcely seem to need confinement. There is little more than severe headache, with slight vertigo and nausea, while fever is absent or slight. Occasionally there are slight stiffness of the neck and vomiting. In a few days the patient is convalescent. The diagnosis may be difficult, except when the case occurs during epidemics of the disease. It is to be borne in mind that all the symptoms of such cases may suddenly become very severe.

4. *The Abortive Form.*—In this form the attack begins with severe symptoms, which last only two or three days and then suddenly ameliorate. It would appear that in these cases the initial constitutional infection is marked, but that the other essential constituent of the disease, the meningeal inflammation, is present in very light measure.

5. *The Intermittent Form.*—This is another well-recognized variety to which reference must be made. In it there occurs daily or every other day a decided exacerbation of fever, with great increase in the severity of all the symptoms, these exacerbations being separated by periods of almost complete subsidence of all the manifestations of the disease. There is, however, not the regularity in time which is seen in malarial fever, and the temperature curve resembles rather that of pyæmia. The intermissions appear either at the beginning of the attack or toward the close. Cases of this form are apt to be prolonged.

Of the remaining forms which have been described by writers may be mentioned the *nervous*, with numerous sub-varieties, as the ataxic, adynamic, cephalalgic, neuralgic, delirious, etc.; the *typhoid;* the *inflammatory;* the *chronic*. Nearly all of these, however, find their places naturally among the types already described, and consequently need no further mention in this connection. Yet it is necessary to be aware of the special features of the chronic form, as in my experience it has usually given rise to embarrassment and uncertainty as to the nature of the disease, as it runs its protracted course to a fatal issue or to gradual and usually partial recovery. Such cases are marked by continued gastric irritability from central nervous lesions; progressive extreme emaciation, until the patient is literally a living skeleton; various and erratic symptoms of perineuritis and of subacute meningitis, and irregular paroxysms of fever. I have watched the course of such symptoms for ten or twelve weeks, and cases are recorded of sixteen weeks' duration. Unquestionably, there is central disease persisting in the membranes or sub-

stance of the nervous centres. More commonly death closes the scene, but when recovery finally ensues the patient is left with damaged special senses, impaired mind, palsy or muscular atrophy, and persistent nerve-pains.

Description of Individual Symptoms.—Certain of the symptoms of cerebro-spinal fever demand a somewhat more extended consideration.

Chill is very common as an initial symptom, especially in adults. It may vary from simple chilliness to a rigor of the greatest severity, and it may be repeated several times on the first day. Prostration is an early and prominent symptom, and is conspicuous throughout the whole attack. A sense of faintness so often is present that the affection has been called *typhus syncopalis.* Emaciation, too, takes place early, and in severe cases is rapid and great. The emaciation may proceed so rapidly that it would appear to be due to some tropho-neurotic disturbance. Painful swellings of the joints, resembling those of rheumatism, but sometimes attended with purulent effusion, were first described by James Jackson, Jr., and have since been repeatedly observed. The facial expression is one of severe pain. The features are much distorted, or, when the pain is persistent, are fixed and rigid and the expression is dull. At the outset the face is often pale and sunken. There is not the sleepy expression of typhoid fever nor the dusky appearance of typhus.

Of the sensory symptoms, pain in the head is one of the earliest, most constant, and most distressing. It is of an agonizing nature, except in the mildest cases and in those of the malignant type. In the latter there seems to be no time for headache to develop. The headache is subject to remissions and exacerbations. Its situation is variable, as is its quality. The severity of the headache seems to have little bearing upon prognosis, although its cessation is a very favorable symptom. Even during profound unconsciousness the patient may raise his hands to press his head or may utter cries from time to time. Headache may persist to a greater or less extent long after recovery from other symptoms.

Pain in the neck and back, though likewise a very frequent symptom, is more variable than headache in duration, intensity, and extent. In some cases it only occurs when attempts are made to move the patient. The intensity of the pain is not always proportionate to the degree of retraction, provided no effort is made to overcome the latter. Pain in the extremities, especially in the legs, is also a common symptom. Movement of the body often brings it on or intensifies it. It shifts from place to place, and is of a darting character. Severe darting pain frequently attacks other parts. In the abdomen it is often situated in the epigastric and umbilical regions or is accompanied by obstinate vomiting, and in the chest it is attended by difficulty in breathing. In a recent sporadic case in a boy aged thirteen years, already referred to, the attack began with intense pain. The child, who was out playing ball, came home at noon in a state of marked collapse, with pale, sunken features; very weak, small pulse; subnormal temperature; vomiting, and such severe epigastric suffering that the first supposition was that he had received a blow there or that he had

taken some irritant poison. Reaction was brought about with extreme difficulty. When I saw him in consultation on the second day the diagnosis could be made out, and the case ran a long, desperate course to a final lingering recovery. Pain often begins in one portion of the body and darts with lightning-like rapidity to some other, perhaps leaving the first region at the same time, but perhaps also continuing there. After a few days pain usually begins to grow less, and by the end of the second week is much less marked.

Widespread hyperæsthesia of the skin, and afterward of the soft parts and the joints, is a common and important symptom, though by no means always present. It seems to vary with the epidemic. It is oftenest observed in the lower extremities. The skin may be so painfully sensitive that the slightest touch will cause an outcry. An attempt forcibly to open the eyelids or to straighten the neck or back gives pain, independently of that in the muscles which is produced by this action. The introduction of the thermometer into the rectum sometimes evokes an outcry. Hyperæsthesia is one of the early symptoms, appearing by the second or third day. It is often associated with great sensitiveness to light, sound, and odors. Partial anæsthesia sometimes occurs, but is not frequent.

Vertigo is often present, and may develop early with the headache. It may be one of the prodromes, and be so severe that walking is nearly impossible, and patients may fall and be unable to rise. It is sometimes present even when the patient is reclining.

The mental state varies. Many patients seem simply apathetic. Restlessness is of common occurrence except in mild cases or in those in coma. The severity of the pain causes constant tossing, so that the patient may move all over the bed. Sleeplessness, too, is often present, and is sometimes one of the prodromes. But little genuine sleep is obtained, although a drowsiness which borders on coma is common. Delirium is a very frequent symptom, and exhibits the greatest variety both in degree and in kind. One form may rapidly change into another. Though sometimes one of the earliest symptoms, it usually does not develop until the second or the third day. Not infrequently it is so violent that restraint is demanded. It may also show itself as a simple delusion, or it may resemble intoxication or hysteria. It is seldom continuous throughout the whole attack, but is liable to alternate with lucid intervals or with somnolence. Coma eventually follows delirium in nearly all fatal cases, but usually only a short time before death. Patients may, however, exhibit pronounced coma and yet recover.

Of motor symptoms the most characteristic, and one rarely absent, is contraction of the muscles of the nucha, causing retraction of the head. It may appear by the close of the first day, but far more often not until the end of the second day. When once developed it may be persistent, lasting even into convalescence. It varies in intensity from a slight stiffness to a retraction so great that swallowing is difficult. Hart reports a case in which a slough in the tissues of the back was produced by the occiput pressing between the scapulæ. In a large number of cases there also exists a tonic contraction of

the erector spinæ muscles, producing gradations from mere stiffness to complete opisthotonos, though the latter is unusual. It renders the raising of the patient in bed both difficult and painful. The stiffness lasts several weeks, even sometimes well into convalescence. Rarely the muscles upon only one side of the spine have been contracted.

Less common than the spasm of the muscles of the neck and back, though still quite frequent, is that of those of the abdomen and extremities. The thighs are flexed upon the abdomen, the legs upon the thighs, and the forearms upon the arms. Both active and passive movements are painful and difficult of execution. Trismus is occasionally seen, and is a most unfavorable symptom.

Clonic spasm of the muscles is less frequent than the tonic contraction. It is oftenest seen in young children. It may vary in degree from twitching of certain muscles to general epileptiform convulsions. In children general convulsions sometimes take the place of the chill in ushering in the disease, but they may exceptionally constitute the first symptom in adults as well. Violent convulsions may occur repeatedly during several days, or even throughout the disease, and yet the case may recover. Or, again, they do not occur until late in the disease, and are then, as a rule, accompanied by a decided increase in the severity of all the symptoms. Clonic spasm may be associated with paralysis of other muscles or may alternate with tonic contraction. Choreiform movements have been sometimes observed. Paralysis is one of the less common symptoms. It has been noticed even among the initial symptoms, but this is very rare, and it is generally one of the later ones. It affects most often associated groups of muscles, as those of deglutition or articulation, or of some one of the limbs, or it may develop in the region supplied by some one of the cranial nerves. Hemiplegia and even general paralysis have been reported, but are rare. Recovery from paralysis usually takes place as convalescence advances, but the condition may be more or or less permanent.

Of the special senses, that of smell is not often affected, as far as can be determined. Patients are sometimes sensitive to odors, and J. L. Smith reports an instance in which the sense was entirely lost in one nostril. Taste appears to be no more affected than would naturally result from the influence of the febrile state upon the tongue and mouth. The eye is often involved. Intolerance to light is a very frequent symptom. The condition of the pupils varies greatly. They may be normal or dilated or contracted either early or late, or one may be dilated and the other contracted. Dilation is perhaps more common than contraction. They very usually do not react well to light. Strabismus, generally convergent, is frequent, and may develop at any time, and last from hours to weeks or even be permanent, or may occur several times during the attack. Nystagmus is uncommon. Inflammatory hyperæmia of the conjunctivæ often occurs, and may pass into intense conjunctivitis with great tumefaction of the lids. Exceptionally the cornea ulcerates and the globe collapses. Suppurative irido-choroiditis or optic neuritis sometimes occurs. As a result of the ocular lesions permanent blindness has been

repeatedly reported. Transient loss of vision is also observed, and, it has been claimed, may be one of the earliest symptoms. The symptoms connected with the ear are important. The patients early become sensitive to noises, and often complain of ringing in the ears, and become somewhat deaf within the first two or three days. These symptoms are generally bilateral. A catarrhal inflammation of the middle ear is of frequent occurrence; a suppurative lesion less common. As the patient emerges from the stupor or delirium it may be found that he is totally deaf. This may or may not be followed by perforation of the membrana tympani and purulent discharge. Even when the deafness is complete and lasts for weeks, it may gradually subside and hearing be partly regained.

Alterations of the skin are very common in certain epidemics, though rare in others. Their nature, too, varies with the epidemic. The *tâche cérébrale* is usually to be obtained. On the second or third day, or occasionally much later, an eruption of herpes is apt to occur. This usually begins as herpes labialis. The individual lesions are numerous and large; often they become confluent and form an area of racemose form with thick, purulent contents, which soon dry into crusts of peculiar thickness and dark color. Not rarely the eruption spreads to the cheeks, or separate patches of herpes form about the nose, eyelids, or ears, and greatly disfigure the patient. Herpes facialis is thus more common in cerebro-spinal fever than in even malaria or pneumonia, and although the large racemose patches which may appear on the chest or other parts of the body are less frequent than those on the face, they are decidedly more often observed in this affection than in any other disease. Thus they possess a certain diagnostic value.

Petechiæ and larger purpuric spots, and even extensive ecchymoses, are frequent in some epidemics. They gave rise to the name "spotted fever," and were formerly regarded as more characteristic and of higher diagnostic value than at present. I have repeatedly seen the true nature of typical cases overlooked on account of the absence of such an eruption. It may be limited in extent or widely diffused. The color is sometimes bright red at first, growing darker in a short time, but is often dark purple from the beginning. The extensive ecchymoses and larger spots are characteristic of the more malignant cases. In the Philadelphia epidemic of 1863 an eruption of petechiæ occurred in the vast majority of cases, and usually on the first or second day, often indeed within a few hours of the attack. Petechial rashes have certainly been more frequent in America than in Europe, and even here seem oftenest to have been observed in the earlier epidemics. A dusky surface with livid mottling may appear without any distinctive eruption. Among other eruptions sometimes seen are erythema, sudamina, urticaria, ecthyma, pemphigus, and rashes resembling measles or scarlatina. Erysipelas has been observed. Gangrene of the skin may develop with a rapidity which indicates direct disturbance of the trophic innervation. Several different eruptions often occur on the same individual. A symmetrical distribution of the eruption is often a very noticeable feature. A marked

liability to the production of bed-sores is found in some cases. It is consequently necessary to guard all points which are subjected to continuous pressure. This tendency comes at times from the profound dyscrasia of the blood, while at other times it seems rather to depend on the impaired trophic influence of special nerves involved in the morbid process.

The temperature of cerebro-spinal fever is exceedingly irregular, and there is no characteristic curve. The fever is usually moderately high by the second or third day, if not, indeed, upon the first. Ziemssen places the average temperature at 100.4° to 103° F. In many cases, however, there is but little fever, and it may be that only when the thermometer is placed in the rectum does an increase of temperature above the normal become manifest, the axilla and mouth showing no alteration. The temperature at the onset may be subnormal a short time, and the same thing may be observed temporarily later, as during the brief remission occasionally witnessed about the third day. In certain severe cases the temperature may be truly hyperpyrexial. Wunderlich has recorded it in one instance as 107.5° F., and as still somewhat higher shortly after the death of the patient. The curve exhibits variations which are great, sudden, and rapid. It fluctuates remarkably from day to day, and even from hour to hour, and there is no regularity in the differences between morning and evening temperature. Sometimes accessions of pain are accompanied by increase of fever. In the intermittent form, as already stated, there occur daily or every second day exacerbations of fever with alternating periods of apyrexia, but without the regularity characteristic of malarial infection. The variations are apt to be so marked in all forms of this disease that when it does not too greatly annoy the patient it is well to take both the axillary and the rectal temperature.

The pulse is likewise very variable. It is generally full and strong at first, but becomes feeble and small in states of general depression. It may occasionally be abnormally slow at the outset, but soon increases in frequency, in fatal cases becoming too rapid to permit of being counted. Its rate is not at all in proportion to the elevation of temperature, and it is nearly always rapid in children. The pulse may change from slow to fast, and *vice versâ*, even within a few minutes, this variability forming a very constant and characteristic symptom of the affection. Not infrequently the pulse is irregular.

Palpitation of the heart is sometimes a very annoying feature. Blood taken from patients by venesection usually shows an increase in the amount of fibrin—an observation which is explained by the fact that it is only in cases of inflammatory type that any one would think of bleeding. In cases of grave, infectious type the blood becomes quickly dark and diffluent.

Respiration may be unaffected, but in severe cases is liable to become sighing, labored, intermittent, or slow. In fatal cases it may assume the Cheyne-Stokes type.

Vomiting, the most prominent of the digestive disturbances, is in reality dependent purely upon nervous influences. It is an initial symptom very constantly present. It may last a few hours to one or two days, and then disap-

pear, perhaps to recur later in the course of the attack. It is often accompanied by vertigo, and usually by faintness. Sometimes it is a troublesome symptom throughout the whole sickness, and may become a most dangerous one, on account of the exhaustion which ensues from lack of sufficient nourishment. Appetite may persist in full force in spite of the vomiting, but in other cases is lost. Taste is impaired. The tongue may be but slightly coated, and continue moist throughout the disease, even though there may be decided hebetude and delirium. This has seemed to me to be in part due to the fact that the mouth is less apt to be kept open than in typhus or typhoid fever. In cases which run into the typhoid state the tongue becomes brown and dry, and sordes form freely. The throat is sometimes inflamed. Aphthous stomatitis has been reported. Thirst is generally very great. Inflammation of the parotid gland has been occasionally met with. Jaundice has been seen in a few instances. The abdomen may be as strongly retracted as in tubercular meningitis. I have frequently seen this continue for weeks during the whole duration of prolonged cases, occasionally becoming so extreme toward the close of those fatal cases which run a very long course with great marasmus that the spinal column and the various abdominal organs might be felt with extraordinary distinctness through the wasted abdominal walls. On the other hand, in cases of the typhoid type distension of the abdomen, partly, at least, of paretic nature, with uncontrollable looseness of the bowels, may develop. Constipation rather than diarrhœa is, however, the rule in cerebro-spinal fever.

The secretion of urine is sometimes greatly increased, even when the temperature is high. The amount passed is, in fact, oftener increased than normal, but is sometimes diminished. Albumin or sugar has been occasionally observed, and casts and blood are more rarely found. Retention of urine may occur in coma and necessitate catheterization. On the other hand, there may be incontinence. The spleen is generally not sufficiently enlarged to produce an increase of percussion dulness.

Complications and Sequelæ.—The complications have already been outlined to some extent in the description given of the symptoms. Certain others in addition to these may be passed in review. Broncho-pneumonia, croupous pneumonia, pleurisy, atelectasis, bronchitis, endocarditis, and pericarditis are not uncommon. The frequent combination of croupous pneumonia with meningitis often renders it doubtful which is to be considered the primary disease in any individual case. It is certain, at least, that pneumonia frequently develops during the prevalence of epidemics of cerebro-spinal fever, and that the two affections are often closely associated. Various other infectious diseases, as malaria, measles, typhoid fever, scarlatina, and cholera, have occurred in connection with cerebro-spinal fever. Intestinal catarrh is also seen, and parenchymatous changes of the liver and kidneys are occasional complications.

Of the sequelæ, permanent blindness and deafness are among the most important. Cerebro-spinal fever has been a very frequent cause of deaf-mutism, and careful inquiry will elicit the fact that a considerable proportion of all cases in institutions for the deaf and dumb are traceable

to meningitis. The deafness is probably oftenest due to an inflammatory involvement of the labyrinth. Aphasia and imperfect articulation may, like the deafness, be produced by the disease. Headache is often the most troublesome sequel, persisting for months or even years. Mental feebleness is often observed. Ziemssen regards chronic hydrocephalus as a sequel by no means rare. The symptoms of this condition consist of "paroxysms of severe headache, pains in the neck and extremities, with vomiting, loss of consciousness, convulsions, and involuntary discharges of fæces and urine." He regards the prognosis of this condition as nearly always hopeless.

Paralyses of single extremities or of the parts supplied by the cranial nerves are not very uncommon. They depend on lesions of the brain or cord or of the nerves themselves. Most of the cases recover after a few months; which suggests that they have been due to lesions of the nerve-trunks, and is confirmatory of the view that perineuritis and neuritis are of common occurrence in this disease.

Relapse and Recurrence.—Relapses are common. They sometimes occur so frequently, and prolong the case to such an extent, that a "chronic form" of cerebro-spinal fever has been made a distinct type by some writers. I have, however, already expressed the opinion that more frequently the protracted course of the case is due to a persistent or progressive lesion, such as chronic meningitis, chronic hydrocephalus, or even abscess of the brain. The occurrence of the disease does not afford a complete immunity from a recurrence of a second attack. Miner found several instances of a second attack of the affection in which the patients had suffered from it the year previous.

Diagnosis.—Ordinarily the diagnosis of cerebro-spinal fever is a matter of no great difficulty. Sometimes, however, it is almost impossible in the early stage, as when the disease occurs sporadically or at the beginning of an epidemic. In young children also, or when in combination with other diseases, the affection may be very difficult to recognize.

The most characteristic features are the sudden onset and rapid advance; prostration; intense pain in the head, neck, back, and limbs; vomiting; faintness; vertigo; tonic contraction of the muscles of the neck, and, later, of the back; clonic local or general convulsive movements; hyperæsthesia; delirium alternating with somnolence; very irregular pulse and temperature; and the cutaneous eruptions. The diagnosis of mild sporadic cases is rendered additionally difficult by the fact that in these the eruption, the hyperæsthesia, pain in the back and extremities, and stiffness of the spine may be mostly wanting, while the pain and spasm at the back of the neck may not be as well marked as in the epidemic form.

Several affections are likely to be confounded with cerebro-spinal fever.

Tubercular meningitis very closely resembles it if the case is seen after the development of partial or complete unconsciousness, with stiffness of the neck, cerebral cry, irregular fever and pulse. But if there has been an opportunity to observe the case from the start, it will be readily recognized as one of tubercular nature by the longer prodromes and more gradual onset, with headache,

vomiting, and constipation; the characteristic stages of alteration of the pulse; the earlier appearance of retraction of the abdomen; the less degree of retraction of the head, of hyperæsthesia, and of pains in the extremities; the rarity of petechial or herpetic eruptions; the greater frequency of Cheyne-Stokes respiration; the detection of choroidal tubercles by the aid of the ophthalmoscope; and the longer course and invariably fatal ending.

The *cerebral form of typhoid fever* may occasionally simulate cerebro-spinal fever very closely. Cases of it may exhibit high fever, headache, delirium, stiffness of the neck, tremor, and spasm of the muscles. Ordinarily, however, typhoid fever differs widely in the slow onset, absence of vomiting and of muscular spasm, and presence of continuous hebetude, typical temperature curve, characteristic cutaneous eruption, epistaxis, abdominal tenderness with diarrhœa, and greater enlargement of the spleen.

Typhus fever has been repeatedly confounded with cerebro-spinal fever, and it is true that in certain epidemics cases present themselves which are difficult to discriminate. This is, of course, pre-eminently the case when outbreaks of the latter occur in localities or under conditions which render the occurrence of typhus plausible or where it already exists. Between cases of the cerebro-spinal type of typhus fever and of cerebro-spinal fever the points of resemblance are the sudden onset with rapid development of delirium and stupor, the extreme hyperæsthesia and muscular soreness, the headache, rigidity of the cervical muscles and occasional retraction of the head, and the occurrence of a petechial eruption. On the other hand, typhus fever is a highly contagious disease, which does not occur sporadically in this country nor in epidemic form unless in seaports, where it occasionally spreads from imported cases. High initial fever is almost constant in typhus, and the temperature curve is characteristic; the facies is distinct; the eruption is constant, and appears about the fourth day as a roseolous rash, becoming gradually petechial instead of occurring as it does, when present in cerebro-spinal fever, almost at the very onset of the attack; herpes does not occur in typhus; vomiting is rare; convulsions are much less frequently seen; there is much less pain, and muscular rigidity is less marked.

Influenza may not seem likely to be confounded with cerebro-spinal fever, yet there are points of striking analogy which demand attention. These two affections are distinguished from other infectious diseases by their remarkable pandemic character. In the various epidemics of influenza the utmost variety in its manifestations has been exhibited. Although catarrhal irritation of the mucous membranes, with fever of irregular type, is the usual expression, there is a proportion of cases, varying in different epidemics, where the force of the disease falls on the nervous centres, and cerebro-spinal meningitis is developed with severe pains of perineuritis, muscular soreness, rigidity of the cervical muscles, and retraction of the head, convulsions, delirium, and stupor. Death is frequent in these cases, but when they recover lesions of the organs of special sense, of the peripheral nerves, or of the nervous centres may remain. It is well known now that even in ordinary cases of influenza peri-

neuritis is of frequent occurrence. During the recent severe epidemic many cases of meningitis, as above sketched, have occurred. It is altogether probable that in some instances these were sporadic cases of cerebro-spinal fever, but it is also clear that there was a sudden development of meningitis in other cases which were unmistakably influenza.

Rheumatic fever, with little or no joint involvement, but with muscular soreness and rigidity, retraction of the head or trismus, and with marked cerebro-spinal symptoms, either connected with hyperpyrexia or with actual meningeal irritation, may occasionally closely simulate cerebro-spinal fever. It undoubtedly happens that at times cerebro-spinal rheumatism is regarded as cerebro-spinal fever, and the reverse.

Pernicious malarial fever may resemble malignant cerebro-spinal fever in the rapid development of collapse and coma. It can be distinguished from it by the consideration of the etiological circumstances and by the fact that the first malarial paroxysm rarely exhibits the malignant character. The detection of the malarial organism in the blood, the greater enlargement of the spleen, and the specific effect of quinine properly administered will establish the diagnosis.

Malignant scarlet fever may occasionally resemble cerebro-spinal fever in the sudden onset with high temperature, vomiting, convulsions, and stupor. Its presence, however, may be suspected from the early redness of the fauces. The appearance of the scarlatinal rash settles the difficulty unless death takes place before this occurs.

Small-pox may also be mistaken for it, the resemblance being in the severe pain in the back and head, in the vomiting, and in the development of a purpuric rash in some cases. The papular character of the eruption and the general course of the disease aid in distinguishing it.

During epidemics of cerebro-spinal fever occasional cases are noted where death occurs in a few hours, when there has been little or no rise of temperature, but such extensive appearance of petechiæ and ecchymoses as to raise the question of the fulgurant type of *purpura*.

Meningitis secondary to croupous pneumonia and other acute diseases is with difficulty distinguished from cerebro-spinal fever. In the cerebral form of pneumonia, whether a true meningitis or a pseudo-meningitis be present, there are often considerable tremor and muscular spasm, but the stiffness of the muscles of the neck and back and the hyperæsthesia are not so marked. In some cases, however, it may be very difficult to determine whether we have to do with pneumonia complicated by meningitis or with cerebro-spinal fever complicated by pneumonia.

Mild cases of cerebro-spinal fever have sometimes been mistaken for *hysteria*, but the severe pain, the muscular rigidity, and the occurrence of fever should prevent the mistake.

Duration, Mortality, Prognosis.—The course of the disease is very variable. In the milder forms and in the most malignant variety the duration varies from a few hours to five days. The moderately severe

cases begin to recover after one or two weeks, but may be prolonged for months. Convalescence is comparatively slow and subject to many interruptions from complications and sequelæ. The first week of the disease is usually the time of greatest danger, and patients who live until the close of the second week will probably recover. At the same time, the prognosis in any individual case is a matter of the greatest uncertainty, both as regards life itself and as regards the persistence of permanent danger of some part. In moderately severe cases no prognosis at all can be made for some days, and even then it must be most guarded. Malignant cases nearly always die, but even to this there are exceptions. Mild and abortive cases generally recover, but here, too, a guarded prognosis must be given. Under five years and over thirty years of age the prognosis is less favorable than between these periods. In children under two years of age the rate of mortality and the danger of grave sequels reach the highest point.

Symptoms generally unfavorable are abrupt and violent onset, evidences of great excitement, hyperpyrexia, coma, convulsions, great prostration of strength early in the affection, irregular respiration, unusually intense headache, persistent vomiting, evidences of extensive disorganization of the blood. The occurrence of complications, especially those connected with the lungs, increases the gravity of the prognosis.

The mortality of cerebro-spinal fever is very great, varying much in different epidemics. Ziemssen places it at 30 per cent. for mild epidemics and over 70 per cent. for the most severe ones, the general mortality averaging 40 per cent.

Treatment.—*Prophylaxis.*—Little can be done in the line of prophylaxis, inasmuch as we know so little regarding the cause of the affection. The avoidance of unsanitary conditions in streets and houses is of course an important matter. It is also advisable that the inmates of a house in which the disease has broken out should leave it until after the epidemic is over, since there sometimes seems to be a tendency for the affection to spread in families. Linen used about the patient should be disinfected or destroyed. As in the case of other serious infectious disease, it is important during an epidemic of cerebro-spinal fever to avoid fatigue of any sort, to lead as quiet a life as possible, and to preserve the general health in the best possible condition. Indeed, it is to be recommended that persons in poor health leave the locality while the epidemic lasts.

Treatment of the Attack.—The great variety of type in different epidemics and in different cases in the same epidemic, and the highly complex group of symptoms presented, explain the impossibility of formulating any uniform plan of treatment. It is necessary in each individual case to adapt our remedies to the grade and to the special localization of the morbid process.

The room should be kept dark and quiet. The diet should consist of easily assimilable liquid substances, given freely and often, since the disease is one in which exhaustion so readily supervenes, and in which there is rarely any lesion of the gastro-intestinal tract. As soon as the fever has abated solid

food should be administered. Water is to be given freely at all times. In many cases the obstinate cerebral vomiting interferes with the administration of food at first, and in such nutrient enemata may be employed. It is often difficult to secure the ingestion of enough food, but I have never found it necessary to resort to forced feeding by means of a stomach-tube. Even when prostration is apparently not great, the patient should not assume the erect position in bed, as dangerous syncope may follow. Until convalescence is complete all exertion and excitement must be shunned, and a return to the ordinary methods of life is well deferred for some time.

Venesection was early advocated, and the symptoms of acute, intense cerebro-spinal irritation often suggest it forcibly. It must be remembered that when fever is high the blood becomes rapidly disintegrated, and such profound debility soon develops as to render depletion dangerous. In young children, also, it is nearly always inadmissible, and even local bleeding has been followed by alarming depression. On the other hand, in the onset of cases of sthenic type, where the pyrexia was moderate and the pain and cerebro-spinal irritation were extreme, I have bled healthy adults with great advantage, finding the blood highly coagulable. The prompt relief afforded to the pain and central congestions has been followed by improvement in the force and volume of the pulse. In carefully selected cases I would therefore advise moderate venesection soon after the onset. When the propriety of this is doubtful, as in delicate or young persons, when the fever is high, or when the first day or two has passed, wet cups or leeches, or even dry cups alone, may be applied to the temples, the mastoid regions, the nape of the neck, or along the spinal column.

Cold to the head and spine is a valuable therapeutic measure. It should be applied for hours continuously in the form of ice-bags or in Leiter's tubes. Cold affusions or cold sponging may be substituted sometimes. Cold is particularly useful in the early stages when pain in the head is at its worst. It often decidedly relieves the suffering and produces quiet sleep. The application should be renewed as often as pain returns. Cold baths to reduce temperature may be given systematically if the fever be high. As, however, the temperature is rarely either high or apparently provocative of dangerous symptoms, this measure is not often indicated.

Moist or dry heat locally applied is of value both in mitigating violent symptoms and in anticipating or removing the collapse which is so apt to come on in this disease. Hot mustard foot-baths, hot bottles, bricks, or sand-bags, hot moist flannels and the like are all useful for this purpose. It is well to apply heat to the rest of the body while cold is being used about the head and spine. In this way any depressing effect of the cold is prevented.

Blisters have long been employed to relieve pain and to lessen congestion. It is true that sometimes they seem to diminish pain, delirium, spasms, and coma. It is questionable, however, whether they are, as a rule, of any lasting benefit, while, on the other hand, they are liable to add to the annoyance and suffering of the patient. If used at all, they should be applied at the back

of the neck very early in the disease, and should be allowed to vesicate superficially only. It is, however, in my judgment, much safer and more efficient to use light applications of the thermo-cautery over the mastoid or at the nape of the neck.

Alcohol is often of the greatest value. Many cases do not need it at any time, and those of an inflammatory type may be injured by it if administered at the onset; but its use should be promptly commenced when there is the slightest sign of exhaustion. The amount administered should in every case depend upon the effect produced, care being taken that too much is not given, although patients with cerebro-spinal fever often bear unusually large amounts. If under its use the pulse grow stronger and the heart-sounds better, stimulants are doing good; but if excitement increase and the heart beat more rapidly, the stimulants must be diminished or entirely withdrawn.

Opium is the drug upon which the greatest reliance is placed by the majority of writers. Its use began with American physicians, but has since become widely extended. It may be given by the mouth or rectum, or, in the form of morphine, hypodermically. The tolerance for it in this disease is remarkable. As much as the equivalent of a grain of opium hourly may be required in severe cases, and even larger doses have been given. The drug lessens the spasm, pain, hyperæsthesia, and sleeplessness, and strengthens and retards the pulse. Its employment must be commenced early in the disease, and the dose must be repeated and, if necessary, increased until the severe symptoms come under control. As already stated, in severe cases of sthenic type in vigorous adults a moderate venesection may be used at the outset with marked relief to the suffering and with the effect of rendering the action of opiates much more efficacious in doses smaller than are usually required.

The action of opium must be carefully watched, especially in children. I have usually found the best results from the administration of a hypodermic injection of morphine and atropine morning and evening, followed up in the intervals by the use of deodorized tincture of opium in appropriate doses as indicated by pain and restlessness. It is possible that the relaxing effects of the opiate, associated with the action of cold and derivatives, may exert some influence in lessening the amount of meningeal exudation. After effusion begins opium is of much less value and the dose must be diminished.

Mercury, given in the hope of influencing meningeal inflammation, is now generally considered to be of no value in the early stage, though formerly much used for this purpose. Ziemssen recommends it both by inunction and internally, but admits that it is doubtful whether it exerts any beneficial influence. It is of much greater value in the treatment of the sequelæ.

Quinine in very large doses has been tried, but there is no evidence that it exerts any definite effect, even in the intermittent form of the disease. There is danger, too, that it may disturb the digestion, as well as exert a depressing action. In moderate doses it may be used for its tonic action in the form of suppository or enema. I often direct with apparent advantage a suppository morning and evening, containing 10 grains each of quinine and asafœtida.

Ergot and belladonna have been employed on account of their power to lessen congestion of the cerebro-spinal capillaries. Although favorable results have been reported, there does not seem to be good reason to attribute them to the action of these remedies. Rosenthal believes that belladonna must be given with great caution. In small doses it may be combined with opium.

Calabar bean was recommended by N. S. Davis on the ground that it was useful in tetanus. Cannabis Indica, gelsemium, chloral, inhalations of chloroform or ether, bromide of potassium, aconite, and veratrum viride have all had their advocates. Certain of them, as chloral, aconite, veratrum, and chloroform, are certainly dangerous in a disease in which severe depression so readily develops. Inhalations of ether can be safely employed in very bad cases in order to give temporary relief from extreme restlessness, convulsions, or great pain. Bromide of potassium is sometimes of value, particularly in children and in the milder cases; but in doses sufficient to exert a decided effect in severe cases it is depressing to the general strength of the patient. It may be advantageously combined with opium.

Antipyrine, phenacetin, and drugs of this class would naturally suggest themselves on account of the severe pains, but they must be used, if at all, only with caution and close watching. Unless decided relief follow moderate doses at rather long intervals, it is safer to omit them. Their administration must never be pushed.

Any tendency to exhaustion or collapse may be treated by ammonia, turpentine, digitalis, and, as already stated, by alcohol and heat. Hypodermic injections of ether may be required to favor reaction from the condition of collapse. The urinary bladder must be watched so as to guard against retention. The formation of bed-sores must also be anticipated and prevented by proper measures.

As soon as the acute stage declines it is well to give potassium iodide to favor the absorption of exudation and thickening. In conjunction with tonics, such as strychnine, iron, arsenic, cod-liver oil, its use may be continued during convalescence. When serious sequelæ, as deafness, blindness, perineuritis, persistent neuralgic pains, paresis, or evidences of exudation or effusion, indicate a continuance of morbid action, it is well to associate mercurial inunctions with the internal use of potassium iodide, and to employ repeated blisters or applications of the thermo-cautery at proper intervals. Electricity, hydrotherapy, rigid dieteties, and hygiene are also of great value. I have frequently seen the patient use of such combined treatment followed by the slow disappearance of sequels which threatened to be permanent.

INFLUENZA.

By WILLIAM PEPPER.

Influenza is an acute microbic fever, moderately contagious; sporadic, epidemic, and pandemic; associated with catarrhal inflammation of the mucous membranes and with disturbance of the nervous centres and trunks; often running a short and favorable course, but apt to be attended with many serious complications and sequelæ.

Synonyms.—As might be expected from its widespread prevalence and from its peculiar features, few diseases have had so many names bestowed upon them. Some of these names refer to a supposed geographical origin of the disease, as the Chinese, Russian, or Spanish catarrh. Others refer to the epidemic or contagious nature of the catarrh which is so prominent among its symptoms: thus, epidemic catarrhal fever, and its Latin and French equivalents, catarrhus a contagio, and many analogous terms. Many descriptive names have come into popular use, not a few of which refer to the fancied insignificance of the disease as observed in mild epidemics. The events of the past few years have, however, secured both professional and popular approval of three names only—influenza, an old name of Italian origin, given in allusion to the part played by astral or atmospheric influence in the causation of the disease; la grippe, derived from the French verb *gripper*, to seize; and grip, the familiar English equivalent. Gripped, grippal, to be gripped, grippo-toxine, and so forth, are terms whose convenient brevity may secure their continued use.

History, Nature, and Causation.—It is altogether probable that extensive epidemics of influenza have prevailed from the earliest ages, though it may be admitted that the first accurate descriptions of the disease date back only to the beginning of the sixteenth century. Since then there have been frequent epidemics, not a few of which have spread rapidly over entire continents, and have appeared almost simultaneously in widely distant countries. Repeated outbreaks have occurred in America since at least 1655. Careful study of the annals of influenza proves conclusively the identity of the disease with all its protean manifestations from the date of the earliest records down to the last great epidemic, which seems to have broken out in Bokhara in May, 1889. It had established itself in St. Petersburg in October of the same year; it was recognized in Paris as early as November; England was invaded early in November, if not in October; cases began to be of frequent occurrence in America toward the close of October or in November. The epidemic reached its height at almost the same date in January and February of 1890 in widely

distant localities; smouldered away during the ensuing summer, only to awaken to renewed activity in the late autumn of that year, and to prevail extensively until the spring of 1891, when it again subsided, but for a third time revived, in milder and less prolonged form, in the winter of 1891–92.

In the intervals of epidemic waves of influenza there occur in many, if not in all, large towns and thickly-populated districts sporadic cases which present all the features of the disease. It is probable that careful bacteriological study will identify the nature of these cases, which may be due to the action of the specific cause of influenza in a comparatively feeble form, so that it can affect only those who are specially susceptible.

The analogies between influenza and the infectious fevers of ascertained microbic origin are so close that for a long time the existence of a specific micro-organism has been assumed as its effective cause.

Early in this year (1892) Pfeiffer, working at the Hygienic Institute of Berlin, discovered in the sputa of influenza numbers of a bacillus which he was soon able to recognize as characteristic and to cultivate. The bacillus of Pfeiffer is a short organism about one-half the length of the bacillus of mouse-septicæmia, and nearly the same thickness as the latter. It stains with difficulty, but may be well shown, by using Zichl's carbol-fuchsin or with other stains, to consist of two bulbous ends joined by a narrower and less intensely staining central shaft. It might easily be mistaken for a diplococcus if care were neglected and the examination made hurriedly.

The bacillus occurs abundantly in the sputum, and is said to have distinct relation in number to the severity of the disease, and to disappear with cessation of the fever and cure of the disease. It has never been shown to be present in any other malady.

In the lungs Pfeiffer showed that it penetrates to the peribronchial connective tissue and to farther outlying portions of the lungs.

It was demonstrated in the blood of 20 consecutive cases at the Moabit Hospital of Berlin by Canon, and since then in many other cases by competent persons.

It has been cultivated in agar containing a small percentage of sugar, and grows as small watery droplets along the line of inoculation. It could easily be overlooked. It grows scantily in bouillon, which remains clear. It cannot be cultivated in gelatin, as it requires temperatures which liquefy gelatin. Letzerich used potatoes, finding a temporary growth. The bacillus was cultivated by Kitasato, who fully substantiates Pfeiffer's statements, to the fifteenth generation, when it still presented its characteristic features.

Inoculation has been repeatedly practised. Apes and rabbits get quite typical influenza. Cornil and Chantemesse injected some of the cultures into the anterior chamber of a rabbit's eye, and soon found the bacilli in the blood. Some of the latter, mixed with sterile sugar-agar for twenty-four hours, was introduced into a monkey's nose, soon causing coryza, fever, depression, but not death, the bacilli being present in the bronchial and nasal mucus. Mice succumbed to inoculation, and the bacilli were found in the congested viscera.

The observations of Pfeiffer have been confirmed, among others, by Kitasato, Canon, Cornil, Chantemesse, Babes, and Letzerich. The last named examined the sputa of 50 cases not influenza, and failed completely to demonstrate influenza bacilli. In no genuine case has Pfeiffer failed to find them.

Canon finds them in the blood as clumps of ten or twenty, sometimes fewer. He stains with solutions of eosin and methyl blue in alcohol. The bacilli stain blue, the red corpuscles pink.

The isolation of the bacillus of influenza does not by any means clear up all the difficulties as to its remarkable outbreaks. It would seem as though no ordinary mode of propagation by contagion and by fomites could explain its almost simultaneous appearance at widely-distant points and its wonderfully rapid spread throughout large communities. The micro-organism may be almost universally distributed, but capable under ordinary circumstances of causing only occasional sporadic cases, while under special atmospheric or telluric conditions it acquires a degree of virulence that renders all subject to its attacks. It would seem that the susceptibility to this poison is more general than in the case of any other infection save that of variola.

The evidence is clear that the disease is readily communicated by contagion. It is possible that the specific poison can be carried by fomites, and there are instances where it seems to have been conveyed by the corpse of the patient dead of influenza. It appears that it is received into the system by means of the inspired air, or at least there is as yet no evidence to show that water or milk can convey it. It must be remembered that the early scattered cases which precede a great outbreak may be overlooked as to their real nature, and the seeds of the disease be gradually distributed, so that when the conditions favorable to its active development arise there is already widespread preparation.

There are no known conditions of climate, soil, elevation, or season which affect it. It has occurred at sea as well as in the driest localities.

It is well known that epizoötics, or epidemics of catarrhal nature, have occurred frequently among domestic animals. The exact nature of these infectious diseases is, however, not established with accuracy; nor is the evidence clear as to their transmission to the human subject, nor as to the acquisition by animals of influenza from man.

During the prevalence of an epidemic of influenza all are liable to be affected. Young children are, however, less frequently and less seriously attacked than older persons. On the other hand, aged and infirm persons, those of nervous temperament, and those whose vitality is depressed by fatigue and anxiety, are specially liable; but the most robust health does not give immunity.

Relapses of the disease are common, and second, third, or even more numerous attacks in one individual may be noted. The susceptibility of the system during and immediately after an attack is extraordinary, and slight exposure or exertion may induce grave complications with startling rapidity and violence.

Morbid Anatomy.—There are no anatomical lesions characteristic of influenza. The bacilli now regarded as associated in the production of the disease are found in the sputum, the tissue of the lungs, and in the blood. In the great majority of uncomplicated cases recovery follows. If death occurs from the intensity of the fever or from debility, catarrhal inflammation of the respiratory and digestive mucous membranes is found, with the ordinary changes of congestion and swelling. The intestinal glands are but slightly, if at all, enlarged. The disease may, however, at the very onset excite pneumonia or meningitis, or these may supervene as complications; and in the same list must be placed nephritis. The pneumonia may be either lobular or lobar: even in the lobar form both lungs are often involved and the lesions may be unusually extensive. Plastic pleurisy is commonly associated with the pneumonia. Pleurisy with sero-fibrinous or purulent exudate also occurs independently. Purulent pericarditis was observed with unusual frequency during the recent epidemic of influenza.

But few autopsies have been made of cases complicated with cerebro-spinal meningitis, but it can scarcely be doubted that this lesion is actually present in not a few instances. Perineuritis is of common occurrence.

General Clinical Description.—So varied are the phenomena of influenza that it is difficult to present a brief sketch of its symptoms. The main features in a majority of cases in most epidemics are a sudden onset with chill of moderate severity; fever, which comes on quickly, does not usually exceed 103° F., pursues an irregular course of from four to seven days' duration, and tends to terminate by crisis; naso-pharyngeal and bronchial catarrh, with sneezing and coughing; headache and pains in the back, with myalgic pains in the trunk and limbs, and with a general soreness as though bruised or beaten; depression of spirits, with great malaise and restlessness; marked general and cardiac debility; slight enlargement of the spleen; absence of characteristic eruption.

In all epidemics there are many cases of a type so mild that the patients pay little or no attention to the attack, regarding it as an ordinary catarrhal cold, and continuing at their usual avocations. It is impossible to estimate the number of such cases, but it is certainly extremely large in some outbreaks. In spite of the apparent mildness of the attack, the temperature will often be found much elevated. During the epidemics of 1889–90 and 1890–91 it was common to have patients come to physicians' offices or to find patients walking about their houses with a temperature of from 102½° to 104°. A widespread febrile tendency exhibited itself; the most trivial ailments were attended with high fever; the entire population seemed to be infected. There was danger of a sudden development of grave or rapidly fatal complications even in the mildest cases if exertion and exposure were continued. Doubtless many instances which seemed like the sudden onset of malignant infection were really of the above nature.

Cases of grave or malignant type do, however, occur in varying proportion in most epidemics. Sometimes the character of an outbreak is so uniformly

grave that the cases bear but little superficial resemblance to the common conception of influenza. The unusual severity of the infection shows itself by hyperpyrexia, alarming prostration, or the early development of dangerous pulmonary, nervous, or gastro-intestinal lesions, or by a profound blood-dyscrasia from the intense virulence of the poison.

In addition to these varying degrees of gravity it is essential to recognize the fact that influenza manifests itself only in a proportion, though a large one, of all cases by catarrh of the respiratory tract, while in others it appears as an acute gastro-intestinal catarrh; and in still a third group marked disturbances of the cerebro-spinal functions constitute the prominent symptoms.

Certain cases present one or the other of these aspects exclusively, but for the most part there is a blending of the various phenomena. This is most notably so in the case of the nervous element, which is so generally pronounced as to have led many observers to consider the localization of the infectious process in influenza essentially in the cerebro-spinal axis, and the widespread functional disturbances or serious lesions of other organs to be secondary results of impaired nervous action.

In the thoracic form catarrhal symptoms are prominent, coryza is usually marked, and the cough is severe, paroxysmal, and painful. The pain is referred to the substernal region, and there are sharp myalgic pains about the chest. The sputa are scanty and tenacious. There is often a marked sense of oppression. The physical signs consist of irregularly distributed râles, large and small, crackling in character for the most part, which often change their location and quality very rapidly. The respiratory murmur is apt to be feeble. The fever is moderate, and the pulse-respiration ratio not greatly disturbed unless complications ensue. The tongue is coated yellow, but remains moist; appetite is impaired, but thirst is usually marked. The bowels are quiet and the abdomen normal. Headache is common. Insomnia is a frequent and distressing symptom, but delirium is rare. There is a sense of profound weakness, and debility is in fact often so marked that the least exertion causes rapid breathing and heart-action with exhaustion. Profuse sweats are not unusual. The fever declines in from three to seven days; critical sweats or diarrhœa may occur, the chest symptoms subside, and convalescence ensues, marked by a strong tendency to recurrence of catarrhal irritation.

Pneumonia is a frequent complication of this type. It may develop insidiously while the patient is in bed; it may occur at the very onset of the attack; and in many cases it is induced by exposure even in cases of apparently trifling character.

In the gastro-intestinal form the symptoms may be of moderate severity, consisting of nausea and occasional vomiting, heavily-coated tongue, complete anorexia, fulness and tenderness of the epigastric region, some distension of the abdomen, and a tendency to looseness of the bowels, and fever not exceeding 102½° F., with headache and myalgic pains, and running a favorable course of from seven to ten days. But in other cases the onset is abrupt, with the

violence of cholera morbus, and indicating intense irritation of the mucous surface, with profound disturbance of the splanchnic nerves.

The nervous or cerebro-spinal form may be primary, but is often developed in the course of cases originally of another type. The headache is agonizingly acute; sight and hearing are morbidly acute; the pain in the back and legs and the general muscular soreness are intense; delirium may be marked, with an increasing tendency to stupor; rigidity of the muscles of the nucha, with retraction of the head, may be present; and general convulsions may occur both in children and adults. The temperature may be high, but in other cases it falls below normal, with slow, irregular pulse and breathing. It can scarcely be doubted that the more grave symptoms of this type are due to the development of a meningitis.

In all of these forms epistaxis is of occasional occurrence. There are no characteristic eruptions, but herpetic groups often appear on the lips or face. Urticaria is also common.

The identity of these different types of influenza is shown conclusively by the fact that all may be illustrated in a single family at the same time, that any one may be contracted by contact with a patient suffering with another type, and that the symptom of one form becomes associated with the fully-developed features of another as the case assumes additional gravity.

Complications and Sequelæ.—The most frequent complications are those connected with the respiratory organs. A certain degree of bronchial catarrh is to be regarded as among the usual symptoms, but there is a marked tendency in most epidemics to the occurrence of severe bronchitis of the larger tubes, or even to capillary bronchitis, associated in old or feeble subjects with œdema of the lungs, and attended with fever and often with a low form of delirium. In all probability patches of broncho-pneumonia coexist in many of these cases. A fatal result from cardiac failure or from progressive cyanosis is apt to occur.

Pneumonia, both croupous and catarrhal, is a frequent and fatal complication. This was pre-eminently so during the recent epidemic. In some cases the attack begins as one of severe infectious fever, with dyspnœa in excess of any demonstrable cause; but in two or three days the physical signs of pneumonia become manifest: cough may be trifling and expectoration almost absent, though the pneumonic area is extensive. It is by no means only in cases which present severe bronchitis that there is danger of this complication developing. In some epidemics it is so frequent as to suggest that it is induced by profound depression of nerve-force, so as to resemble the pulmonary lesions developed after section of the vagi. Guitéras and White have suggested that in some cases the bronchial glands may be acutely enlarged, and the nervous tract about the root of the lung may be involved. Children and aged and infirm subjects are especially liable to this complication of pneumonia. The readiness with which violent pneumonia may be induced by slight exposure, even in mild cases of influenza in vigorous adults, was remarkably shown during the late epidemic. A single instance will illustrate what was of frequent occurrence, though usually with less violence: A young

man of thirty years returned from a hunting-trip in fine physical condition to his home, where members of his family had influenza. He promptly contracted the disease in so mild a form that he did not consult a physician nor even remain in his chamber. By the fourth day he felt so much better that he insisted on walking about two hundred yards in the raw evening air. Within one hour he was taken with severe chill; the temperature was 105° F. by the time he was conveyed home; he vomited blood; pneumonia began in the left lower lobe, involved the entire left lung, then spread to the base of the right lung, was complicated with nephritis, jaundice, and delirium; and the autopsy showed that finally there was but a small area at the right apex which was not consolidated.

I secured statistics of 35,413 cases of influenza occurring in the practice of 272 physicians in Philadelphia: pneumonia occurred in 1485, or about 4 per cent., with a mortality of 173, or 11.65 per cent. Dr. Latta, chief medical examiner of the Pennsylvania R. R. Voluntary Relief Department, kindly gave me the statistics of influenza as affecting its members, who may be regarded as a carefully selected body of men. In 1890 and 1891 there were 6680 cases of influenza, showing that over 14 per cent. of the entire membership were so severely affected with influenza as to confine them to the house; pneumonia occurred in 138 cases, or in 2 per cent., with a mortality of 29, or 20 per cent. The accompanying chart (Fig. 12) exhibits the extraordinary prevalence and fatality of pneumonia during this epidemic. Undoubtedly in very many cases of death reported as from pneumonia the disease was grippal in nature.

Many cases of pneumonia presented an extraordinary feebleness of respiratory murmur, both before and after the appearance of consolidation. Typhoid delirium and a tendency to heart failure were common. Intense abdominal engorgement with jaundice, and slight intestinal hæmorrhage, were not rare. Both lungs were often involved and the mortality was high. Alison of Baccarat describes a focus of contagion which gave rise to eight cases of pneumonia in closely related families, and in every case a fatal result followed.

Plastic pleurisy is almost universally associated, and empyema may occur, as may also purulent pericarditis, either with or without pneumonia. Abscess and gangrene of the lungs are rare sequels. Pulmonary phthisis must be noted among the sequels also, and when influenza attacks those already affected with phthisis, the mortality is high and the course of the organic disease is greatly hastened in those who survive. The same statement must be made, in identical terms, in regard to Bright's disease.

The occurrence of severe gastro-intestinal catarrh as a complication, with vomiting and purging, has already been mentioned, together with the much more rare hæmorrhage from the stomach or bowel. After the subsidence of the acute symptoms a chronic gastro-intestinal catarrh is apt to persist, with grave impairment of nutrition, and to prove rebellious to treatment.

Cerebro-spinal meningitis undoubtedly occurs as an occasional complication. In some instances the onset is indistinguishable from cerebro-spinal fever save

FIG. 12.

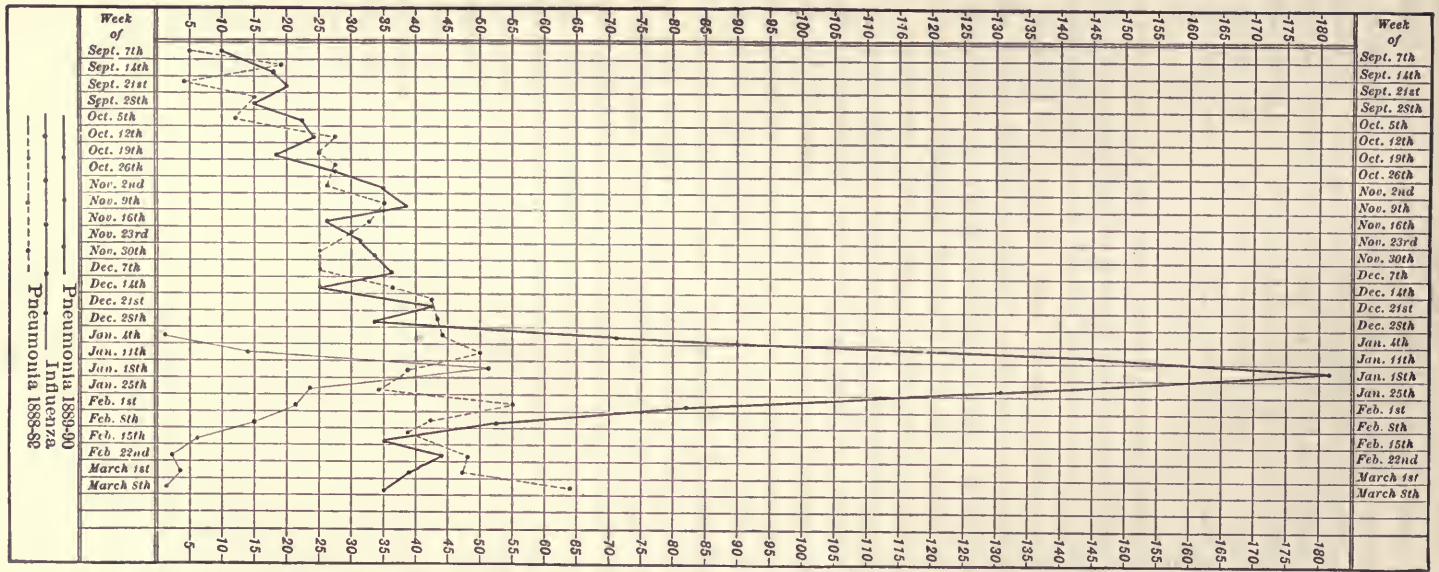


Chart showing the Mortality from Pneumonia in Philadelphia during the Winters of 1888-89 and 1889-90, and from Influenza during the Winter of 1889-90.

by the absence of petechial eruption; and it is important to bear in mind that not a few epidemics of influenza have been regarded as of that nature by experienced observers. The cases either run an acute course with intense headache, delirium, stupor, general convulsions, extreme retraction of the head, and terminate fatally, or the symptoms are less violent and the case goes on to gradual recovery or to the development of all the symptoms of exudation with fatal pressure. Several cases of abscess of the brain were reported in the late epidemic by Bristowe, and one case occurred in the P. R. R. series.

Persistent headache, insomnia, and neuralgia are common sequels. Otitis media was reported in a number of cases, and affection of the optic nerve likewise. Melancholia, impairment of mental power, and even mania are among the sequels. In no less than 18 cases in the P. R. R. series did it become necessary to confine the patients to insane asylums: 5 of these cases have proved fatal. The dejection of spirits which often attends convalescence is remarkable, and has not failed to attract attention in many epidemics.

Perineuritis is one of the most frequent complications and sequels. Much of the suffering in the disease may be referred to this cause. The nerves of special sense may be involved, and it is not impossible that implication of branches of the pneumogastric may explain some of the grave pulmonary and gastric conditions which arise. Persistent peripheral neuritis, muscular atrophy, and partial palsies have been exceptionally frequent since the last widespread outbreak of influenza.

Enlargement of the lymph-glands, and especially those of the cervical chains, is not infrequent in some epidemics as a sequel. It may prove persistent and troublesome, and occasionally ends in suppuration.

As already stated, an attack of influenza affords but little protection against subsequent attacks. Several well-marked attacks have been suffered by many individuals during the past two years. Relapses also are not infrequent. In the 6680 cases of the P. R. R. series they occurred in 762 instances, or in over 11 per cent.

Diagnosis.—During an epidemic of influenza the entire mortality of the community is greatly increased. At the height of the recent epidemic the number of deaths in many cities was quite double that of the corresponding period of preceding years. In Philadelphia the total mortality for the month of January, 1889, was 1862, and for December, 1889, was 1488; in January, 1890, the epidemic of influenza was at its height, and the mortality rose at a bound to 3044, of which number only 116 were reported as influenza. It is evident that this sudden increase in the number of deaths is due in large part to the serious mortality of influenza itself when complicated, as with bronchitis or pneumonia. But, in addition, many chronic affections, such as Bright's disease, phthisis, heart disease, are awakened to rapidly fatal activity by the influence of the grippal poison. While, therefore, there is a tendency during the prevalence of all epidemic diseases to err in regarding almost every case of illness as belonging to the prevailing malady, it is doubtful whether,

in severe outbreaks of influenza, the extraordinary diffusion of the infection is even sufficiently appreciated. On the other hand, it is no less true that sporadic cases of influenza are liable to have their true nature overlooked, and to be regarded as idiopathic catarrhal fever or to be confounded with other infectious diseases. Not until the bacteriological diagnosis of influenza comes to be widely practised will the protean manifestations of this disease be fully determined.

Cases of the ordinary thoracic type should be readily recognized by the sudden onset, the absence of the usual causes of bronchitis, the character of the cough, the pains in the head, back, and limbs, the prostration and sense of illness out of seeming proportion with the degree of fever or the physical signs of pulmonary trouble.

The gastro-intestinal type is more apt to be mistaken either for acute catarrhal gastritis, for simple continued fever, or even for typhoid fever. The absence of the usual causes, the greater degree of prostration, and the characteristic pains distinguish it from the first, while typhoid fever is known by the more slow onset and gradual development, by the greater enlargement of the spleen, the appearence of the stools, and the characteristic eruption, although in influenza abdominal distension, diarrhœa, epistaxis, bronchial catarrh, fever, headache, and delirium may occur. During an epidemic of influenza many cases which are regarded as ephemeral or simple continued fever are doubtless grippal in nature.

Cases complicated with the early development of pneumonia are peculiarly liable to have their true character overlooked.

Allusion has been made in the article on Cerebro-spinal Fever to the resemblance between that disease and the cerebro-spinal type of influenza. It appears that from the earliest period these two diseases have often prevailed coincidently or in close sequence. When the meningitic symptoms ensue in a case which has begun as of the catarrhal type, there is less danger of overlooking their grippal nature. But when, as happens with considerable frequency during certain epidemics, patients are seized with intense pain in the head, back, and limbs, slight fever, rapidly developing delirium and stupor, muscular rigidity, and possibly retraction of the head, or even general convulsions, herpetic and possibly petechial eruption, and when at the autopsy the lesions of cerebro-spinal meningitis are discovered, it is evident that careful bacteriological work is needed to decide as to the exact character of the infection. The cases recorded by H. B. Allyn in 1892 are extremely interesting as illustrations of the above.

In the article on DENGUE reference is made to the diagnosis of this peculiar epidemic disease from influenza, with which it has points of strong analogy.

Prognosis; Mortality.—Apart from serious complications, the mortality of the ordinary catarrhal types of influenza is extremely small, certainly less than ½ of 1 per cent. In 6680 carefully observed cases in the P. R. R. series the total mortality was 94, or about 1.4 per cent, a little less than one-

third of which was from uncomplicated grippe. The number of deaths in the 35,413 cases of influenza collected by myself was 257, or 0.72 per cent., and of these 84, or about ¼ of 1 per cent. of the whole number, were from uncomplicated grippe.

The liability to severe complications is, however, so distinctive a feature of many epidemics of influenza that the prognosis should never be regarded as trifling. The development of intense bronchitis, pneumonia, nephritis, or meningitis at once renders the case very dangerous. In some epidemics the mortality is very large, owing especially to the extreme prevalence of pneumonia or of meningitis.

The prognosis is much more serious in the aged or in persons of infirm health. When patients with phthisis, paralysis, heart disease, or organic kidney disease are attacked with influenza, the danger is always considerable. If they recover from the attack, there is great reason to dread subsequent aggravation of their organic trouble.

Treatment.—No reliable means of prevention are known. Althaus urges wholesale protective revaccination of the population with animal lymph. The evidence on which this startling proposition is based seems wholly inadequate. One of the worst cases of influenza I ever saw followed by recovery occurred in a child nine months of age, who had just passed through a typically successful vaccination with animal lymph. Fatigue and excesses of all kinds, and especially in venery, predispose to the contraction of influenza. Any exposure which induces catarrh during the prevalence of influenza almost ensures the reception of the infection. Great care in clothing, in the avoidance of damp, of draughts, and of any sudden check of perspiration, should be observed during the epidemic by all persons, and especially by those of feeble vitality or who are affected by any organic disease.

Rest in bed must be insisted on from the onset until convalescence is established. Most of the mortality may be traced to a neglect of this cardinal rule. Unusual care should be used to avoid draughts or sudden changes of temperature in the sick-room. In addition to the avoidance of complications by strict nursing, the indications are to support the system, to relieve suffering, to secure sleep, to allay cough, and to control fever.

Purgatives should be avoided. If the tongue is heavily coated, the stomach embarrassed, and the bowels constipated, a few fractional doses of calomel or of a mild saline may be given; but, as a rule, any laxative effect is best secured by enema or suppository. The diet may be as supporting as the digestion will admit. It is not well to restrict the patient to liquids save in cases of the gastro-intestinal type.

The fever is not often high enough to demand vigorous antipyretic treatment. Hydrotherapy should be used with extreme caution, on account of the wholly exceptional tendency to catarrhal and inflammatory complications. Phenacetin in moderate doses, gr. iij to v, repeated two or three times in twenty-four hours, reduces temperature, relieves suffering, and tends to secure sleep. It is less likely to cause undue depression and relaxation of the system

than antipyrine, though all drugs of this class must be used with great caution in influenza. Small doses of antipyrine, gr. iij, combined with quinine or sodium salicylate, may also be given two or three times in twenty-four hours, but require watching as to their effect. Quinine in tonic doses is indeed indicated in most cases. It may be combined advantageously with opium, and the following formula is often useful as meeting several indications:

℞. Quininæ sulph.,	ʒj;
Pulv. digitalis,	gr. xx;
Pulv. scillæ,	gr. xx;
Ext. opii,	gr. v;
Ext. glycyrrhizæ,	q. s.

Misce et ft. pil. xxx.

Sig. A pill three or four times daily.

The condition of the stomach demands careful attention: if there be marked irritability, the diet must be restricted rigidly, and no remedies given internally save those which tend to soothe it, such as small doses of the tincture of aconite-root as a febrifuge, and bismuth subnitrate or cerium oxalate or silver nitrate, with minute doses of cocaine, for local action on the mucous membrane.

The severe headache may be, especially if associated with high temperature, treated by cold applications to the head and small doses of phenacetin. But it is so often accompanied by insomnia that codeine, or even morphine, may be required. Sulphonal may prove adequate to afford sleep when pain is not prominent.

The pulmonary complications require prompt attention. Mild counter-irritation should be used, and the chest be enveloped in a raw-cotton jacket. Most expectorant remedies are contraindicated by their tendency to disorder the stomach or to relax and depress the system. Upon the whole, strychnine in full doses is the most important remedy against these complications—a fact which supports the view that depressed pneumogastric power has much to do with their production. The following is often valuable:

℞. Morphinæ sulph.,	gr. j;
Quininæ sulph.,	gr. xxxvj;
Strychninæ sulph.,	gr. ss;
Acid. phosphoric. diluti,	fʒiij;
Glycerinæ,	fʒv;
Aquæ,	q. s. ad fʒiij.—M.

Sig. A teaspoonful in water from three to six times daily.

Or in very severe cases with tendency to cardiac and respiratory failure hypodermic injections of small doses of morphine, gr. $\frac{1}{12}$, with large doses of strychnine, gr. $\frac{1}{24}$, may be used several times daily.

Aromatic spirit of ammonia, or carbonate of ammonium, or oil of turpentine in emulsion if acceptable to the stomach, may be given internally. A certain measure of relief to the severe pains in the back, chest, and limbs is also afforded by external applications, such as St. John Long's liniment, or compound chloroform liniment containing aconite and ammonia, or veratria ointment, or the following applied over areas of persistent pain:

℞. Tr. aconiti radicis,
Chloroformi, āā. fʒj;
Tr. iodini, fʒvj.—M.
Sig. Apply locally.

The only condition in which depletion is to be thought of in influenza is when severe cerebro-spinal symptoms appear abruptly. In vigorous adults I have then used moderate venesection with immediate and lasting advantage. The fear that meningitis will be established is the controlling motive. If this measure seems too severe, local depletion by wet or dry cups or leeches may be substituted. If the symptoms are less urgent, cold applications to the head, hot footbaths, or sinapisms to the back and limbs may suffice in connection with suitable internal remedies.

Convalescence demands the closest supervision. All details of personal hygiene must be insisted upon. Nutritious diet, avoidance of bodily or mental exertion, careful avoidance of undue exposure, the continued use of tonic remedies, and, if possible, suitable change of air, will promote more full and rapid return to health. The depressing and enfeebling influence of the disease will, however, be often found to be strangely persistent. It is of especial importance that those who are still suffering from this depression should not be exposed to the danger of reinfection, as fresh attacks contracted under such circumstances are apt to prove very serious.

DENGUE.

By WILLIAM PEPPER.

Definition.—Dengue is an acute specific disease, occurring in epidemics which are chiefly confined to tropical and subtropical latitudes, without definite anatomical lesions, and characterized clinically by two paroxysms of fever with a marked remission, severe pains in the muscles and joints, anomalous eruptions, and a very low rate of mortality.

Synonyms.—The number and grotesque variety of the names applied to this disease attest its peculiar and variable character. Many refer to the pains, as breakbone and eruptive rheumatic fever; others to the eruptions, which are prominent in some epidemics; others, again, are popular terms descriptive of special symptoms. The name *dengue*, equivalent to "coquettish" in Spanish, seems to have been given on account of the stiff, affected gait of those recovering from the disease.

Etiology.—Dengue is essentially a disease of warm latitudes. It was first recognized in 1779 in Java by David Brylon, who called it articular fever. The earliest accurate accounts are of epidemics in India in 1824 and later. According to Matas, the conclusion of epidemiologists is that it was originally an Asiatic tropical infection, starting perhaps in India. It is known to prevail sporadically there, and also in Africa, and probably also in our Gulf States. Of European countries Spain alone has suffered. Frequent epidemics have occurred in South American countries, in the West Indies, and in the Southern United States. Rare outbreaks have been noted in Philadelphia, New York, and even in Boston. The usual limits of its epidemics are 32° N. and 22° S. lat. The summer seasons appear to favor its occurrence. Neither race, age, sex, nor social condition exerts any influence.

There can be little doubt as to its contagiousness. Its epidemics spread with extreme rapidity along the routes of travel. It is probable the poison may be carried by fomites. McLaughlin of Texas has found in the blood of patients with dengue a micrococcus which he believes to be characteristic of the disease, but Osler holds it to be still *sub judice*.

The susceptibility to the infection of dengue is almost universal. In the great Texas epidemic of 1885, McLaughlin estimates that in the city of Austin alone, out of a population of 22,000, no less than 16,000 persons were attacked in the course of a few months. The occurrence of an analogous disease among domestic animals simultaneously with the prevalence of dengue has been noted several times.

The frequency with which relapses and recurrent attacks occur would seem

to differ in different epidemics; but on the whole they are much more frequent than in most infectious diseases. In some epidemics relapses have occurred as often as in 15 per cent. of all cases. It has even been claimed by a few that an attack of the disease predisposes to a subsequent attack.

Morbid Anatomy.—There are no lesions known to pertain to the disease itself. Death scarcely ever occurs, except from complications, and even then is of extreme rarity.

Symptomatology.—The onset of dengue is usually abrupt, after a period of incubation of about four days' duration, and without prodromes. There is a chill, which in young children may be replaced by a convulsion. The temperature rises quite rapidly, and at the close of the first or second day usually reaches its height, from 102° to 106° F., according to the severity of the attack. Intense headache, backache, and pain in the joints ensue quickly. The muscles also are painful and sore. There is a sense of extreme depression and prostration. Delirium and hebetude are slight, if at all present, and the patients are usually keenly conscious of their intolerable sufferings. The pulse and respiration are rapid. The tongue is moist and yellow-coated; appetite is lost; nausea is moderate; the bowels are quiet. The urine is febrile, but scarcely ever albuminous. A transitory erythematous rash appears in a varying proportion of cases in different epidemics. The joints are often painful, stiffened, and even red and swollen.

In some cases the symptoms assume a marked gastro-intestinal type and severe vomiting and purging occur. More rarely cerebral symptoms may be prominent, and increasing stupor and evidences of meningitis ensue. Doubtless these nervous symptoms are often due to hyperpyrexia: Holliday reports a fatal case in which the temperature rose to 109½° F. Epistaxis may occur, and so may hæmorrhage from the stomach or bowels.

The primary febrile paroxysm lasts from three to five days, and is terminated by a critical fall with sweating or diarrhœa. The temperature may become subnormal and the pulse abnormally slow, but more commonly it is only a remission. The symptoms are greatly relieved, but the patient still feels stiff and weak. At this time or with the return of fever, which occurs after an interval of two or three days, a second eruption appears, with varying frequency in different epidemics. It is not characteristic, but may resemble urticaria, herpes, lichen, or erythema. The second paroxysm of fever is usually mild and short. The pains, restlessness, and anorexia return. Defervescence occurs again after two or three days, and subnormal temperature and pulse are noted not rarely. The eruptions fade rapidly, and desquamation commonly follows.

The entire duration of an ordinary case is from seven to nine days. Convalescence may be prompt, but is apt to be slow and protracted, and to be attended with a singular degree of mental depression and loss of energy or actual debility.

As already stated, the disease terminates habitually in recovery in spite of the painful or alarming symptoms.

Complications and Sequelæ.—The occasional occurrence of severe nervous symptoms has been mentioned. The character and favorable course of these indicate their dependence on the high fever as a rule. Any lesion, such as meningitis, which is mentioned by some observers, must be extremely rare. Insomnia may persist for some time after the disease, but more commonly the only nervous sequels are neuralgic pains and marked prostration and depression of spirits, which may last for weeks, especially among the weak and infirm. Rush states in his report of an epidemic of dengue in Philadelphia in 1780 that a young lady remarked it might be called *break-heart* instead of *break-bone* fever; and this remark might be applied with equal fitness to influenza. Severe catarrhal inflammation of the respiratory or gastro-intestinal mucous membranes, catarrhal jaundice, or hæmorrhages may occur occasionally. Enlargement of the lymphatic glands in various parts of the body is not rare, and may prove obstinate: in some epidemics crops of furuncles and superficial abscesses have been noted.

Diagnosis.—Sporadic cases of dengue may readily be mistaken for mild rheumatic fever, but the presence of eruptions, the absence of acid sweats, the peculiar course of the fever, and the absence of cardiac complication serve to distinguish it. The disease to which it presents most resemblance, both in its sporadic and epidemic forms, would seem to be influenza. The accounts of some epidemics of dengue leave doubt as to whether they may not have been outbreaks of mild influenza in a warm latitude. The diseases resemble each other in the rapidity of development in great communities and over large areas; in the large proportion of the population affected; in the frequency of relapses and the liability to successive attacks; in the disproportion between the apparent gravity of the symptoms and the very small mortality of the uncomplicated disease; in the sudden onset, the peculiar severity and character of the pains, the great mental and bodily prostration. But in influenza the only eruption frequently present is herpes; there is no affection of the joints (although Matas states that in dengue also true evidences of arthritis, such as redness and swelling, are exceptional); the remission and recurrence in the course of the fever are not present; there is a far greater liability to serious complications; and the disease is wholly independent of geographical restrictions. Bacteriological research must, however, complete the separation of the two diseases.

When yellow fever and dengue prevail simultaneously, care is required to avoid confounding the latter with mild and imperfect cases of the more grave malady. The initial eruption may lead dengue to be mistaken also for some one of the eruptive fevers.

The **prognosis** is, as already stated, almost invariably favorable. In American epidemics it has been rare for even a single death to occur. Matas quotes a statement that in Madras dengue was sometimes fatal in adults from pericarditis and in children from convulsions, 20 deaths occurring out of 3647 cases collected by one observer.

Treatment.—The uniformly favorable and self-limited course of dengue

calls for merely symptomatic treatment. Strict rest in bed should be insisted upon, and rigid attention should be paid to all details of nursing and hygiene till convalescence is established. A mild laxative may be administered at the outset.

The fever may be high enough to call for small doses of phenacetin or antipyrine, which should also be very useful in allaying the peculiar arthritic and myalgic pains. Quinine or salicylate of sodium may also be used. Hydrotherapy is rarely indicated, but should be used if the pyrexia is high and accompanied with severe nervous symptoms. The bromides and codeine or morphine are often required to secure sleep and relieve suffering.

Convalescence demands a continuance of careful regulation of diet and hygiene. Tonics and nutrients should be given to improve appetite and overcome the persistent debility. If myalgic or arthritic pains continue, potassium iodide or sodium salicylate will be found useful. A change of residence may be required to promote complete restoration of health.

MILIARY FEVER.

By WILLIAM PEPPER.

Definition.—An acute, infectious, and at times epidemic disease, characterized by a sudden onset, with profuse sweating, sense of oppression in the epigastric region, and the appearance of a papulo-vesicular exanthem.

Synonyms.—Sweating sickness; Schweissfriesel; Suette miliare; Suette des Picards; Sudor anglicus.

History.—In August, 1486, there broke out in the army of Henry VII., after its return from the battle of Bosworth Field, an acute, virulent, infectious, and highly dangerous malady that soon spread throughout England and raged until November of the same year. In 1507 the disease again became epidemic, but was confined to England. In May, 1529, London was again attacked, but on this occasion the continent of Europe was also affected. In 1551 another epidemic occurred in England, being confined by the boundaries of that country. In 1718 a disease resembling in most of its essential features these earlier epidemics appeared in Picardy, and from that point spread to other parts of France. Hirsch has tabulated 194 epidemics of this "miliary fever" that occurred between the years 1718 and 1874. In various parts of Italy and Germany also small epidemics have been described. In 1887 there was quite a severe epidemic in France. The disease is practically never seen now save in the north-eastern provinces of France and in a small portion of Italy.

Etiology.—Regarding the auxiliary causes of the earlier epidemics of "sweating sickness" but little is known. There are, however, a few facts in relation to the later outbreaks of what has been most generally called "miliary fever" that show, at least in the majority of epidemics, some uniformity. Most of the epidemics have occurred in spring and summer; the disease usually is most prevalent in low-lying and damp areas, but has been observed at higher elevations with a porous, dry soil; women are more subject to attack than are men, and the middle period of life furnishes the greatest number of cases; social condition as to habits of life influence the liability to the disease to no appreciable extent. The disease is not contagious, and is not particularly prevalent in institutions, barracks, etc., where people are congregated in large numbers. No immediate exciting cause has ever been discovered.

A curious relation between epidemics of this disease and of Asiatic cholera

has been noted, the diseases interchangeably following each other or the two diseases occurring at the same time.

Symptomatology.—After a brief prodromal period of malaise, weakness, and headache the patient is attacked, usually in the night, with profuse sweating, fever, and a sense of oppression referred to the epigastric region. The pulse is rapid, there is elevation of temperature that is rarely excessive, and there is found to be marked tenderness over the upper portion of the abdomen. After a period of three or four days, in some cases later, there appears a characteristic eruption, with temporary increase in all of the symptoms previously observed. The exanthem consists of small reddish spots of irregular contour, but of a generally round form, varying in diameter from $\frac{1}{16}$ to $\frac{3}{16}$ of an inch. The lesions are either closely aggregated or confluent. In the centre of each spot there appears, after a few hours, a small vesicle, which gradually enlarges to the size of a millet-seed or pea. The clear contents of the vesicles soon become opaque, owing to a purulent transformation, and then after two or three days dry into crusts which are cast off as scales.

While the skin is the usual site of the eruption, it may also be found upon the nasal, oral, and conjunctival mucous membrane. The first appearance of the eruption is generally upon the neck and chest; after which it is seen upon the back and extremities. More rarely the scalp and abdomen may be the seat of the exanthem.

From mild cases, wherein almost the only prominent symptom is the occurrence of frequent and profuse sweatings, the disease varies in severity to fatal cases with most severe symptoms and intense anguish. In addition to the symptoms that have been mentioned there may be insomnia, vertigo, cephalalgia, complete anorexia, thirst, nausea, and marked constipation. The sense of oppression at the epigastrium may in grave cases become so intense that the patient tosses from side to side, clutching at the bed-clothes in order to obtain relief; indeed, death from apnœa has been stated to occur. During the sweating stage convulsions may occur, sudden and fatal collapse may end the scene abruptly, or the patient may fall into a typhoid condition. Convalescence is prolonged.

Morbid Anatomy.—No characteristic lesions are found. Decomposition is said to occur rapidly, and the blood is thin and dark in color. In some cases œdema of the meninges of the brain has been found. In most cases the lungs are found to be congested and the heart soft; the pericardium the seat of ecchymoses; the mucous membrane of the alimentary tract congested; the liver full of blood; the spleen enlarged, soft, and friable. Some observers claim that they have found upon the mucous membrane of the intestine vesicles similar to those upon the skin.

Diagnosis.—In time of epidemics this should present no special difficulty. The only diseases with which miliary fever would be apt to be confounded are acute rheumatic fever, measles, and malarial infection. The absence of localized articular pains, the peculiar sense of oppression in the epigastric region, and the appearance of the eruption distinguish it from the first of these. In

measles the prodromal catarrhal symptoms, the absence of vesiculation in the centre of the eruption, and the distribution of the latter would prevent a mistake in diagnosis, even without the absence of profuse sweating, From intermittent fever this disease would be distinguished by the absence of marked rigor, the lack of periodicity, and the failure to respond to specific antimalarial treatment, while the discovery of Laveran's micro-organisms in the blood would positively announce the presence of malaria.

Prognosis.—Different epidemics vary so much in their extent and severity, and individual cases in an epidemic present such wide variations in the intensity of their symptoms, that the outlook in each case must be judged upon its own merits. In some epidemics of considerable extent the mortality has been *nil*, while in others it has reached as high as 50 per cent., or even 80 per cent., of those attacked. The greatest mortality occurs during the sweating stage. Epidemics vary much in their duration, the usual time of prevalence being from one to four weeks.

Treatment.—At one time attempts were made to abort or stay the severity of the disease by covering the patient warmly and administering diaphoretic remedies. This practice was very justly abandoned, and it would seem that the expectant plan of treatment is the best. The diet should be light, easily digestible, and nutritious. The patient should be lightly covered, and cool acidulated drinks may be permitted. Quinine in moderate doses has seemed to have some beneficial influence. Stimulants should be given in accordance with the condition of the patient; they are not required in the milder cases. After recovery tonics are needed to restore the patient to his former condition.

MILK-SICKNESS.

By WILLIAM PEPPER.

Definition.—An acute disease occurring in the sparsely-settled and uncultivated regions of the United States, primarily affecting cattle, but also attacking human beings as the result of eating the flesh or drinking the milk of animals so affected; characterized by great weakness, marked constipation, vomiting, fœtor of breath, and twitching of muscles.

Synonyms.—Trembles; Slows; Puking fever; Sick stomach.

During the early settlement of various portions of the central region of the United States this disease was very prevalent and of great virulence. As civilization advanced and the land became more highly cultivated, it gradually disappeared from regions where it formerly abounded, until at the present time it is limited to a few localities of small area in but a few of the States lying west of the Alleghany Mountains.

Etiology.—Several theories have been advanced in the attempt to arrive at the cause of this affection, but as yet none has been found that furnishes a satisfactory explanation of its mode of production. It most commonly occurs in summer and autumn, and is more prevalent in years of drought and in hot and dry weather. The three theories that have been most strenuously advocated as explaining its causation are—(1) that the poisonous principle is furnished by some variety of rhus; (2) that it is due to a mineral poison contained in the drinking-water of cattle in the area affected; and (3) that it is produced by a miasm. Regarding the first of these theories, which is much more rational than the others, it may be said that the point has not been proven, and that the history of its propagation and transmission from animals to man and from diseased animals to healthy animals through the ingestion of the flesh or milk of the former would point toward some poison capable of increase in the animal body, rather than toward one that attained its highest development while existing in its natural condition as a plant. The second theory is untenable from the well-established fact that laborers who have drunk of the same water as animals that became diseased failed to be affected, although the consuming of the flesh or milk from such animals produced the disease in man. Against the third theory all that need be said is that the fencing in of limited areas of a farm may cause the total cessation of the appearance of the disease—a measure that would have no effect were the disease propagated by a miasm.

Symptomatology.—The symptoms in animals occur at times with great

abruptness; at other times the onset of the disease is slow. The former manner of attack is most frequently observed after the animal has undergone, or while it is undergoing, some unusual amount of physical exertion. The chief and characteristic symptoms are—marked muscular weakness with tremor upon motion, inability to stand, at times vomiting, and a peculiar fœtor of the breath. The tremor may amount to a positive convulsion; the inability to stand may develop so suddenly that the animal drops during or after exertion, and lies trembling in every muscle and tossing the head from side to side. In man the disease is ushered in by chilly feelings with hot flushes, by pain in the head and limbs, with great muscular debility. With this weakness there is marked unsteadiness in performing muscular acts or even tremor of the muscles at rest. There are also developed various disorders of the digestive tract. There is marked fœtor of the breath, the odor being described as characteristic and being likened to various odors or combinations of odors of familiar objects, being most frequently said to resemble that of chloroform and the odor produced by mercurial salivation. The tongue is coated and marked by the teeth; later, dry, fissured, and swollen. Vomiting is a frequent symptom, the vomited matters consisting first of the food last ingested, later of a peculiar "soapy" material of a yellowish or greenish hue, or it may consist of mucus stained with blood or of a material resembling coffee-grounds. With these symptoms there is associated marked tenderness in the epigastric region, with a sense of oppression. The bowels are obstinately constipated. The pulse is at first full, but later becomes small and rapid. The temperature is, as a rule, elevated somewhat, save in the cases of sudden and violent onset, when it may be subnormal. The skin of the trunk may feel hot to the touch, while the extremities are cool. Respiration is frequently much embarrassed in the severe cases.

Prior to death the patient may pass into a typhoidal condition, while delirium, hiccough, and coma frequently appear. The patient may be violently attacked and die within a few hours; usually, however, the disease continues for from three to five days before either death comes or convalescence begins. The latter is usually announced by a copious evacuation of the bowels, and is apt to be very protracted.

Morbid Anatomy.—But few opportunities for necropsies have been afforded, but in those made the appearances found coincide closely with those noted by Graff in animals killed experimentally by the ingestion of flesh from diseased cattle. These pathological findings are, in brief, as follows: Cerebral sinuses much distended with blood; marked congestion of cerebral and spinal meningeal vessels; pia mater opaque and covered with purulent exudate; brain soft; stomach and small intestines contracted and their mucous membrane injected; liver and spleen soft, the latter being enlarged to, in many cases, twice its normal size; liver, spleen, lungs, and kidneys full of blood; blood fluid. The above conditions point clearly to an infectious process. The occurrence of meningitis is of special importance, and suggests possible analogies with irregular cerebro-spinal fever.

Diagnosis and Relation to Other Diseases.—In those regions where the disease is known to occur the diagnosis is readily made by noting the presence of the characteristic tremor, the peculiar odor of the breath, and the obstinately constipated condition of the bowels. The affections with which the disease is most apt to be confounded are those resulting from poisoning by corrosive mineral substances. From these it may be distinguished by the tremor and by the absence of diarrhœa. From poisoning by animal toxines the diagnosis would be more difficult; and, in fact, this disease itself may in the future be found to be due to the ingestion of ptomaïnes. The absence of diarrhœa would separate this from most of the known forms of animal poisoning.

Treatment.—Formerly, when the disease was much more prevalent than at present, much dependence was placed upon the employment of venesection and calomel in the treatment of this as of every other affection. It was soon found, however, that not only did these measures fail to produce a cure, but that they frequently tended to increase the severity of the symptoms. The most rational treatment is symptomatic and expectant. The external application of counter-irritants to the epigastrium, with the internal administration of antiemetics, such as carbolic or dilute hydrocyanic acid, and the use of appropriate quantities of alcohol or other cardiac stimulant, would seem to fulfil the indications. Large enemata of water or oil have been used with asserted good results. Prophylactic measures are to be rigorously employed. Tracts of land where affected cattle have been grazing should be fenced off in order to prevent the access of other cattle. All animals found to be affected by the disease should be killed and the carcasses buried at once. Where the disease is suspected to be present, though latent, forced exertion may cause the characteristic symptoms of the disease to exhibit themselves.

MOUNTAIN FEVER.

By WILLIAM PEPPER.

Although there are in medical literature accounts of various anomalous fevers to which special names have been applied, it seems desirable to allude here only to "mountain fever," by which name has been designated a mild type of infectious fever observed in the Rocky Mountain region of the United States. It must not be confounded with the acute indisposition which not rarely develops soon after ascent to a high altitude. The symptoms of this are properly referred to the action of the rarefied air upon the circulation and respiration. They are exhaustion on exertion, headache, giddiness, sometimes nausea and vomiting, marked dyspnœa, and undue rapidity of the pulse. Epistaxis often occurs, and the temperature may be found slightly elevated. The symptoms gradually subside as the subject becomes habituated to the altitude. Such results are observed in all countries, and are developed at different altitudes in accordance with the cardiac and nervous force of the individual.

But the group of cases to which the term "mountain fever" has usually been applied present the symptoms of an irregular continued fever. The duration is from two to four weeks or longer; the fever is moderate, the temperature ranging between 101.5° and 103°, and rarely passing 104° F. The marked irregularities in the fever and the apparent efficiency of quinine in certain cases have led some observers to believe in the malarious nature of the affection. Smart in particular advocated this view in 1878, and traced the origin to a water-supply tainted with deleterious vegetable matters which had been carried by the winds to the snow on the upper levels. Cerebral symptoms are not marked. The bowels are usually constipated, rarely relaxed. Rigor, lassitude, occasional epistaxis, have been noted in the early days. No characteristic eruption appears. The spleen is usually enlarged. Pulmonary complications occasionally occur. Curtin refers to four cases called mountain fever in which croupous pneumonia existed as a complication, if it did not constitute the entire disease. Few cases have died, and but two post-mortem examinations are recorded. In both the typical lesions of typhoid fever were present, and perforation of the ileum had occurred in one case. There would seem to be little doubt that such is the true nature of nearly all cases of this form of mountain fever. It is fair to assume that the effect of the high altitude would modify somewhat the symptoms of the disease. It may be admitted that sometimes a malarial element is associated, and that in rare instances the affection may be simply a malarial remittent fever. It is clear, therefore, that no adequate reason exists to longer continue a separate description of a disease which cannot be shown to possess distinctive features.

SCARLATINA.

By JAMES T. WHITTAKER.

Synonyms.—Rubores; Purpura (Forestus); Rossalia (Ingrassias); Scarlet fever; Ger. Scharlach.

Definition.—Scarlatina, from the (old) Italian scarlattina, scarlatto (red), is a treacherous, acute, contagious infection, characterized by a more or less typical fever, inflammation of the throat, a diffuse scarlet exanthem, followed by membranous exfoliation of the skin, occasionally by otitis, exceptionally by arthritis, and not infrequently by nephritis.

The first use of the term febris scarlatina is found in a comment by Lancelotti of Italy (1527), but it was not distinctly applied to the affection as we know it until by Sydenham (1661), who first separated it from measles, with which it had hitherto been confounded. Sydenham saw only mild cases. He considered the disease "only an ailment, we can hardly call it more," but was able to recognize it without the help of throat symptoms, which he does not mention in his brief description.

It was, however, a full century after Sydenham before the ability to separate scarlatina became common property, and no sooner was it firmly set upon its tripod of symptoms, to wit, fever, exanthem, angina—that is, no sooner were the throat symptoms established as an integral factor of the disease—than it became confounded with diphtheria as much as it had ever been with measles. Irregular cases of either are not yet easily disentangled.

Whence it was originally imported or when it first appeared in Europe is unknown, but it was first recognized in England in 1661; Scotland in 1716; Germany and Italy in 1717; Denmark in 1740; North America, at Kingston and Boston, in 1735, New York and Philadelphia in 1746, Ohio and Kentucky in 1791, Toronto in 1843, New Orleans in 1847, California in 1851.

The disease is rare in Asia and Africa, and is said to be (Wernich, 1871) entirely unknown in Japan. Scarlatina is therefore much less widely disseminated than measles and small-pox, both of which have repeatedly ravaged Asia and Africa.

A pronounced peculiarity of scarlatina in distinction from measles and small-pox is the variation in the intensity of epidemics, which are sometimes so mild, as in the time of Sydenham, that the affection "*vix nomen morbi merebatur*" (scarcely deserved the name of a disease), and again virulent and malignant, more especially in villages and small towns, with a mortality as great as that of cholera and the plague. "*Malum hoc grave*," said Sennert almost simultaneously with Sydenham, "*periculosum et sæpe lethale est.*" Bretonneau never saw a single fatal case of scarlet fever for twenty-four years

(1799–1823) in all his practice, but in 1825 he encountered an epidemic so virulent as to cause him to entirely change his opinion regarding the benignity of the disease. From 1801–04 the epidemics of Dublin were marked by great malignancy, but the character of the disease changed in 1804, and for the next twenty-seven years it was mild and benign. "What was more natural," Graves exclaims, "than that the difference should have been ascribed to our improvements in treatment?" But in 1834–35 there was bitter disillusion. The disease reappeared in virulence and malignancy, and in total defiance of hitherto successful methods of treatment. Lewis Smith relates that a distinguished physician of New York treated more than fifty cases of scarlet fever in one of the hospitals without a single death: a few months later the type changed, and his own son died of the disease.

That this virulence is not due to the accumulation of susceptible material in long intervals of absence is proven by the experience of Köstlin of Stuttgart, who observed an epidemic in 1846 so mild as to be without a single death following an interval free of scarlatina for a period of sixteen years. Soil, season, or climate offers no explanation of this peculiarity; we remain as yet, in the language of Drake, "entirely ignorant of the causes or conditions which determine these remarkable diversities of phenomena and danger."

Thus it may be said that since small-pox has been shorn of its terrors scarlatina takes rank as the most dreaded of all the infections which now prevail. It is estimated to cause one-twenty-fifth to one-twentieth of the whole mortality in England and America. In the two years 1863 and 1864 scarlet fever alone took in England more than 60,000 lives. In 1869 the victims in London alone numbered 5803. Estimating prevalence from mortality, making no allowance for unrecognized mild cases, and granting each case but two weeks of contagiousness, it is claimed that London maintains a permanent stock of 2000 centres of contagion. Continuous or constant infection is sustained in nearly all cities of fifty to one hundred thousand inhabitants.

Etiology.—Susceptibility to scarlatina is much less than to measles and small-pox, one member of a large family being often alone attacked. Hence the majority of individuals escape it throughout life. Individual families seem predisposed to or exempt from the disease. Ziemssen says he saw cases which annihilated the posterity, and practitioners everywhere have become demoralized by the loss of one member after another of a family, to literally extinguish it. On the other hand, the family of the physician himself, exposed to frequent infection, may entirely escape attack. It is difficult to find any explanation for this individual or family immunity until at least the etiology of the disease shall have been definitely established. Geil makes an attempt at one with the assumption that the cause of the disease is received into the throat, and may take hold only upon a broken surface. The absent or more or less favorable nidus in the throat denies or admits the disease, fixes the period of incubation, the amount of the infection, the intensity of attack, etc. The explanation is seductive in its simplicity, and is based upon the infection of wounds and

puerperium, but is open to the valid objection that the necessity of a broken surface, as in the case of syphilis or hydrophobia, is not proven of scarlatina. The new doctrines of defensive serums will probably soon clear up this obscure field. This extensive immunity and individual liability accounts, however, for the fact that while epidemics of scarlatina are much less frequent than measles, decades often intervening, individual cases are much more common.

It is said that children have been born at various stages of the disease, but it must be remembered that hyperæmia and desquamation occur frequently in the new-born in health. The curious observation has been made of infants that, though lying constantly by the side of mothers affected with the disease, they escape almost without exception—proof that the exemption of sucklings is innate, and is not due to the greater protection of them from exposure. The age of predilection ranges from two to seven. Sixty per cent. of cases occur before the age of five, 90 per cent. under ten. Attacks later in life are rare, and are usually mild. Here too, however, are observed the same differences in epidemics. Thus, in Ziemssen's report the mortality among adults in 1865–75 was 11.5 per cent., and in 1876–87 but 1.3 per cent.

One attack confers immunity, as a rule, for life. With an observation of 2000 cases Willan never saw it repeat itself. A second attack is possible, but rare, and occurs more especially in cases of exposure in more advanced life in taking care of a younger member of a family affected with the disease. These second attacks are, as a rule, so abortive or rudimentary as to be easily overlooked, and are recognized at times, as are first attacks in the mildest cases, only by sequelæ. Hence the suspicion may be entertained that some of the insusceptibility of certain individuals may be immunity conferred by an attack so mild as to have been considered an ephemeral affection. Subsequent attacks may be accepted only when properly attested, distinctly marked, or attended or followed by recognized complication or sequel. Thus, Thompson saw a second attack followed by dropsy three years after a first which affected also other members of the family. Richardson declared that he had it three times himself, and Stiebel records a remarkable case in a woman aged fifty who suffered four attacks in four successive years—a frequency which must call out some doubt—with desquamation each time "in parchment-like pieces half a foot in length."

Most so-called repeated attacks (aside from relapses to be noticed later) are mistakes—erythema, rubella, septic rashes, etc.; and the rule that scarlatina occurs but once remains to constitute important evidence in diagnosis.

Pregnancy certainly protects against it, but the puerperium and open wounds of any kind invite it.

Regarding puerperal scarlatina, caution must be entered against confounding it with septicæmia, which often shows fever and eruptions (erythema) simulating scarlatina. There is no doubt, however, that the puerperal state confers additional susceptibility to scarlatina. Primiparæ are most liable to attack, but in all cases the disease sets in or shows itself close about the period of delivery, within the first week. Attack later is exceedingly rare. In proof

of the increased susceptibility of the puerperal state, Bonsall declared of his cases that 10 had had scarlatina before, and 1 had had it twice.

In puerperal cases the eruption occurs more quickly, almost suddenly. The throat symptoms are much milder or are absent altogether, while local lesions about the vulva and uterus predominate.

With reference to surgical scarlatina, so called, the same caution must be entertained to prevent confusion with erysipelatous, erythematous, or other eruptions of septicæmia. Many of the cases reported will not stand under close analysis. There is, however, good authority (Paget, Playfair) for the coincidence, and there is no reason why the possibility of it should be denied. In the light of existing knowledge no physician or surgeon in attendance upon a case of any infection may attend a case of labor without previous thorough disinfection.

The disease is conveyed by contact direct or indirect, as by clothing eminently, washing, bedding, furniture, letters, books (as from a library or school), toys, etc. A not infrequent source of infection is milk, sometimes from an infected dairy. Perhaps the most instructive example of this source of contagion was furnished by Miller, who reported 24 cases of infection in this way. The daughter of a dairyman near Brewster, New York, made a visit to the city. On the day after her arrival she fell ill with scarlet fever. Two weeks after her recovery she returned home. Two weeks later her youngest sister, who slept with her, showed signs of the disease. The health officer ordered that the dairy business should be conducted away from the house. Nevertheless, three weeks later a number of cases broke out in the village. By the fourth week 12 cases were reported. Investigations showed that every one who had the fever had drunk the milk, but not one who did not drink it was affected. The injunction of the health officer had been obeyed, but the milkman had washed and wiped his cans with white flannel clothes left in the barn by a peddler of rags, which were probably the cause of the first infection. In all, 24 cases developed directly from drinking the milk.

The disease may be conveyed also by third persons, who may carry the poison in their hands, hair, or clothing, but may themselves remain exempt. Convalescents from the disease carry it to school, church, theatre, train, etc., and disseminate it throughout a community.

The cause of the disease is said to be disseminated from the skin as well as the various secretions, and to be given off during incubation and desquamation, as well as during the stage of eruption. Upon this subject there is need of more exact information. Scarlatina is undoubtedly contagious in the strict sense of the term. Every case owes its origin to a previous case. The disease never originates *de novo*; but it is not yet determined in what way the poison is disseminated. It is believed that it circulates in the blood to contaminate and infect the various exhalations and excretions. It is probable that the disease is conveyed by exhalations, or rather excretions, from the throat, which are received by inhalation (contagium halituosum) into the throat, where it shows its first signs. Children have been born, as stated, in every stage of

the disease from incubation to desquamation. Infection in these last cases could have occurred only through the blood. More importance is to be attached to this fact than to any inoculations of lower animals, as in the well-known experiments of Coze and Feltz, because the symptoms which resulted were not typical or under comparison with so-called "control observations." They might have occurred with other inoculations. The attempts of Williams, Rostan, and Miquel at inoculation with a view of inducing a milder but protective attack for the most part failed, and contradictory testimony is furnished (Radel, Stoll) as to the propagation of the disease by means of the skin. It is assumed, rather than proven, that the poison of scarlatina exists in the skin. The claim is more definitely made of small-pox. It may be considered, in fact, established of this disease by inoculation. There are eminent clinicians (Volz) who deny all infection to the skin, or (notably Leyden) who maintain that the eruption of scarlatina is to be looked upon as only a reflex phenomenon, like that of erythema from gastric catarrh or like a toxic (drug) eruption, in which case it would be useless to look for the poison in the skin. At the present time, for the sake of safety, it is wise to believe in dissemination from the skin, and to act accordingly. It is singularly tenacious, adhering to clothing after months of disuse and to rooms after months of vacation and seemingly thorough disinfection and ventilation. Thus, Von Hildebrandt's coat retained its contagiousness for a year and a half. Adams reports that he found the disease to have been communicated by a convalescent who showed no signs of ill-health as late as the forty-third day. In the experience of the writer the opening of a closet in a house vacated for three months after the death of a child, and the handling of garments suspended in it, communicated the disease to another child of the same family.

Surgeon Brooke, U. S. A., detailed an instance of apparently spontaneous scarlatina in a child that had been subject to no discernible exposure. It was subsequently ascertained that one of the domestics had nursed a case of scarlet fever in a distant city a year before. Some of the clothing which she had worn at the time was packed away in a trunk, and this trunk had been opened, the contents removed and handled by the child a short time before the attack. The poison of scarlatina literally lurks in long-discarded clothes.

An illustrative case is also reported by Richardson of London. A family consisting of a man, his wife, and four children lived in a small thatched cottage. One of the children was attacked with scarlet fever and died. The remaining children were removed four or five miles. After several weeks one of them was allowed to return. This one took sick within twenty-four hours and quickly died. The cottage was now thoroughly cleansed and whitewashed, the floors scoured, and the wearing apparel destroyed. Four months later another of the children returned, to be stricken down with the disease in malignant type on the following day. The author believed that the poison had become fixed in the thatched roof, whence it could not be dislodged.

This tenacity of life is counteracted in great degree by limitation of range.

The poison of scarlatina is not widely disseminated. On the contrary, its area of distribution is confined to very narrow limits. The field of infection is pretty closely circumscribed about the body. The poison is entangled as a particulate body in the texture, or is fixed upon the surface of fomites. It is therefore much easier to sequestrate cases of scarlatina than measles, and thus to protect other members of a family or house. Confinement to a different story of a house or to a room absolutely isolated in its ins and outs—that is, in its exits as well as entrances of attendants and things—will generally suffice. Stay in an adjoining room with separate entrances, though with an unopened door between, has proven protective.

The tenacity of the poison—that is, the maintenance of the disease—is helped also by its intensity. But very short contact with a case suffices for infection. Thomas quotes from Palante the case of a mother in contact with a patient "but a moment," who returned immediately to her home at a distance of six miles, but whose contact had been long enough to collect and carry the disease to her children, in whom it showed itself in the course of a few days; and from Hennig the case of a child attacked four days after association "but for a short time" with another child which had had the disease six weeks before. The mere handling of woollen goods, clothes, shawls, blankets, curtains, furniture covers, etc. has repeatedly conveyed the disease.

Cold does not affect it. It is destroyed, however, by heat, by boiling water, especially by steam, and quickly by steam in motion, so-called "live steam." Henry, after subjecting the flannel garments of scarlatinous patients to a dry heat of 212° F., felt safe in having had them worn by unaffected children from six to thirteen years of age, and no infection followed.

Therefore the cause of scarlatina must be a micro-organism, though it has not yet been definitely isolated and determined. It has been described as a plasmodium, and even as a pilz (mould-fungus). Klebs (1880) pictured and described a structure found in the blood as the *Monas scarlatinosum*. Eklund (Stockholm, 1881), found constantly in the urine as well as in the soil and ground-water an immense number of discoid corpuscles without further proof of pathogenesis than presence. Power, Cameron, and Klein (1885–86) described coincidently with an outbreak of scarlatina a disease of cows in the Hendon (England) dairy, an acute general inflammation attended with the formation of pustules and ulcers on the bag, communicable to other animals, and from the pustules as well as from internal organs could be isolated and developed micrococci (streptococci) which when introduced into field-mice produced the same phenomena as injections from cultures from the blood of scarlet fever in man. Baumgarten believes these structures to be varieties of the streptococcus pyogenes. Marr (1891) contends that they correspond to Flügge's streptococci, and that the symptoms shown by animals, including the kidney affections, followed inoculation by other poisons.

Jamison and Eddington (1887) were able to isolate from the blood and from desquamations of the skin no less than eight different bacteria, one of which, designated the bacillus scarlatinæ, introduced into guinea-pigs and

rabbits, produced temporary fever and erythema. An inoculated calf died with fever on the following day, showing the same bacilli in the blood. The bacteria of mouse-septicæmia, rabbit-erysipelas, and the swine-plague will, however, all produce erythema, and often even desquamation.

These various micro-organisms are now believed to be varieties of the ordinary pyogenic bacteria. It cannot as yet be maintained of any of them that they are found uniformly or only in scarlatina, nor that the disease produced by them is really scarlatina. It is not yet established that any of the lower animals are susceptible to the disease. It would be more natural to look for the poison in the throat and in the blood in the earliest stage of the disease than in the secretions from the kidneys or in the substance or exfoliations of the skin. The kidney affection is doubtless the result of a chemical poison in its escape from the body, and the exanthem must also be regarded as toxic, like that produced by certain drugs.

Luff has succeeded in eliminating a hitherto unknown alkaloid from the urine of scarlatina, and Leyden declares it to be useless to look for the poison of scarlatina in the skin.

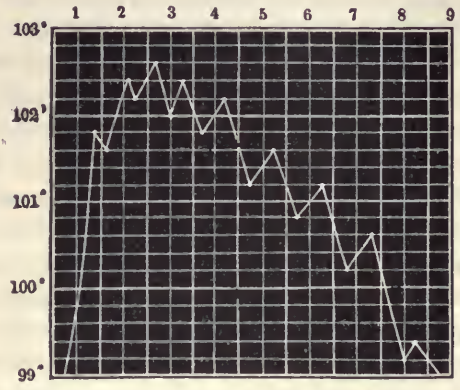
Regarding the relationship of diphtheria, it is admitted that one affection may follow the other, or that they may even coincide, but in all cases only as exceptions. The rule is that the diseases prevail in communities and exist in individuals independently of each other. Experimental evidence at the hands of the most competent and conservative observers multiplies to support this view, which was first clinically established by Henoch and Huebner. It is certain that most of the cases of so-called scarlatinal diphtheritis distinguish themselves by the absence of the Klebs-Löffler bacillus, and by the presence only of the streptococcus, which stands in some, though not specific, genetic relation to the development of the membrane. When inoculation is made early—*i. e.* so soon as the membrane is visible—on the very first day of its appearance, and when the matter is taken only from typical cases of scarlatina, as in the studies of Tangl, the culture shows in no cases the bacillus of diphtheria.

Course of the Disease.—*The period of incubation* is short, ranging from four to seven days. Ziemssen declares that the few unimpeachable observations that we possess put it at seven days. English writers make it generally less, and declare that from the second day after exposure liability of attack grows progressively less. In all the twenty-four cases mentioned in this article as having been caused by milk the symptoms showed themselves within twenty-four hours after the drinking of the milk. On the other hand, Pons extends the incubation to four, Moore to seven, and Veit to fourteen days. The most critical observers (Gerhardt, Thomas) admit these periods as exceptions, but place the general average at four to seven days.

The *invasion* is usually sudden and violent, grave, dangerous, sometimes fatal illness developing within a few hours. An initial chill or series of shiverings is attended by a quick and high rise of temperature. It is more frequently the case that the disease is ushered in without any chill at all.

A child presents evidence of attack in a shock manifested by extreme pallor and prostration. A highly susceptible, sensitive child may be seized with a convulsion. Usually the scene opens with vomiting. Early vomiting belongs to all the grave, acute infections, but it occurs with especial frequency in scarlatina, because of the gravity of the disease. After the vomiting it is noticed that the patient has fever. Inspection thus early reveals angina or the child complains at once of sore throat. Vomiting, sore throat, and fever at the start should excite the suspicion of scarlatina, or, in the presence of an epidemic or proximity of another case, establish the existence of the disease. The temperature distinguishes itself by the rapidity of its ascent (See Fig. 13).

FIG. 13.



Temperature-chart of a Mild Case of Scarlatina.

The ascent of the temperature in scarlet fever is more rapid than in almost any other disease, reaching often within twenty-four to forty-eight hours 104° to 107° F. *Calor mordax* was the term applied by the older pre-thermometric writers to express the biting heat of the skin. If there is no question of complications, the fever reaches its height with the appearance of the eruption, or at least with its full efflorescence, to gradually subside in the course of one or two weeks in a mild or sharp average case respectively. In cases in which the eruption "sinks in" or disappears in the face of, or is a result of, a grave complication, the coldness of the surface is only apparent. The thermometer in the rectum or vagina registers high grades (105° to 108° F.), to fall, often rapidly, under a hot bath, which may bring the blood, and with it the eruption, to the surface. The persistence of the complication, meningitis, pneumonia, etc., reproduces the surface coldness after the bath.

In correspondence with the height of this fever, especially in young children, nervous signs, as stated, show themselves—viz. delirium and convulsions. The delirium may deepen rapidly into coma. There is in every marked case profound prostration. It is seen on the most superficial inspection that the child is seriously ill. This stage of invasion lasts from one to two days. Comment has been made upon the irregularity or variety in severity of epidemics of scarlatina. The same variety is noticed in individual cases

of the same epidemic. In the same family side by side with a malignant case occurs an attack so mild that it may be scarcely recognized. Lightly-affected brothers and sisters may be playing about the house where one member lies fatally ill or has just fallen a victim to the disease. So there may be every grade of intensity in the onset, but as a rule the disease is announced by a sudden attack of fever attended by vomiting, which assumes more and more importance when it may not be accounted for by a sufficient provocation, as by indigestion, or by other infection—croupous pneumonia, cerebro-spinal meningitis, small-pox, etc.

In the absence of these symptoms the diagnosis must be held in abeyance until the appearance of the eruption. The eruption shows itself in from twelve to twenty-four hours after the initial symptom—chill, vomiting, or shock—on the face, over the forehead, cheeks, the chin, and often at the same time—as a rule, in fact—the clavicles. Here, at least, it is first seen. When search is made, it may usually be discovered soonest on the neck, breast, and back. It usually spares or skips the region of the mouth, which is left blanched by contracted capillaries, in striking contrast with the scarlet flush of the rest of the face. The white line about the mouth and the apparent bleaching of the chin make the diagnosis easy as between this disease and measles or small-pox. Seen at some distance, the eruption appears uniform, but close inspection shows it to be punctate with confluent halo; yet, though confluent, there are here and there lines or spaces of unaffected surface. This marbled appearance of the skin is very characteristic, and is due to the intense irritability of the vaso-motor nerves, which show paralytic dilatations and spasmodic contractions in the same sets of capillaries. The deeply-colored skin is bleached out by pressure in lines or surfaces, so that figures or letters may be inscribed upon the surface with a vividness equalled in no other disease.

The color is scarlet, that bright red which is designated by this hue, in striking contrast to the duskier red of measles. The Germans speak of the crushed-raspberry color of scarlatina as distinct from the mulberry hue of measles. The shade becomes darker, however, in bad cases or especially under defective hygiene, when it may be substituted by hæmorrhage itself. In a pronounced case the affected skin is more or less œdematous. The eruption lasts from four to six days, extending meantime over the body, but with less uniformity over the extremities, where it may show itself only in blotches or patches. It begins to fade in the order of its appearance, first from the face, neck, and chest, later over the body, and disappears with a desquamation or exfoliation of the skin which constitutes one of the peculiar features of the disease. From regions covered by a thick epidermis, the hands and feet, more or less perfect casts, epidermic gloves and stockings, may be detached. More or less perfect specimens of this kind are to be found in the museums. The desquamation begins usually on the sixth day. It may in a mild case be furfuraceous. It may, indeed, be absent altogether, but it is usually, as stated, membranous or lamellar, the skin peeling off in strips and flakes. The process may be repeated several times during the course of the disease, to form at

times an interesting diversion or an annoying occupation of convalescence. It is impossible to overrate the value of this process of desquamation. It confirms the diagnosis in a doubtful case; it establishes the pre-existence of the disease; it reveals the nature of a meningitis, rheumatism, an ear disease, or nephritis which has suddenly or insidiously developed.

The sore throat is one of the cardinal symptoms of the disease. It precedes the eruption, as has been said, and constitutes at times an overshadowing symptom. Suspicion is excited of the existence of scarlatina by the fact that the child complains of the throat, and it is seen that there is some hesitation or difficulty with deglutition. The act of swallowing is marked by an expression of pain, by the application of the hands to the neck, sometimes—or, as a rule, later in the course of the disease—by regurgitation of fluids through the nose. In many cases an inspection of the throat discloses at a glance the true character of the disease. As a rule, the sore throat of scarlatina differs in no way at first from that of a simple catarrh. There is redness, dryness, and swelling. The mucosa is puffed or glazed, especially about the soft palate and uvula. The glands of the neck become swollen and tender. Diphtheritic patches, often gangrenous sores, may appear later, while an extensive interglandular cellulitis may swell the neck to such degree as to obliterate its natural outlines. It is, therefore, not at all strange that scarlatina is often confounded with diphtheria. It is to be remarked, however, that no individual symptom shows such variation of intensity as the angina. Throat symptoms may be so mild as to be detected only on close inspection (scarlatina simplex or sine angina), or so severe, as said, as to overshadow all other signs (scarlatina anginosa of the older writers). It is now no longer an unsettled question whether the diphtheritic exudations which occur in grave cases belong intrinsically to scarlatina or to a complicating diphtheria. It was maintained, on the one hand, that the false membrane of scarlatina differed essentially from that of diphtheria, and, on the other, that the inflammation of scarlatina renders the individual more susceptible to an attack of diphtheria. The view now prevails that the membrane is primary, that it belongs to scarlatina, and that it may in the vast majority of cases be differentiated from that of diphtheria.

Among the disturbances of the digestive organs common to all the infections, the condition of the tongue is peculiar in scarlatina. The tongue is coated white and studded with red spots, the protruding swollen papillæ, to constitute what is known as the strawberry or mulberry tongue. While this condition is not absolutely peculiar to scarlatina, it occurs in it much more frequently than in any other affection, and from its obtrusiveness is regarded as a sign of much value. It is unfortunately not always present, but when present it should excite at once suspicion of the existence of this disease.

Scarlet fever shows predilection for three organs besides the skin and throat—namely, the ear, the joints, and the kidneys. The way is open to invasion of the ear from the throat through the Eustachian tube. So scarlatina is the most fertile source of earache, otitis media, and otorrhœa. The

membrane of the drum is seen to be intensely reddened on inspection, or it may be paler and pushed outward by a fluid pent up within the drum-cavity. Voss calls attention to the close association that exists between certain cases of ear disease and nephritis. The deafness and pain which announce this complication often stand in relation to the secretion of urine. They speedily disappear with diuresis and resolution of the nephritis. Under the persistent diminution of the quantity of urine and continued albuminuria the hyperæmia of the drum-cavity quickly progresses to exudation, redness, and swelling of the membrane of the drum, which is perforated to give vent to a serous, later a sero-purulent, discharge. Frequent inspection of the ear should be made in the management of every case of scarlatina. Many ear complications set in so insidiously as to be recognized only after irreparable mischief has been done.

The prognosis of the ear affection depends largely upon the period of its recognition, for most cases terminate favorably if treated before serious lesion has occurred. This treatment has reference here not only to the local affection, but also to the action of the kidneys, so that the early recognition and appropriate treatment of nephritis may prevent many cases of ear disease.

From the cavity of the drum to the dura and pia mater the way is often open. Affections of the ear constitute by far the most fruitful cause of lepto-meningitis. Of these affections, chronic suppurative inflammations of the tympanic cavity, which result chiefly from scarlatina, and which constitute over 20 per cent. of all diseases of the ear, most frequently lead to meningitis through caries of the osseous roof of the tympanum. A mere microscopic breach in the thin wall of bone that forms the upper covering of the tympanic cavity will bring pus from the tympanum to the dura. The roof of the tympanum is composed, at best, of an excessively thin plate of bone, which is at times congenitally defective, so that in the young a fold of the dura often pushes itself directly into the cavity of the tympanum. Every meningitis whose cause is not obvious should excite the suspicion of ear disease, which may reveal itself to the sense of smell in an offensive odor before the appearance, or in the absence, of visible discharge; and every case of otitis or otorrhœa in the course of scarlatina calls for warning as to the remote dangers of its neglect.

Affection of the joints is much more uncommon, but there occurs in certain cases or certain epidemics a peculiar scarlatinal rheumatism affecting chiefly the larger joints, ankle, wrist, elbow, and knee. The affection runs usually a mild and short course, but may, unlike true rheumatism, result in suppuration or leave permanent deformity. The joint affection is probably to be referred in these cases to a mixed septic or pyogenic infection.

Of all the signs, complications, or sequelæ connected with scarlatina, no one assumes such prominence and importance as the affection of the kidneys. Scarlatina is said to be the mother of acute nephritis. Aside from the transitory albuminuria which may attend any high fever, disease of the kidneys is comparatively frequent. Epidemics are distinguished from each other in this regard with entire or comparative absence and frequency of this complication.

Bartels declares that it occurred, in 1853–54, 22 times in 180 cases; in 1863, 13 times in 84 cases, and in other epidemics not once in 100 cases. The severity of the individual case or of the epidemic does not necessarily indicate the probability of nephritis. It cannot be said that early exposure as to cold predisposes to it. It may not be ascribed to the affection of the skin, as no such sequel follows small-pox with its much more destructive lesions. Every case marked by high temperature shows, as stated, some albuminuria, but the albuminuria which excites apprehension is that which appears not at the height, but in the later course of the disease—at the end of the third week, after the disease proper and during convalescence. Strictly speaking, the process, is therefore, a post-scarlatinal nephritis. It sets in on the tenth to the thirty-first, on the average in twenty days, after the first show of the rash. It is an acute parenchymatous process, from which the patient recovers or succumbs quickly, very rarely developing into chronic Bright's disease. It is announced often by nervous symptoms, headache, neuralgia, vertigo, insomnia, restlessness, blindness, convulsion, or coma. Puffiness of the eyes, any local œdema, or dropsy should excite suspicion of its presence.

Sweeting showed by statistics that albuminuria stood in direct relation to crowd-poisoning, so that the percentage of cases was in direct ratio to their number. Thus, in 1882, when the hospital ward contained but 64 patients, the percentage of albuminuria was 14, while in 1887, when it contained 1046, the percentage increased to 34.9. It is questionable, however, whether this albuminuria may be regarded as evidence of the true scarlatinal nephritis, which depends more, as stated, upon the character of individual epidemics. Thus, Barthez found 80 per cent., Friedreich but 4 per cent., of cases. It must, however, be admitted that the albuminuria which attends cases of high fever is often the origin of a later nephritis. The typical nephritis presents, as a rule, a picture very different from that of ordinary albuminuria. It distinguishes itself by the gravity of the nervous symptoms, by the extent of the dropsies, as well as by the marked changes—presence of blood, reduction in quantity, even to anuria, etc.—in the urine. It distinguishes itself further by the fact that even the gravest symptoms do not preclude recovery.

A not infrequent sign to announce the advent of the true nephritis is vomiting. Vomiting without cause, especially if repeated several times, should excite suspicion. The patient is found pallid or there is a dusky hue about the face. On inspection of the body it is seen that there is œdema. It may be observed first about the loins, but is, as a rule, noticed first under the eyelids. It appears soon about the feet and in the subcutaneous connective tissue generally. The œdema becomes an anasarca extending over the body, and shows such degree of distension as is hardly equalled in any other disease. The hydrops invades also the serous cavities, the pleuræ, peritoneum, and pericardium. Effusions here may be fatal by mere mechanical pressure. There may be superadded new or mixed infectious elements to contaminate the clear serum with pus or blood. Severe cases begin with tempestuous signs—chill with rapidly rising temperature, vomiting, lumbar pain, headache, amaurosis,

convulsion, delirium, stupor, coma—and such cases may terminate in a few hours. Usually, however, the outlook is not so bad, and even in the presence of grave uræmic symptoms the prognosis is not necessarily fatal.

It is upon the condition of the urine that the recognition of nephritis really rests. The disease is an acute parenchymatous nephritis. It begins, as stated, insidiously or suddenly, and, as also stated, late in the course of the disease. This late beginning is, however, really only apparent. The fact is, the nephritis begins early and develops itself insidiously until it has attained an extent sufficient to show signs.

Scarlatinal nephritis may be divided into two periods, in the first of which there is a diminution in the quantity of urine, albuminuria, and some of the general symptoms mentioned. The second period is distinguished by hæmaturia, with the discharge of formed elements, granular and epithelial casts, also with an increase in the quantity of urine and diminution of the general signs, so that should the urine become more abundant, contain more blood, and exhibit formed elements, though grave symptoms may still show themselves for a time, the worst is over, and, as Sörensen puts it, "the kidneys are beginning to free themselves of the disease." Perhaps the most grave single symptom of nephritis is anuria, but even long-continued anuria is not incompatible with recovery. While it may be said that the gravity of the case corresponds in a general way with the degree of oliguria or the duration of anuria, there need never be despair as to the possibility of recovery, as Whitelaw reported a recovery after a total absence of urine for twenty-five days. As a rule, it may be said that the blood and albumin disappear in mild cases, and the patient entirely recovers from the nephritis in two to three weeks.

Varieties of Scarlatina.—Besides the typical form described, scarlatina shows itself in variation as follows: 1st. Abortive, in which the eruption disappears after a short duration without, or with very mild, throat symptoms, but usually with lamellar desquamation and sometimes with subsequent nephritis. 2d. Fulminant, in which the patient is killed by the poison of the disease before the period of eruption. 3d. Anginose, in which throat symptoms predominate. 4th. Malignant, with the *status typhosus*, in which all symptoms are intense, and hæmorrhage may occur superficially from the various mucosæ or into the skin, or with rapid collapse after signs of a cholera morbus. In some very exceptional instances of undoubted scarlatina the eruption is entirely wanting, throat symptoms only being present. In these cases careful inspection will usually disclose some eruption on covered parts, especially on the posterior aspect of the body. It may be seen at times on or over any part of the body immediately after death in fulminant forms.

True diphtheria may coincide with or follow scarlatina; much more frequently, as a rule, the membrane which forms in the throat is *sui generis*. The membranous angina or pseudo-diphtheria of scarlatina is much less amenable to treatment than true diphtheria.

Relapse must be distinguished from second attack or recurrence. Such cases only should be considered as relapses where the fever and the eruption

more or less immediately follow the first attack, as in the course of typhoid fever. Recurrence or second attack is, as stated, very rare. One attack gives immunity, as a rule, for life. A relapse occurs before there can be any question or consideration of immunity as a result of reabsorption of toxic matter from the throat or wherever lodged. Thomas, who studied this subject most thoroughly, admits a relapse not later than four or five weeks after the first attack. The disease repeats itself in relapse in all its details, and for the most part in equal severity. Should they vary in severity, the second attack is apt to be the less than the more severe. At times it is only rudimentary. Notwithstanding the renewed infection, relapses have, as a rule, a more favorable prognosis.

Diagnosis.—The diagnosis rests upon—1st, the absence of previous attack; 2d, the existence of other cases; 3d, the short period of incubation, one to seven days as a rule; 4th, the violence of the invasion, especially the occurrence of unprovoked vomiting (80 per cent. of cases) and the nervous symptoms; 5th, the early appearance (second day) of the eruption, which shows itself first usually about the clavicles, is scarlet-colored, diffuse, but punctate upon close inspection—in its disposition about the face, commonly sparing the mouth, showing in vivid contrast the blanched lips and the blazing cheeks; 6th, the strawberry tongue; 7th, the early appearance of throat symptoms, with glandular enlargements in the neck; 8th, the lamellar desquamation; 9th, the ear complications; 10th, nephritis.

In very mild, sporadic, or anomalous cases the diagnosis may be determined only by desquamation, complications, or sequelæ.

Scarlatina is differentiated from measles by knowledge of previous attacks of either, of the existence of other cases of either, especially in the same family, neighborhood, or school; by the longer incubation of measles when the period of exposure may be (exceptionally) known; by the coryza which precedes the eruption of measles, and the angina that of scarlatina; by the shorter or more intense invasion of scarlatina with vomiting and sharp nervous symptoms not so common in measles; by the time of appearance of the eruption, twenty-four to forty-eight hours after initial chill or vomiting in scarlatina, four days in measles; by the color, character, disposition, and duration of the eruption, dark red, aggregated in patches, and disappearing in two to four days in measles, scarlet-colored, punctate, diffuse over the chest and face, sparing the mouth, disappearing in eight days or more in scarlatina; by the complications or sequelæ—bronchitis, catarrhal pneumonia in measles, joint and ear affections, nephritis in scarlatina; by the desquamation, usually branny in measles, membranous in scarlatina.

Scarlatina is differentiated from rubella (rötheln) by the longer incubation or shorter or absent stage of invasion; by the darker-colored and shorter duration of the eruption of rubella; by the associate catarrh of the nose and eyes in rubella (absent in scarlatina); by the much more severe faucial inflammation and gland implication in scarlatina; by the much milder character and shorter duration of rubella.

Septicæmia and pyæmia show, with the history of a cause, successive chills, irregular temperature, efflorescences in appearance and in order of distribution quite different from the eruption of scarlatina, more marked enlargement of the liver and spleen, more common general affections, metastatic processes, and a longer duration. Erythema shows a diffuse rather dark redness without points or desquamation, though sometimes with a light furfuraceous desquamation, with absent or but very slight fever (up to 100° F.), has neither the throat symptoms nor complications of scarlatina, and disappears in a few days. Drug eruptions, copaiba, cubebs, and antipyretics, have a history of administration, no fever, and no complications.

Scarlatina differs from diphtheria in its cause. At least it may be said that the cause of diphtheria has been now quite definitely determined, and that, while the same cause is not to be found in unmistakable cases of scarlatina, it must be held in mind that the diseases, as stated, may coincide, and that either may be a sequel to the other. These things, however, are exceptional, the rule being that the diseases exist alone, and that, as stated, the exudation of scarlatina is not that of diphtheria, but is *sui generis*.

Clinically, the affections differ as follows: The false membrane appears at once in diphtheria, later in the course (three to five days) of scarlatina. It shows itself in nearly all cases of diphtheria, but in only severe cases of scarlatina—namely, such as are marked by high fever, delirium, etc. at the start. It shows a preference after the pharynx for the larynx in diphtheria, and for the upper respiratory passages in scarlatina. In connection with it suppuration of the cervical glands and affections of the ear are frequent in scarlatina, rarer in diphtheria. The interglandular connective tissue is indurated in scarlatina and only œdematous in diphtheria. Paralysis, which is frequent in or after diphtheria, is almost unknown in scarlatina. On the other hand, nephritis, a frequent sequel of scarlatina, is very rare after diphtheria. Lastly, as stated, treatment has much less effect on the membrane of scarlatina. For all these reasons it is proposed by good clinicians to abandon the use of the term "diphtheritic" in scarlet fever, and to designate such cases as membranous scarlatinal anginas.

Complications.—Scarlatina distinguishes itself by the intensity of its poison. It is therefore natural to expect to find frequent and various complications. Perhaps there is no disease in which complications are so many and manifold. The gravest are those which affect the brain. The disease sets in with symptoms of shock, with profound prostration, with delirium, convulsions, and coma, and these accidents may occur at any time in the course of the disease. They may be due, in the first place, to direct effect of the poison upon the nervous system. They may be the effects of septicæmia or of suppurative processes about the throat and neck. They may be due to mechanical pressure of the swollen tissues upon the great vessels in the neck, thus interfering with the circulation in the brain. They may be due to meningitis, or, finally, they may be due to kidney disease.

The nervous symptoms which are due to direct intoxication are, as a rule,

the most intense. They most directly and frequently threaten and take life. They inspire the dread of the disease. These severe nervous symptoms, indicative of fresh influx or inundation of the poison, not infrequently precede the eruption, or, occurring after the eruption, even in its full efflorescence, cause it to quickly fade away. These are the cases in which the eruption is said to "strike in." The people understand the significance of the subsidence or disappearance of the eruption. It is often found to occur simultaneously with a sudden elevation of temperature to 105° or 107°. General symptoms or evidence of septicæmia, meningitis, endocarditis, etc. may appear upon the same or the following day to account for the change. It is, however, an entirely erroneous view to ascribe these changes to the subsidence or disappearance of the eruption. The disappearance of the eruption is to be interpreted as a coeffect of the more severe poisoning of the blood, or it is to be attributed at times to a mixed infection, as to invasion by the micro-organisms of pus.

So, too, under these circumstances the eruption may entirely change its character, lose its individuality, and become hæmorrhagic. This change is observed, however, in scarlatina very much less frequently than in measles. The danger in scarlatina is on the part of the brain and the kidneys, and the secondary affections which occur in its course are due mainly to suppurative processes which display themselves rather in metastatic abscesses, and more especially in affections of the serous membranes.

Complications on the part of the ear belong almost naturally to scarlatina, and result from simple extension of the infectious process from the throat. Allusion has been already made to the changes which take place in the drum-cavity in the course of the disease. In certain cases these changes assume prominence, so that the inflammation about the ear, exudative processes, suppurations, involvement of the mastoid sinuses, direct attention especially to this organ. Implication of the ear is usually earliest announced by earache, ringing in the ear, and deafness. Next to cerebro-spinal meningitis, scarlet fever is the most fruitful source or cause of deafness, and the condition of the ear is watched with solicitude throughout the course of the disease, that treatment—and, if necessary, operative interference—may be resorted to before irreparable damage has been done. Where any history of aural affection is wanting or inspection may show no change, an insidiously developed otorrhœa may make itself manifest, as stated, to the sense of smell.

The complication on the part of the brain which results from extension from the ear as a lepto-meningitis has been sufficiently noticed. It may be said that meningitis is observed more frequently as a sequel than as a complication in scarlatina, but it remains true that scarlatina is the most frequent cause of simple non-specific meningitis.

The eye is seldom affected by scarlatina. There is rarely such a degree of coryza as to make the disease simulate measles. In exceptional cases, more especially in tuberculous subjects, there may occur keratitis and kerato-malacia, or even panophthalmitis, with destruction of the globe.

The kidney disease may cause amaurosis, which disappears, as a rule, as

suddenly as it sets in, or albuminuric retinitis, which distinguishes itself from that of other forms or causes of kidney disease by its transitory character.

Grave complication is not infrequently presented by the intense inflammation about the neck. The glands, the lymph-vessels, the interglandular tissues, are swollen and amalgamated into a mass of board-like induration which fixes the head to the body, compresses the great vessels, and results not infrequently in extensive suppurations. Phlegmonous processes, gangrenous destructions, occur frequently in connection with diphtheritic or pseudo-diphtheritic deposits in the throat. The pressure may compress the trachea or suffocate by œdema of the glottis. Pus burrows down into the chest, to at times erode in its course large vessels, to lead to fatal hæmorrhage, or to destroy important nerves or other structures. The inflammation of the throat may extend to involve the larynx and bronchial tubes. Pneumonia, both bronchial and croupous, occurs not infrequently in grave cases. Hæmorrhage of the lungs, gangrene, more especially œdema, hypostases, take life directly or indirectly by over-strain of the heart.

The most frequent and fatal so-called end or terminal complications are the affections of the serous membranes. Meningitis heads the list, in that it is not only the most severe, but most early, of these affections. It occurs at times, as stated, almost with the onset of the disease, so that the separation of this affection from toxic effects of the blood upon the brain itself may be difficult. In meningitis the headache becomes more intense, or recurs if it have subsided. The special senses suffer extreme hyperæsthesia. There is usually evidence of affection of the membranes of the spine—opisthotonos, vomiting, convulsions. The pleura is next most frequently affected. Scarlatinal pleuritis distinguishes itself, strange to say, by its unilateral character, and differs from pleuritis from other causes in the fact that it so easily becomes purulent. Pericarditis is usually so much more rare as to be generally overlooked, while endocarditis is readily recognized by the evidence of valve lesions, hypertrophies, and embolic products.

All these various complications are now known to be due to the micro-organisms of pus, which, strictly speaking, have nothing really to do with the cause of scarlatina. The cause of scarlatina opens the way for their invasion, and these micro-organisms—or the evidence of their invasion—have been traced directly from the throat as their surface of origin.

Prognosis.—The prognosis of scarlatina varies greatly, perhaps as in no other disease. On account, however, of the severity of certain epidemics, and of the suddeness with which the disease may assume gravity in any individual case by reason of its own poison or by reason of complications, the prognosis is always grave. Reimer, who studied the subject from this standpoint most thoroughly, says that the prognosis progresses from simple, uncomplicated cases which have no mortality, through complicated cases of medium gravity, with a mortality of 25 per cent., up to the severest cases, whose mortality reaches 83¾ per cent. The ratio of its gravity to other disease is shown in the difference in the prognosis of a case, whether preceded or followed by

scarlet fever. These figures show that when scarlet fever is followed by other diseases the mortality is 44 per cent., but when scarlet fever follows other diseases the mortality is 82 per cent.

In estimating the prognosis it may be said that the pulse, as a rule, corresponds to the temperature. Arhythmia is usually a forerunner of complications. Grave nervous symptoms are always ominous, as are also extensive suppurative processes. It is not a good sign for the eruption to "sink in." The complications on the part of the kidneys, however severe the signs, though grave, are never necessarily fatal. From the gravest accidents recovery is still possible.

Pneumonia, pleurisy, with its wonted suppurative course, peritonitis, often of such insidious onset as to have escaped detection at first, intensely aggravate the prognosis. Peri- and endocarditis are almost necessarily fatal. An undue protraction of the disease, by reason of reabsorption of products to constitute a relapse or by reason of complications, makes the prognosis grave, in correspondence with the intensity of the signs, duration of the disease, or character of the complication. Some of these cases succumb finally to atelectasis, heart failure, decubitus, or marasmus.

Morbid Anatomy.—The morbid anatomy of scarlatina is the picture of the infections in general; that is, the lesions encountered are those common to all the acute infections, among which scarlatina takes place according to its gravity. The body may or may not show exanthem. Usually, because death occurs early, the eruption is faded to a mere residue, or spots of hyperæmia may with difficulty be distinguished from post-mortem changes. There are occasionally encountered, as stated, the fulminant forms, in which the eruption never appears, or appears only after death to establish the nature of the hitherto unrecognized affection. The various stages of desquamation may be observed along with the changes produced by hydrops in cases where death has occurred in consequence or in the course of renal disease. Protracted cases will show emaciation and decubitus. The blood is fluid, dark, and scant of fibrin. The clot is small, black, friable. The heart is lax and flabby. The spleen is swollen, its pulp soft and red. The various serous membranes show ecchymoses.

The most persistent changes are seen about the throat. There may be slight evidence of hyperæmia, though all œdema will have subsided. The glands about the neck remain enlarged and indurated, to present at times a conglomerate mass, at times, again, suppuration. The liver and kidneys show histological change in swelling and opacity of their parenchymatous structure. Inasmuch as death usually occurs, as stated, early in the history of the disease or as the result of blood-poisoning and nervous symptoms, there is usually to be found hyperæmia of the brain and of its membranes, and in quite fresh autopsies œdema of the brain.

Prophylaxis.—Isolation is the only prophylaxis; and, inasmuch as the area of infection is so closely circumscribed, isolation is much more effective in scarlatina than in measles or small-pox. As, however, the disease may undoubtedly

be carried by third persons, the attendants upon the sick should not come in contact with unaffected members of the family. The best protection is offered by removal from the house of all children liable to—that is, unprotected by previous attack of—the disease. Unaffected children remaining at home should not attend school or other assembly for several weeks after perfect recovery of a member of the family. It is believed, whether it may be proven or not, that contagium emanates from the body so long as desquamation continues, and the child should not be allowed to associate with its fellows until the last scale of skin has been removed. Desquamation continues longest on the soles of the feet, and inspection of these parts gives the best answer as to the time when all desquamation shall have ceased. Such desquamation has been observed as late as forty days after the disappearance of the eruption, though it usually ceases entirely in less than half that time.

Prophylaxis implies also the destruction or thorough disinfection of all articles which have come in contact, direct or indirect, with the patient. The room should be disinfected, ceilings whitened, walls rubbed down with bread, floors scrubbed with corrosive-sublimate solutions, carpets or rugs beaten and suspended in the open air for a long time, bedding and clothing boiled, if not buried or burned, or, if preserved, subjected when possible to live steam. It is a sad comment upon our sanitation that public disinfecting stations are not yet established in all our cities and towns. Prophylaxis involves attention to detail. The disease has been conveyed by letters sent out from a sick-room, by cushions of chairs, curtains, piano-covers, etc. Special attention must be paid to the discharges, to sponges, cloths, and towels used in ablutions. The fact is, that sponges, handkerchiefs, etc. are best substituted by rags, which may be immediately destroyed by fire. It must not be forgotten that the hair of the head retains and conveys contagium. Thorough ablution with soap and water or with the carbolized soaps will disinfect the hair. The hair of the boy patient should be cut short. It must be remembered that even the shoes require disinfection. They may be painted inside and out with carbolic acid and glycerin, equal parts. Ventilation of the sick-room throughout the whole period of the disease is not only a necessity of treatment, but also of disinfection. The frequent bathing of the body, with the subsequent application of some unctuous material—cocoa butter, lanoline, vaseline, etc.—not only gives great comfort to the patient, but confines the poison to a narrower field. After the recovery of a patient, and more especially after a death, outside windows should be thrown open and the room ventilated for a week. Here, too, attention must be paid to detail. Closet-doors must be opened and the inside of closets with their contents disinfected and ventilated as before. The fact is, the city government should take charge of all such apartments. They should be disinfected and ventilated under the inspection of health authorities. The inside doors to halls and to other rooms should be closed by the seal of authority, and the same precautions taken as in the prevention of entrance or interference in cases of crime. Scarlet fever is for the most part spread by ignorance, by carelessness, by blunders which are worse than crimes.

Reference is made here to the premature return of children to school or the constant attendance at school of unaffected cases in a family, to contact in street-cars, railroad-trains, steamers, etc. Parents, nurses, and even physicians, are all too careless in this regard.

In the way of drugs there is no preventive of scarlet fever. The claim that a drug may protect against the disease because when administered it produces a symptom which resembles that of the disease is, in the light of our present knowledge regarding the infections, worse than mediæval gibberish —worse, because it may beget a false security in reference to exposure. This claim has been made for belladonna because it flushes the face. It has no more foundation in fact than protection by a blush, which has the same effect. Belladonna by making a child sick rather predisposes to than protects against scarlatina.

The hope that has been cherished regarding protection by vaccination has proven equally vain. Attempts have been made to inoculate certain disease-products of animals, horses, dogs, and rabbits with a view of producing a milder or modified form of scarlatina. Claim has been set up in this direction, as by Strickler, who introduced the nasal mucus of horses supposed to have been affected with the disease into the bodies of twelve children, in all of whom it produced sores at the point of introduction, with circumjacent inflammation of the skin and lymph-glands. It was stated that these children thus inoculated failed to contract the disease after exposure to scarlatina. These experiments were made in imitation of the first experiments of Jenner with small-pox, but the objections to accepting such conclusion are numerous and obvious. In the first place, it is not known that any of the lower animals really suffer from scarlatina or any allied disease. Secondly, it has not been established by experiment that the disease which results from the introduction of scarlatinal matter of man into animals is really scarlatina. Third, it could not therefore be known that matter taken from animals was the product of this disease. Fourth, susceptibility is so much less in scarlatina that failure to contract the disease after exposure has not the same weight as in small-pox.

Treatment.—The treatment is wholly symptomatic. The sick-room requires constant, thorough ventilation from the outside air. The temperature should be held at from 65° to 70° F., as registered by a thermometer—not at the door, window, or fire, but at the head of the bed. An open fire in winter is preferable to any other method of heating. The patient should wear a long muslin night-dress without other clothing. The bed-covering must be as light as is consistent with comfort.

Milk and meat soups make the best diet. Water, carbonated water, seltzer, apollinaris, lemonade, toast-water, barley-water, should be given freely to relieve thirst and to keep the kidneys flushed. Drink should be proffered once an hour in high fever during the day.

The utmost cleanliness is to be maintained by frequent sponging and bathing of the surface. Daily tepid baths (full length) give the greatest comfort throughout the disease. Fever above 103° F. is best combated with cold

sponges, cold packs, or cold baths. Cold baths are most effective, but are seldom practicable as yet. It is not essential that the temperature be brought down to the normal degree. A reduction of a few degrees suffices to give the patient comfort, and relieves all danger attendant upon high temperatures. A warm or tepid bath will reduce the temperature one or two degrees, and patients solicit such bathing when the cold bath may excite terror. While it is true that the temperature reaches the highest grades in scarlatina, and the patient suffers corresponding discomfort and danger, it is not true that the danger is caused by the fever. The danger, the discomfort, and the fever are produced by a common cause—namely, the poisoning of the blood; and there can be no question of radical therapy until after the discovery of some agent, some antitoxine, which will neutralize the chemical poison circulating in the blood. It is indeed a question if some fever be not salutary. We combat the fever in our day more especially with regard to the comfort of the patient. A difference of two degrees makes great difference in the feelings of the patient. The reduction of high temperatures by cold bathing is attended, as a rule, with diminution of discomforts and dangers. The bath addresses the cause indirectly through its effects. Frequent bathing is the best therapy in the treatment of scarlatina as of any other infection. There may be reasons which render a bath impossible. In these cases resort must be had to frequent ablutions. It may become necessary to substitute a bath by drugs, especially in the presence of other indications. Resort may then be had to the antipyretics. Phenacetin is the least injurious. It may be given to a child in a dose of 2½ to 5 grains—to an adult in double this dose once or twice in the course of a day. It is of especial value in headache or other nervous distress. It is best administered in capsule or in powder, taken directly upon the tongue or stirred—that is, suspended—in milk, or, in case of high fever with dry tongue, floated upon the surface of a teaspoonful of water. Only in case of failure with phenacetin should resort be had to antipyrine or antifebrin, either of which must be given in half the dose of phenacetin. Burning and itching of the skin are best allayed by application, after tepid baths, of vaseline, cocoa butter, lanoline, goose-grease, bacon, or fresh lard. Quiet, peaceful, and more or less restorative sleep is wont to occur after a bath and inunction in this way.

Nervous distress, jactitation, convulsions, insomnia, headache are best combated by bromide of sodium in doses of from 5 to 10 grains to a child, 30 to 40 to an adult, largely diluted, or if more obstinate by chloral, 5 grains to a child, 15 grains to an adult. No other single remedy gives the comforts of chloral in repeated doses of 2 or 3 grains. Broken doses of Dover's powder in grain doses to a child, 3 to 6 grains to an adult, may substitute it in a suitable case. Ice-bags should be applied to the head for meningeal symptoms. The vomiting which occurs in the inception of the disease is often sufficiently relieved by carbonated drinks, the best of which is the German seltzer water, with milk equal parts, or by lime-water and milk 1 : 3, by small doses of bismuth (5 to 10 grains), by the bicarbonate of sodium in equal dose, or by sips of excessively hot water. The most powerful drug we possess is

chloral. The most refractory vomiting, of whatever cause, will yield to the administration of a few doses of from 2 to 5 grains of chloral diluted in a dessert- to a tablespoonful of peppermint-water. Should the remedy be rejected before it can be absorbed, it may be introduced into the bowel in double dose. It must be a remarkable case to resist chloral in one or other of the modes of use or to justify resort at last to a subcutaneous injection of morphine.

Throat symptoms call for inhalations of steam, best from the steam vaporizer, simple or medicated with bicarbonate of sodium, saturated solution of boric acid 3 drachms to 4 ounces, or carbolic acid ½ a drachm to 4 ounces, or thymol 5 grains to 1 ounce alcohol or 3 ounces of water, or gargles of hot water, of carbolic acid 15 to 30 drops to 4 ounces, perchloride of iron 1 drachm to 4 ounces, or direct applications of carbolic acid, with glycerin equal parts, bichloride solutions from 1 : 1000 to 1 : 100, or intraparenchymatous injections (tonsillar) of a few drops of the carbolic-acid solution by means of a hypodermic syringe with a fine long aspirator needle. Cloths wrung out of boiling water, applied about the throat and covered in by thick dry cloth, relieve the pains of extreme distension.

Affections of the ear are best treated by a douche of hot water and a Politzer inflation with air. Tension in the membrane of the tympanum may require puncture, and suppuration of the mastoid cells trephining. Earache is best relieved by instillation of hot water or solutions of atropine, 1 grain to the ounce. Otorrhœa is best treated by filling the external canal with powdered boric acid after thorough cleansing with a cotton-wrapped sound, or direct application to accessible granulations of chromic acid, London paste, or the galvanic cautery.

Nephritis calls imperatively for hot baths, under which all the symptoms of this complication, including vomiting, are wont to speedily subside. The bath must be hot (100° to 110° F.); the patient must be rolled in a blanket after it, and be allowed to sweat for an hour. Rheumatism calls for the salicylates in saturating dose. Alcohol, digitalis, and nitro-glycerin may become necessities in the later course of all grave cases, and may be urged in overdosage, together with other analeptics—camphor, ether, musk—in the way of a "forlorn hope" in fulminant forms.

MEASLES.

By JAMES T. WHITTAKER.

Synonyms and Definition.—Measles (Sanscrit), masura, masern (German, spots), rubeola (Sauvages), ruber, rougeole (French, red), morbilli (Italian, diminutive of morbus),—an intensely contagious, acute infection, characterized by coryza and bronchitis, a red spotted eruption with branny desquamation, fever of typical course, subsiding at efflorescence, with liability, mostly as sequel, to catarrhal pneumonia, sometimes to tuberculosis.

Ahrún (Aaron), a Christian priest and physician of Alexandria (A. D. 610–641), is celebrated as having been, by universal acknowledgment, the first writer to have mentioned small-pox and measles. Though existent from time immemorial, measles was first described by Rhazes (900 A. D.) in an attempt to separate it from small-pox. Rhazes noticed among the symptoms of measles "redness of the eyes, with a great flow of tears, nausea, and anxiety," remarking also that the measles "that are green or violet-colored are of a bad kind, especially if they sink in suddenly, for then a swooning will come on and the patient will soon die."

The disease was described under the name *hhasbah*. Nearly all subsequent writers adopted the Italian term morbilli up to the middle of the eighteenth century, when Sauvages substituted for it or re-established the term, said to have been first used by Haly Abbas, rubeola, which the French accepted in their own equivalent of rougeole. Fagge laments the substitution of rubeola and its adoption by later English writers, but surely without cause, for morbilli refers simply to the mildness of the malady—a fact to which there are many exceptions. Morbilli is, anyhow, too indefinite to be the name of any disease. It holds its place only under the sanction of antiquity and authority. Rubeola means something definite. It expresses a characteristic feature of the disease—in fact, the most characteristic feature—the redness of the eruption. It is unfortunate that this term, rubeola, has been adopted by certain German writers to express that particular subvariety, special, or hybrid form of disease known as German or French measles or popularly in Germany as rötheln. As scarlatina would seem by universal acceptance to be the most appropriate name for scarlet fever, rubeola must be the most appropriate for measles.

Forcest, the Dutch Hippocrates, in 1565 first pointed out certain distinctions between measles and scarlet fever, though the separation of the affections is usually credited to Sydenham (1665), the English Hippocrates.

Sydenham described the rigors which constitute the chill in the inception

of the disease, and furnished an account close and succinct enough to entitle him to the position of pioneer. Thus: "It generally attacks infants, and, with them, runs through the whole family. It begins with shiverings and shakings, and with an inequality of heat and cold which, during the first day, mutually succeed each other. By the second day this has terminated in a genuine fever, accompanied with general disorder, thirst, want of appetite, white (but not dry) tongue, slight cough, heaviness of the head and eyes, and continued drowsiness. Generally there is a weeping from the eyes and nostrils; and this epiphora passes for one of the surest signs of the accession of the complaint. But to this may be added another sign equally sure—viz. the character of the eruption. The patient sneezes as if from cold, his eyelids (a little before the eruption) become puffy; sometimes he vomits; oftener he has a looseness, the stools being greenish. This last symptom is commonest with infants teething, who also are more cross than usual. The symptoms increase till the fourth day. At that period (although sometimes a day later) little red spots, just like flea-bites, begin to come out on the forehead and the rest of the face. These increase both in size and number, group themselves in clusters, and mark the face with largish red spots of different figures. These red spots are formed by small red papulæ, thick set, and just raised above the level of the skin. The fact that they really protrude can scarcely be determined by the eye. It can, however, be ascertained by feeling the surface with the fingers. From the face—where they first appear—the spots spread downward to the breast and belly, afterward to the thighs and legs. Upon all these parts, however, they appear as red marks only. By the eighth day the spots have disappeared from the face and show but faintly elsewhere. On the ninth day there are no spots anywhere. In place thereof, the face, trunk, and limbs are all covered with particles of loosened cuticle, so that they look as if they had been powdered over with flour, since the particles of broken cuticle are slightly raised, scarcely hold together, and, as the disease goes off, peel off in small particles and fall from the whole of the body in the form of scales."

The primeval home of measles is unknown. From its earliest recognition it has prevailed in epidemic form in Asia, Europe, and South America. It was imported to the United States with the first settlers, to gradually spread over it with the march of the pioneers. It reached Oregon in 1829, California and Hudson's Bay in 1846, the Sandwich Islands in 1848, whence it was carried to Australia in 1854, Greenland in 1864. Though the disease has now become indigenous everywhere and individual cases are of continual occurrence in large cities, measles usually prevails as an epidemic over a wide extent of country, with intervening periods of absence. Epidemics die out in two or three months from lack of material. Measles does not hold over in sporadic cases like scarlatina, but disappears completely, to reappear with reaccumulation of material every three or four years.

Measles is a disease which is characterized by singular uniformity of appearance. It has always presented itself in exactly the same way and with

the same signs, course, complication, and duration from its earliest recognition. It has in it much less of the irregular, capricious, and unexpected than has scarlet fever. It does not show the sudden changes, storms, and strokes of lightning in the midst of comparative fair weather that may occur in scarlatina. Consequently the disease is by no means so much feared.

As the name indicates, it is considered a comparatively mild disease, a diminutive disease. It is the nature of measles, aside from complications and surroundings, to be mild in its course, but it may assume, especially under bad hygiene, a malignancy and mortality which associate it with the plague and the worst forms of infection. The fact is, the mildness of measles is overrated, or, at least, is to a great extent counterbalanced by the frequency of the disease and the quality of its complications. Thus the statistics from the whole of Austria and Saxony from 1873–87 show that there died in every 10,000 people in Austria of measles 27, of scarlet fever 67; in Saxony of measles 25, and of scarlet fever 48. Henoch quotes the mortality of measles in Berlin in 1887 at 0.74, scarlet fever at 0.85 per cent. To get some idea of the respect which is due to measles as a malign disease, as one of the veritable plagues of mankind, we must study the record (Hirsch) of its ravages in savage and semicivilized lands. As late as 1749 measles carried off among the aborigines about the Amazon 30,000 people, whole tribes at a time; in Astoria one-half of all the inhabitants fell victims to measles in 1829; and the same proportion was observed among the Indians at Hudson's Bay in 1846, the Hottentots in 1854, the Tasmanians in 1861, and the Mauritians in 1874. Squire relates that a frightful epidemic of measles in the Fiji Islands carried off 20,000—that is, nearly one-fourth of the whole population. Cruikshank reports of this attack that later in the epidemic, when it was said to be like the plague, the people with fear abandoned the sick. The excessive mortality resulted from terror at the mysterious seizure and the want of the commonest aids, thousands being carried off by want of care, as well as by dysentery and congestion of the lungs, which set in as complications. The effect of crowd-poisoning in measles was well illustrated in the mortality of measles among the Confederates in the War of the Rebellion, where 1900 of the 38,000 cases terminated fatally. In two of the larger hospitals the mortality (still according to Hirsch) amounted to 20 per cent., and in some of the improvised hospitals about Paris in the Franco-Prussian War (1871) it reached 40 per cent. Masterman says that at the beginning of the Brazilio-Paraguayan War measles swept off nearly one-fifth of the national army in three months, not from the severity of the disease, for he treated about fifty cases in private practice without losing one, but from want of shelter and proper food.

Etiology.—Measles knows no consideration of geography, climate, sex, race, or caste—respects nothing but sanitation, which puts a muzzle on it and makes it mild. If, therefore, the disease seemed to prevail more extensively and severely among the colored race, it was not because of physiological preference, but of unfavorable hygiene.

Susceptibility to the disease is almost universal, so that it has been said that

if measles had the mortality of scarlet fever the human race would have long since become extinct. The eminent contagiousness of the disease is shown in the attack of whole communities previously entirely or for a long time exempt, as in the Faröe Islands, where 6000 people were seized at once, and in the cases of extensive prevalence just mentioned. In 1886 the disease overran nearly the whole of Russia. The universal susceptibility is best observed in the cases of isolated islands. Up to the present time the disease has visited the Faröe Islands four times (1781, 1846, 1862, 1875), and Iceland four times (1644, 1694, 1846, 1868). In some of these cases the intervals between epidemics have been so great as to have furnished a large amount of material for attack, so that upon some occasions only a few old people, they who had the disease in infancy, were left to attend upon the sick. Measles has in these cases suspended all business operations and inflicted upon a community as much distress as the gravest infections. Measles, therefore, makes up for its mildness by its range.

Universal susceptibility implies exquisite contagiousness. The poison of measles is eminently diffusible. It must be very light. It floats in the atmosphere about a patient and does not stick long to things.

The first experimental proof of the contagiousness of the disease was furnished by Home of Edinburgh in 1758, at the instigation of Munro. Home soaked rags in blood from cuts made through the spots of measles and applied them for three days upon fresh cuts in the arms of healthy persons, inducing thereby veritable but much milder attacks than the prevalent form. The saturated rags retained their infectiousness but ten days. There seems to be no doubt as to the infectiousness of the blood. Katona in 1842 failed to inoculate the disease in but 7 per cent. of 1222 cases. He used blood mixed with other fluids, sometimes with the fluid of vesicles, sometimes tears. A red areola formed about the point of inoculation, to be followed in seven days by fever and the ordinary prodromata. The eruption appeared in nine or ten days, and the disease ran a regular but milder course. Joerg and Wendt made the same experiments, produced measles, but failed to find any mitigation of type, so that any hope of protection by inoculation proved futile. Mayr claimed to be able to inoculate measles with the nasal mucus applied directly to the mucous membrane of children; and Berndt asserts that Monroe and Lock succeeded in producing the disease with desquamations of skin, with tears, and with saliva.

There is, however, uniformity of opinion only as regards the blood. The fact is, there is need of more modern investigation with modern methods of control. The disease is certainly contagious throughout its entire course, most intensely so at the period of fullest effloresence—*i. e.* at its acme—less during the stage of incubation, least, if at all, during and after desquamation.

Measles prevails more distinctly in the colder months. Of the epidemics tabulated by Hirsch, 3390 occurred in the colder and 191 in the warmer months. The frequency of epidemics in winter has usually been ascribed to the closer contact of people at this season. It is certainly observed in cities

that the disease assumes epidemic proportions with opening of schools and kindergärten. These institutions especially seem to disseminate the disease, because measles is a children's disease, and a children's disease because it attacks at the earliest exposure. Escape in childhood by no means secures exemption, as is evidenced by the attack of people of all ages in isolated regions. Kindergärten are thus sometimes ironically said to be institutions for the dissemination of infectious diseases. Hirsch is unwilling to admit that the greater frequency of the disease in winter is due to closer contact, as the same frequency is observed, he says, in the tropics, in India, South China, and Brazil.

Measles occurs at all ages, preferably from one to five, the period of earliest exposure—rarely among sucklings, the age of least exposure. Part of the exemption of very young infants under six months must be due to comparative insusceptibility. Geissler reports of Meerane in 1861 that 1754—that is, nearly 60 per cent.—of the children were attacked in the following proportions regarding age: under three months, 12.07 per cent.; three to six months, 18.05 per cent.; six months to one year, 35.06 per cent.; one to two years, 56.5 per cent.; two to three years, 61.2 per cent.; three to four years, 67.9 per cent.; four to five years, 70.9 per cent.; five to six years, 72.5; six to seven years, 77; seven to eight years, 81.3; and thereafter a progressive decline, based of course upon smaller numbers, as children at more advanced age had secured exemption by previous attack.

The susceptibility to measles is so great, however, that even the youngest children do not entirely escape. Steiner reports cases in children of four or five weeks of age; Monti recorded two cases in children under two months; Lomer and Williams declare that the fœtus may be affected; Thomas says that after considerable search he was able to discover but six authentic accounts of children born with measles where the diagnosis could be established by the actual presence of the eruption at the time of birth. Redness and desquamation alone cannot be accepted as evidence of the disease, as these appearances are often presented in perfectly healthy children. It is certain that pregnancy is no defence.

As a rule, but very slight exposure suffices for the reception of the disease. It is assumed, because the fact may not be demonstrated, that the poison is inhaled, and that it is received also upon the exposed mucosæ. The fact that affection of the conjunctiva and the nasal mucosa assumes such prominence in the very early history of the disease lends support to this view. The virus of whatever nature—it is almost safe to declare it a micro-organism—probably does not require a broken surface to secure absorption, but penetrates to the superficial lymph-vessels in the mucous membrane upon which it lodges. The length of exposure necessary to secure or escape infection will necessarily depend upon the intensity or concentration of the cause in the atmosphere. Thus, in a close, hot, badly-ventilated room emanations accumulate to such degree as to render the atmosphere highly infectious, whereas a larger, well-ventilated apartment may so dilute and so diffuse the poison

as to require a much longer stay to secure infection, if the individuals exposed do not escape it altogether. The better ventilation, as by open doors and windows, and the presence of fewer people in apartments, have been brought forward to account for the comparative freedom from the disease in summer.

The poison of measles has, however, by no means the tenacity of life or duration of existence of that of scarlet fever. Rags soaked in the blood of measles may retain infection, as stated, for ten days, but clothing contaminated under ordinary exposure soon loses its infectiousness. The cause of measles clings, however, for a time to all objects upon which it may fall. Cases are abundantly recorded in which the disease has been conveyed by third parties and things, the so-called fomites. Thus, Panum records an instructive case where measles broke out in an isolated house visited by no one except a physician, who had reached the house two weeks before after having travelled four miles in an open boat in stormy weather. Thuessink declares that he knew of a case caused by a letter which had been sent from an infected house, and a similar case produced by an engraving sent by mail.

As a rule, measles attacks but once. One attack confers immunity for the rest of life. The older writers (Willan, Rosenstein) made this declaration dogmatic, and maintained that subsequent attacks were mistakes in diagnosis. Henoch believes this statement to be exaggerated. It is certain that authentic cases of second and third attacks have been recorded. Spiess declared that in the Frankfort epidemic of 1866–67 recurrences were unusually frequent. This testimony is, however, invalidated by the statement that nearly half the cases were recurrences or relapses from this or a previous attack. Most of these cases must have been cases of rötheln. We may not deny the testimony of such competent observers as Henoch, Kassowitz, Prunach, and others, but every case of second or repeated attack should be regarded with scepticism until the evidence of the existence of the disease is incontestably established. When Panum states of the epidemics of the Faröe Islands, which occurred at such distinct intervals, that he never saw a second attack; when we recall the fact also that in the widespread epidemic of the War of the Rebellion in this country the disease was almost exclusively confined to regiments from the country towns, sparing the regiments from the cities, whose inhabitants almost never escape attack in childhood,—we must look with credulity upon statements of repeated occurrences. Where the case in question is undoubtedly measles, the character of the first or previous attack should be established beyond doubt.

Measles may certainly coexist with other infections—with scarlet fever, with rötheln, with typhoid fever, and most especially and frequently with pertussis. An unmistakable coincidence is mentioned by Panum, who vaccinated a child in the incubative stage of the disease, both vaccinia and measles running typical courses. With the exception of pertussis, the existence of an acute disease as a rule postpones an attack of measles until after its subsidence. Coincidence is therefore an exception to the rule. An interesting contribution, as illustrating the difference in susceptibility to measles and scarlatina, was

made by Faber and Heyfelder, who showed that during the prevalence of both diseases the convalescents from scarlet fever were frequently attacked by measles, while convalescents from measles were rarely attacked by scarlet fever. Thomas and Gruel made the same observations in regard to measles and rötheln.

The contagiousness of measles is established beyond a doubt, if only by the rapid dissemination of the disease, but experimental evidence has been established absolutely thus far only with the blood and nasal mucus. The disease is disseminated not from the blood, but from some of the emanations of the body. The infectious character of all the secretions has been so often declared and denied that the source of real infection still remains in doubt. It must, however, be something in the nature of a living organism; if only from the fact of the rapid multiplication of the disease. Hallier believed that he had discovered it in certain micrococci; Salisbury in 1862 claimed to have found it in, and propagated it from, a straw fungus. Wood and Pepper were not able to verify these observations. Coze, Fels, and Keating isolated micrococci from the blood; Le Bel, bacilli from the urine; Eklund in 1882, chain micrococci (torulæ morbillorum) from the sputa; and Braidwood and Vacher collected certain spherical bodies upon glycerin slides exposed to the breath of patients affected with measles; they found the same bodies in the lungs, and hence assumed that the lungs evolved the disease. None of these studies were made with modern methods. None of them disclose any other pathogenetic relation to the disease than presence. Lambroso failed to discover any micro-organisms in the blood, though he found a small round coccus in the eruption in the first three or four days. Leyden also saw the same or a similar micro-organism, but with no other definite relation to the disease.

A further contribution to the bacteriology of measles was made by Cornil and Babes in their studies of the pneumonic complication. These observers found in the lungs of children affected with measles large masses of diplococci, distinguished by their biscuit shape and arrangement in pairs. These diplococci accumulate in the infiltration of the interstitial tissue, and are found abundantly also in the lymph- and blood-vessels, less abundantly in the alveoli themselves. Babes says that he was able to isolate from the blood of the papules of measles, as well as from the lymph-glands and pneumonic centres, a streptococcus which showed in its shape and culture great similarity to the streptococcus pyogenes. These micro-organisms are, however, not believed to stand in any genetic relation with measles itself. They cannot be considered as the specific causes of the disease, but are probably the well-known pneumonia diplococci of Fränkel and Weichselbaum and the common streptococcus of pus. They are the products of mixed or secondary infection. They may account for complications, but not for the disease itself.

The real cause of measles remains as yet undiscovered. A difficulty in the way of observation lies in the fact that the disease is not known to occur in the lower animals. It must soon be discovered at least in the blood, where evidence of the existence of the contagium is proven without doubt. In these

days of rapid discovery in the field of the infections the interval between press and publication may cover its period of disclosure. In fact, in this very interval, on this occasion, Canon and Pfeiffer (1892) declare that they have discovered the specific bacillus of measles in the blood and in mucus from the nose and conjunctiva. They used the method successfully employed in the case of influenza—to wit, concentrated aqueous solution of methylene blue 40.0; ¼ per cent. solution of eosine (in 70 per cent. alcohol) 20.0; distilled water 40.0. The preparations are immersed in absolute alcohol five to ten minutes, then stained in the incubator at 37° C. from six to twenty hours. The bacilli thus disclosed vary in size, but were uniformly present in every one of fourteen cases examined. The cause of the disease probably escapes from the body through the nasal mucus, which, dried and infinitely subdivided, floats in and contaminates the atmosphere about—that is, in the close vicinity of—the patient. Mayr certainly succeeded in propagating the disease with mucus from the nose. Mucus collected from a patient in the height of the eruption was conveyed in a glass tube and inserted upon the mucous membrane of the nose of two healthy children living at some distance from each other, some time after an epidemic of measles. In one of these children sneezing set in in eight, in the other in nine, days. Fever followed two days later, the characteristic rash appeared on the thirteenth day, and the disease ran its regular course.

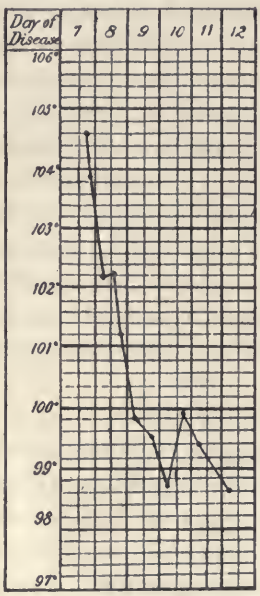
Course of the Disease.—The period of incubation, the lapse of time between exposure and the appearance of the first symptoms, as determined by inoculation experiments and observations by conveyance of single cases to isolated places, is quite definitely established at ten days—that is, fourteen days before eruption. The most indisputable observations were furnished by Panum in the Faröe Islands, so frequently referred to. It was easy in these cases to trace up the source of infection, which corresponded to the landing of a case from a ship. In all these cases thirteen or fourteen days elapsed from the day of exposure to the beginning of the eruption. An almost too perfect case was reported by Myrtle. Measles broke out in a young ladies' boarding-school with thirty-five resident scholars. The girl affected was isolated in an adjoining house, and in the course of twelve days was sent home and the house and everything in it thoroughly disinfected. "Exactly fourteen days after this girl showed the disease a second case occurred, fourteen days after that a third, fourteen days after that a fourth, and fourteen days after that a fifth. Nos. 1, 2, 3, and 5 belonged to different classes and slept in different rooms. Nos. 1 and 4 were sisters and slept together, and No. 4 showed the disease eight weeks after her sister." We may coincide with the author that "comment on these clinical records is needless."

The universal existence of the disease and the infinite sources of infection render accuracy of observation elsewhere almost impossible, hence the variation in time from one week to three given to this period by various authors.

The period of incubation is in the vast majority of cases wholly free of symptoms. Very exceptional cases show malaise or ephemeral fever, which

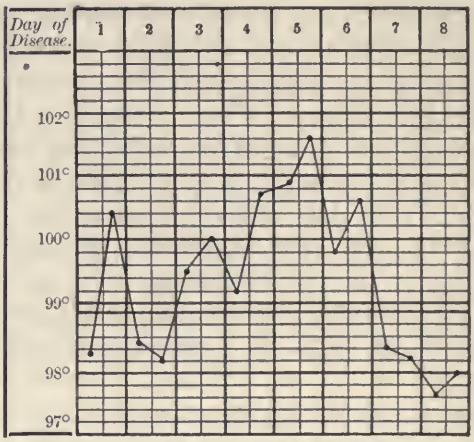
may, but often does not, arise from the poison of the disease. The stage of invasion may be marked by a distinct chill or more commonly by a series of shiverings, to be attended or followed by a rise in temperature to 100°–104° F., with gastric irritation and nervous symptoms in correspondence with the temperature. The fever is in many cases so slight as to be overlooked, when the disease may announce itself with more distinctive signs. After the first remission the temperature again rises with the appearance of the eruption (Fig. 14), to reach its acme at the period of full efflorescence, and to decline as it fades

Fig. 14.



Measles—Defervescence by Crisis.

Fig. 15.



Temperature-Chart of Ordinary Case of Measles.

away. In an average case the fever falls by crisis—that is, within thirty-six hours after the first decisive fall (Fig. 15). During the stage of invasion characteristic catarrhal symptoms show themselves in the mucous membrane of the nose, eyes, throat, and bronchial tubes. These symptoms are summed up under the term "coryza." The eyes grow intolerant to light, the conjunctiva is hyperæmic, the nose "runs," the eyes, nose, and throat itch and burn—sensations but partially relieved by more or less sneezing and cough. The uvula and soft palate now show dark-red spots, and later diffuse redness, the so-called enanthem, the first appearance of the eruption. Bronchitis, the result of direct invasion of the bronchial tubes, belongs to measles as definitely as the eruption.

In an average case the first evidence of affection may be a disturbance of disposition. The child, usually cheerful and animated, becomes listless, indifferent, fretful, feverish; or attention may be first attracted to the child by a sudden fit of sneezing not to be accounted for by any perceptible exposure. Irritation about the nose is further manifested by itching and burning, which the patient may attempt to relieve by manipulation. It is observed also that

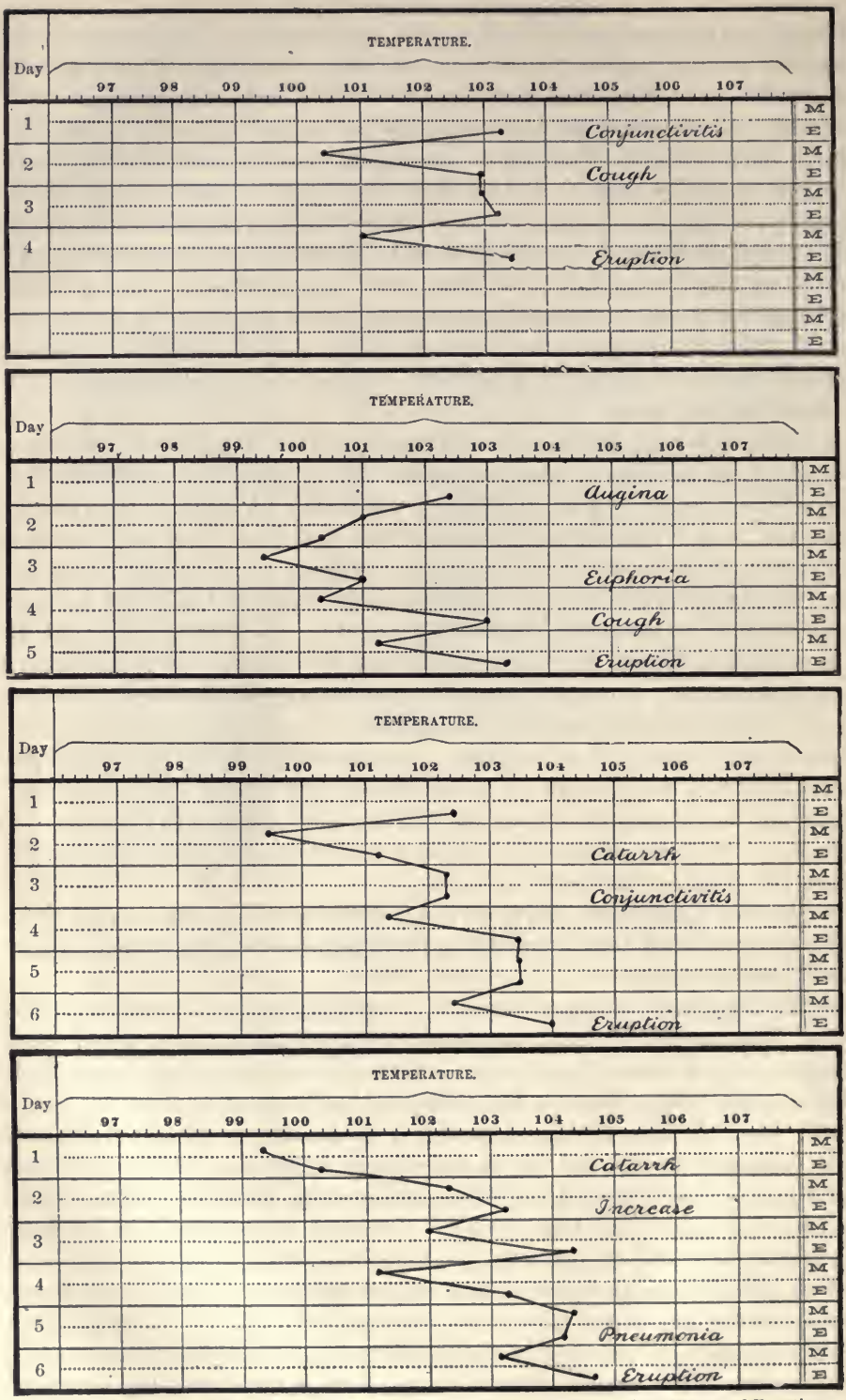
the eyes are reddened and tears flow over upon the face. The dryness of the nose felt at first is soon relieved by a discharge of watery fluid, which may accumulate to trickle down upon the upper lip. There often is complaint of dryness and soreness in the throat, inspection of which frequently at once discloses dark-red spots about the uvula and soft palate, some of which may coalesce to present more or less extensive erythematous discolorations of the surface. There is the same evidence of irritation in the bronchial tubes. The cough is more or less frequent and distressing, and auscultation may reveal an abundance of dry râles thus early in the disease. So long as the bronchitis is confined to the larger tubes all physical signs may be absent. As a rule, these catarrhal signs are obtrusive. They mark the onset of the disease unmistakably as regards the differential diagnosis between this affection and scarlet fever or small-pox.

These signs may, however, be very slight (morbilli sine catarrho), when the nature of the disturbance may be revealed only by careful study of the temperature. It is seen that the temperature rises to 100°–102° F. in the evening, and that it does not entirely subside on the next day. It is, however, as a rule, very irregular during the period of invasion. It may fall to the normal on the second day, to rise slightly on the third, and maintain itself at this elevation or fall again on the fourth or even the fifth day, to meet its characteristic elevation with the appearance of the eruption. The irregularity of the temperature during the period of prodromata speaks thus more definitely for measles, as the temperature of scarlet fever, as a rule, is a continuous elevation up to the period of the eruption. An association of catarrhal symptoms, more especially the presence of an enanthem, with a light rise or irregular course of the temperature during the first few days, announces the advent of measles. The stage of invasion lasts, as a rule, three full days, exceptionally four, still more exceptionally five or six.

The four temperature-charts here reproduced (see Fig. 16), adapted from Henoch (translated into Fahrenheit), illustrate varieties of invasion in perfectly normal measles.

The affection of the upper respiratory tract is a feature of measles so constant as to have been always recognized from the beginning of time. This feature assumes especial value in the colored race, where the discolorations of the eruption proper may be but indistinctly or not at all observed. It is rather the rule than the exception that even as early as the end of the first day, certainly by the end of the second, the hyperæmia which marks the catarrhal process in the throat, more especially the palate, is so intense as to produce the appearance of an eruption. Dark-red spots, varying in size from that of a pin's head to that of a pea, are plainly visible upon the palate and uvula, presenting at times a distinctly spotted appearance. The mucous membrane of the lips, of the cheeks less frequently, occasionally the conjunctiva itself, may show the same spots, the so-called enanthem, which disappears as a rule entirely before the true eruption shows itself on the skin. The enanthem extends also to the deeper mucosæ. Steiner saw it in life in the larynx and

FIG. 16.



Temperature-charts of Four Typical Cases of Measles, from Invasion to Appearance of Eruption.

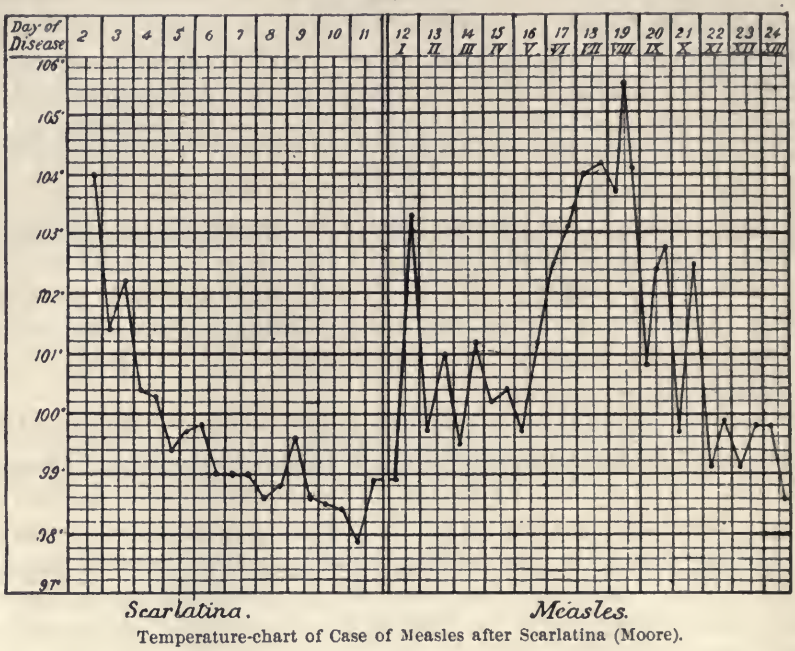
in autopsies deep in the bronchial tubes. Wilson and Ray observed it in the trachea, and Gerhardt on the posterior wall of the trachea. Heyfelder declares that he found an eruption like measles on the mucous membrane of the duodenum; Weber and Lieutaud as far down as the ileum. Fuchs says that the genital mucosa may be covered with the same numerous red spots—a statement which both Henoch and Schomel support.

The eruption proper is seen first, as a rule, on the morning of the fourth day, exceptionally as early as the end of the third or as late as the fifth day. The eruption proper shows itself in "spots" (measles), usually somewhat elevated, dark red, "raspberry" red, or tinged with blue, first upon the forehead and sides of the face. It distinguishes itself, especially upon the face, by coalescence and aggregation into irregular or crescentic patches, with intervening islets of unaffected tissue. During full efflorescence the face seems puffed and swollen. The eruption gradually spreads downward over the neck, chest, trunk, and extremities, to cover the whole surface by the eighth day. More or less confluent on the face and neck, it gradually grows more and more discrete over the trunk, legs, and feet. Reaching the lower extremities, it begins to fade from the face. Desquamation, which is absent in light cases, is furfuraceous as a rule.

The first appearance of the eruption is always, except in the most debilitated subjects, attended with a significant elevation of temperature. The record rises to 102°, 104°, or 105° F., along with the general signs of distress, perhaps even light delirium, heat of the skin, dryness, arrest of the secretions. The temperature rises, the discomfort increases, perhaps as yet the diagnosis may fail to have been established, when all at once at the proper time the eruption displays itself upon the face, often to the relief of the practitioner as well as the parents, if not of the patient himself. It shows itself first, as a rule, about the temples, near the ears, or on the chin in the form of minute, flat papulæ of the size of a pin's head or a pea, usually brighter in color on first appearance. It diffuses itself rapidly over the whole face, neck, and chest, and may indeed extend over the entire body to the toes, within the space of twenty-four hours, being always, however, more confluent in the upper half of the body. When it appears rapidly or spreads rapidly over the face, it usually spreads rapidly also over the body: showing itself but scantily or sparsely about the face, it may take several days to extend over the entire body. This slower progress is observed still more markedly in cases of anomalous appearance, where, for instance, the eruption shows itself first over the chest or back. It may spread irregularly, fitfully, or freakishly over the body, and is then wont to be attended with grave complications. The papules seem to be at first arranged about the hair-follicles or sweat-glands, and aggregate themselves in patches which assume something of half-moon or crescentic outline. Very often the eruption above the surface is so slight that the papular character can be recognized, as Sydenham said long ago, only by the touch. The eruption reaches its height in from thirty-six to forty-eight hours: the period of efflorescence and the height of the fever correspond with its duration.

As soon as the fever has reached its height it begins to fall, and falls rapidly, to terminate, as stated, by crisis within thirty-six or forty-eight hours. This fall of the temperature, with the appearance of the eruption, is so characteristic as to often enable the observer to differentiate measles in cases of doubtful eruption from simulating maladies. (See Fig. 17.) There is the appearance that the

FIG. 17.



Temperature-chart of Case of Measles after Scarlatina (Moore).

body struggled with the disease, and finally succeeded, as the old writers believed, in throwing it off in an eruption. With the discharge by eruption the fever falls. The disease, however, is not really in the eruption. The eruption is probably only a toxic reflex, like urticaria, herpes, etc. The fact is that the fall of temperature is observed, as a rule, before the eruption has reached its full height. When it is on the eve of efflorescence, as if a poison had been voided from the blood, the temperature falls rapidly. So the crisis occurs often within forty-eight hours after the appearance of the eruption, and hence measles, barring complications, is a mild disease. Measles is therefore a little (short) disease. At the end of forty-eight hours after the appearance of the eruption there is in most cases no fever at all, and in many cases actually a subnormal temperature, at least in the morning. So well established is this fact in measles that the persistence of temperature or the existence of an elevation of even one or two degrees on the third or fourth day after the appearance of the eruption betokens a complication which will probably announce itself with its distinctive signs in the course of a day or two. Where records of temperature are faithfully kept complications are thus often announced before they assume distinct proportions.

The clinical history of an average uncomplicated case of measles implies thus ten to fourteen days' incubation—three days' invasion, three days' progress, and three days' decline. The eruption really remains at its height but twenty-four hours. It begins to fade as soon as it has reached its maximum development. It loses its efflorescence by the end of the second day, to show upon the face only pale or lightly tinted spots, while it extends itself over the body in full flush and hue. Light-yellow or yellowish-gray spots are left behind for a few days, which give to the skin something of a marbled appearance. A mild case may desquamate but little. In most cases the desquamation occurs in fine branny dust. Scales of skin are never shed after measles, and strips or lamellæ, such as are removed from the palms of the hands and soles of the feet or from the fingers and toes in scarlatina, are never seen.

Individual cases show every variation of intensity and degree of symptom. It has been remarked that measles is a disease which shows constant phenomena. Measles has always been measles from the beginning of time. There is, however, great range of intensity in the expression of individual symptoms, however uniform they may be in their occurrence. Strong, healthy children are at times affected with a form so mild as to be able to go about continuously and to be scarcely disturbed in disposition. As a rule, however, there is such affection of the conjunctiva as to lead to photophobia and epiphora. On the other hand, burning sensations in the eyes and ocular symptoms may assume such prominence as to call for special treatment. There are always, as stated, sneezing, dryness, and irritation of the nose. Symptoms on the part of the nose, again, may assume such prominence as to lead to hæmorrhage, which may require special procedures for its relief. There is nearly always, in even the milder cases, anorexia, a more or less heavily-coated tongue, some angina, some pain in deglutition. In individual cases the inflammation of the mucous membrane of the mouth and throat assumes distinct proportions. Stomatitis may develop: in cases with bad surroundings even gangrene of the cheeks and noma. Measles is not infrequently announced by vomiting, and in individual cases the eruption extends to lower portions of the gastro-intestinal tract, and announces itself in a severe, profuse, or sometimes bloody diarrhœa. With the height of the fever there is, as a rule, such nervous disturbance as to lead for a few days, or rather nights, to light delirium. Little children easily wander in their minds. Intoxication, which is rarely associated with light fever, 102° or 103° F., may suffice to disturb the intellect of a child. Sensitive and delicate children may show the gravity of the shock of onset in convulsions. With the increase in the fever there is also corresponding increase in the frequency of the pulse (130 to 140) and respiration (30 to 40). So long as this increase corresponds with the elevation of temperature, it excites no apprehension, provided this elevation of temperature occur at the proper time in the history of the disease—to wit, at the period of eruption. Occurring with fever or without it at a later period, it may mean the supervention of very grave complication, especially on the part of the lungs.

Abnormities, Complications, and Sequelæ.—These are of frequent occur-

rence. Either the eruption or the catarrhal symptoms may be absent—*morbilli sine catarrho, sine eruptione*—though some eruption may be seen or found somewhere under close enough scrutiny. Blood-poisoning in the stage of invasion may be so intense as to take life at the start—*rubeola siderans*. Hæmorrhage may show itself in two forms. In the more frequent but less grave form a few or many of the spots become petechial. In the true hæmorrhagic or black measles—*rubeola nigra*—observed only in cachectic cases and degraded surroundings, free hæmorrhage occurs; that is, blood flows from the nose, mouth, kidneys, vagina, or intestines, and vibices and ecchymoses appear upon the surface.

Nervous symptoms may assume prominence: headache is common and at times severe. Invasion, especially in young children, may be marked or marred by delirium, coma, or convulsions. True meningitis is rare. Transitory albuminuria is common, but nephritis is very rare. A very sharp attack is ushered in by anorexia, nausea, vomiting—symptoms which may extend over the period of invasion. Parotitis is very rare, but laryngismus, due at times to laryngeal enanthem, is not at all infrequent. The picture of laryngeal stenosis, sometimes as the result of a true croup (diphtheria), more frequently of a false croup (laryngismus stridulus), sometimes as evoked by a spot of hyperæmia on an exquisitely sensitive surface which may be seen and treated under the laryngoscope, presents itself occasionally in the course of the disease. Noma, a gangrenous affection of the mouth or vulva, is an ugly complication in cachectic cases or under exceptionably degraded hygienic surroundings. It is, fortunately, a rare complication, but measles (Woronichin) is its most frequent cause. Catarrhal pneumonia is the complication most frequent and most feared. It is recognized by rise of temperature, frequency of breathing, increase of cough, dyspnœa, with the physical signs of this disease. Latent tuberculosis is brought to the surface by an attack of measles as a rule. Hyperæmia of the bronchial tubes and glands excites quiescent bacilli to quick and active growth or renders fruitful a soil previously sterile to this disease. The eruption of measles may prematurely disappear—"strike in," at any time, not as the cause, but the effect of complications. This disappearance of the eruption with the development of complication is, however, the exception, and not, as commonly believed, the rule. The eruption runs its course, as a rule, in spite of the complication. Complications are due to the nature of the poison, to the constitution of the individual, not, as commonly believed, to "catching cold," the fear of which interferes with one of the chief means of successful treatment—free ventilation of the sick-room.

It has been repeatedly remarked that measles shows a remarkable uniformity in symptomatology, and has been recognized with the same characteristics from the beginning of time. Measles is the disease selected to illustrate uniformity of type as dependent necessarily upon uniformity of cause; yet individual cases do show anomalies regarding the catarrhal symptoms, the eruption, the fever, complications, etc.

In the first place, the duration of incubation may vary somewhat. Reil

claims to have known of cases in which the incubation lasted several weeks. The eruption varies in every degree of intensity. It has been already noticed that it may appear as early as the third day or as late even as the sixth day, and in its appearance it may show itself almost simultaneously upon the face and trunk, or spread with such rapidity that the interval escapes observation. In certain other exceptional cases it may appear first on the chest or back. It is seen in all cases to show itself in greater abundance and profusion, often also with more marked coloration, on parts of the body subjected to heat or enveloped in embrocations. Surfaces of the body under mustard plasters or poultices show the eruption intensely aggravated in degree and heightened in color. Petechiæ or more extensive hæmorrhagic eruption may occur at any period of the disease. Minute hæmorrhages take place not infrequently in the bluish-red traces of the former exanthem, and may in no way interfere with the further mild course of the disease, and have no more significance of danger than the minute blood-spots seen at times in the palate and pharynx. These exudations have nothing to do with the hæmorrhages which constitute the malignant form known as black measles. They disappear to leave no trace. Where the eruption is very profuse it causes a universal puffiness of the face. It may even close the eyes or block the nares, and at times swell the tissues of and about the ear, presenting some resemblance to the distension and deformity of erysipelas. The skin always lacks, however, the glazed and shining appearance of this disease. There may be usually observed in cases of profuse eruption vesicles scattered about the surface, which assume at times such prominence and profusion as to constitute a form called by the old writers the *morbilli vesiculosæ*. In still rarer cases the vesicles assume sufficient magnitude to constitute blebs or bullæ, an affection of the skin which looks like and is commonly called pemphigus, the so-called *morbilli bullosæ* of the older writers. Occasionally, but more rarely, bullæ appear before the eruption. They may continue throughout its course and protract the duration of the disease. They have a tendency in all cases to aggravate the disease, in that they impart the danger which is associated with burns of the skin, more especially ulceration of the intestine from thrombus. The contracted vessels are literally plugged with glutinous blood-corpuscles (Salvioli). Large vesicles, whose contents may be more or less tinged with blood, are evidence of erosive process, of destructive gangrenous ulceration, and are often associated with dangerous symptoms of collapse. Henoch warns against the confusion of these and similar cases with a coincidence of measles and chicken-pox. Certain cases of varicella show confluent vesicles which may present the appearance of pemphigus. Baginsky saw a fatal case complicated with hæmorrhagic measles.

Of all the affections or complications which pertain to measles, none assumes such prominence as disease of the respiratory tract. The poison—micro-organism—falls upon the upper respiratory passages and is inhaled into the bronchial tubes, to lodge in its course upon the larynx and trachea. Affections of the larynx and trachea, as stated already, sometimes assume

prominence. Even during the period of invasion the cough may assume a ringing character. The epiglottis and the surface of the glottis may show deposits of enanthem. Hoarseness, pain, and dysphagia occur in certain cases. Cough is sometimes so continuous as to harass the patient during the day and exhaust the strength from want of sleep at night. It has at times an exquisitely croupous clang, wholly of nervous or muscular origin, and totally independent of the slightest deposit in the way of false membrane. These symptoms usually disappear with the outbreak of the exanthem: the cough ceases and the voice clears up. In exceptional cases, however, the catarrh of the larynx assumes a more formidable character. Exudation takes place; the epithelial cells undergo transformation; genuine false membrane—that is, croup—develops in the throat, fortunately however, only as a great exception. It must be remembered also that diphtheria itself may coincide with or follow measles. In fact, measles rather predisposes to, or prepares the soil for, the development of diphtheria. In all cases of laryngeal complications the condition of the larynx should be definitely ascertained, when possible, by the use of the laryngoscope.

Bronchitis belongs to measles as an integral factor in the history of the disease. So long as it remains confined to the largest and medium-sized tubes it is unattended with special danger. There is, however, the constant tendency to the extension of the disease, and capillary bronchitis and catarrhal pneumonia are the most frequent and the most grave complications. The mortality of measles is really due to this cause. Catarrhal pneumonia may set in at any stage of the course of measles. Where it begins early it usually delays or disturbs the eruption and leads to its irregular development or disposition. Where it begins late it may actually, though it does not usually, cause the eruption to suddenly disappear.

Catarrhal pneumonia is commonly announced by a more or less rapid rise of temperature, increase in the frequency of the pulse, and rapidity of respiration. Dyspnœa is at times intense. There is pallor of the face, which soon shows signs of cyanosis about the lips. There is rapid play of the alæ nasi. Respiration seems often almost too quick to count—the "breath flies." The pulse cannot keep up with it. Its relation to the pulse must be mentioned later on. The supra- and infraclavicular spaces, the jugulum, the intercostal spaces, the epigastrium, are deeply drawn in with each act of inspiration as if by some powerful internal suction force. The vesicular murmur is drowned under the abundant, diffuse, dry, and moist râles. Bronchial respiration may be sometimes detected, along with dulness to percussion in the lower, occasionally also in the middle, lobes, especially at the posterior inferior aspect of the chest.

Any elevation of temperature after the entire disappearance of the eruption should at once excite the suspicion of broncho-pneumonia. This is the time at which this complication most frequently occurs—that is, during the period of resolution—and the complication assumes gravity in direct correspondence with the age of the patient. In sucklings it is almost universally fatal. The

heart is rarely affected either in the course or sequence of measles, yet cases have been reported of endocarditis, myocarditis, and pericarditis—the last sometimes with suppuration—by Rilliet, Barthez, Bouillaud, and Thomas.

Complications on the part of the digestive system are very frequent. Sometimes stomatitis develops, or various mycoses may occur in the mouth, chiefly in neglected cases. The tongue presents, as a rule, only the furred appearance that belongs to all intense or febrile processes. It very rarely displays that enlargement of the fungiform papillæ so commonly observed in scarlet fever.

As in all infections, the invasion may open with vomiting. Distress on the part of the stomach is, however, much more infrequent in measles than in scarlet fever, from the fact that the toxæmia of measles is so much less. A much more frequent complication is that form of intestinal catarrh which shows itself in diarrhœa. Certain epidemics are characterized by the frequency, and at times the severity, of diarrhœa. The discharges may become so profuse as to lead to rapid prostration, or in some cases tormina and tenesmus with discharge of blood impart a dysenteric character and may lead to rapid collapse. As a rule, however, the intestinal catarrah is light and yields readily to treatment and to time. Nephritis is rare: Kassowitz reported a number of cases. The urine showed albumin, blood, and casts, and there was dropsy in the clinical history. Nephritis is, however, as rare in measles as it is common in scarlet fever. Loeb called attention to the fact that propeptone may be found in the urine of measles. Propeptone (hemi-albuminose) is a mixture, according to Kühne and Chittenden, of four different albuminoid bodies like serum, albumin, and globulin. It is deposited by heat and nitric acid, but, unlike them, only after the process of cooling. Propeptone occurs, however, in so many and such varied diseases as to have, at present at least, no diagnostic value.

Complications on the part of the nervous system are fortunately very rare. In very young or very sensitive children the disease is not infrequently announced by epileptiform convulsions. Headache belongs to the fever as well as to the catarrh. Somnolence, sopor, stupor, light delirium, occur in the height of fever without exciting any especial apprehension. Yet graver symptoms have been recorded. Strabismus, tetanic contractions, cataleptic states, maniacal attacks, have been observed in exceptional cases. It is essential here to bear in mind the relation between measles and tuberculosis. Many of these cases of grave cerebral complication are expressions of basilar meningitis. With its intense hyperæmias, especially in the lungs, measles often awakens quiescent bacilli, and liberates them from the bronchial glands to be distributed over the body. Measles is, in fact, the most frequent exciting cause of tubercular meningitis.

Complications on the part of the organs of the special senses concern more particularly the eye and ear. Measles is often announced by conjunctivitis. Photophobia and pain in the eyes belong among the earliest signs of the disease. Extension of this process to the deeper structures may lead to dangerous lesions: ulcerative keratitis, kerato-malacia, irido-cyclitis, and phthisis

bulbi have been recorded. Tobeitz calls attention to the evil influence exerted by measles upon previous—*i. e.* old chronic or subacute—affections of the eye.

The ear is by no means so frequently affected as in scarlet fever. In many cases, however, aural disease assumes prominence. Cordies considers the affection as a simple catarrh of the cavity of the drum, which is the result, according to Tobeitz, of direct extension of the rubeolar process from the throat through the Eustachian tubes. Otitis media may ensue, with perforations of the membrane of the drum. The aural affections, when they occur, are usually milder and less destructive than those of scarlatina. In his latest report Blau calls attention to the necessity of the early recognition of aural disease in measles. Measles is, he declares, the cause of 2 to 10 per cent. of all diseases of the ear, and of 8 to 10 per cent. of all cases of suppurative otitis media. Affections of the labyrinth, due to the invasion of pathogenic microorganisms in the course of measles, have been studied and reported by Moos. Particulars here belong to the domain of aural pathology.

Any of the various complications of measles may become sequels. They may survive the natural duration of the disease, be protracted into convalescence, or may develop after convalescence has been established. The various affections of the eye and ear, ulcerative processes of the skin, caries of cartilages and bones, as of the nose and alveolar processes of the jaw, set in in certain cases, or the hæmorrhagic diathesis may be imprinted upon a case in the course or convalescence of the disease. Pertussis is very wont to supervene.

The coincidence of whooping cough and measles has long been noticed, and the relation of these diseases to each other is rather intimate. The occurrence of whooping cough in the course of measles or the development of measles in the course of whooping cough intensely aggravates a prognosis which might be, and is, as a rule, naturally mild for either disease alone. These cases are exceedingly prone to the development of more persistent diseases of the lungs. Not infrequently they more directly and quickly take life by exhaustion and collapse.

The sequels at all times most to be feared are broncho-pneumonia and tuberculosis. In a very delicate or debilitated child, especially in every case brought up in the atmosphere of infection, the danger of these diseases is imminent, and the symptoms which announce the advent of either are awaited with apprehension. At any time during the course of the ordinary bronchitis of measles the infection may extend to involve the minuter bronchi and air-cells, and the complication announces itself at times so insidiously as to escape recognition. The increase in the frequency of respiration is, as stated, a most frequent precursor. The respirations increase to 40, 50, 60, or even 80, in the minute—an increase out of all proportion to the rapidity of the pulse. The pulse-respiration ratio becomes 1 to 2 instead of 1 to 4—a much more significant factor in the development of pneumonia than mere increase of frequency alone. The respiration becomes as shallow as short. A child is incapable of sustained effort. The child at the breast must frequently release its hold to breathe. It loses the ability to make a continuous cry. Children

that may continue to nurse uninterruptedly or may utter a prolonged cry have no catarrhal pneumonia. Henoch makes a fine critical comment in saying that "it is a good sign when the child makes the physician wait to hear its respirations in an auscultation of the chest." The physician may become reconciled to the loss of time in this investigation.

Attention must be paid to these factors, because the physical signs of this complication or sequel are so defective. They may often not be dissociated from the signs of finer bronchitis, at least not until retraction of the intercostal spaces and the sinking in of the spaces about the clavicle and the epigastrium indicate occlusion of the lungs. Signs or absence of signs in the islets and tracts of condensation of catarrhal pneumonia are alike drowned under the universal moist and dry râles of diffuse bronchitis. So much more important become the studies of the temperature. Any sustained elevation of temperature after the eruption, or any evening exacerbation of temperature in the course of convalescence, should excite the suspicion of broncho-pneumonia or tuberculosis. The skin often feels hot in these cases. The mother calls attention to the heat of the skin, or the physician is struck by it on application of the hands or more especially of the side of the face in auscultation. Elevations of temperature not so marked, noticed more especially or perhaps exclusively in the evening, indicate the insidious development of tuberculosis; and this indication assumes all the more value in the presence of anorexia and progressive emaciation. The child does not gain strength; it becomes peevish and fretful; toward evening is excitable, difficult to put to sleep, seems disturbed in its dreams, continues to cough, always of course without expectoration, sweats at night, shows later perhaps some diarrhœa and marasmus: this is the history of a developing tuberculosis.

Above all other diseases, as stated already, measles liberates tubercle bacilli from bronchial glands. This is probably the true relation of these diseases. The primary infection is a thing of the past. Penetration to the bronchial glands has been favored by coddling, by the house climate, by various medications, by the administration of cough-mixtures or opiates, under the cover of which the disease has secreted itself in the recesses of the lungs—to wit, the bronchial glands. Measles, with its hyperæmia and its bronchial and pulmonary congestions, irrigates the soil, swells the glands, and arouses dormant or quiescent seed into active life. Ziemssen long ago called attention to the revelations of the laboratory with reference to cervical glands, in that they so often contain tubercle bacilli hitherto quiescent; and the same condition has been revealed of the bronchial glands, which may be called nurture soils of the tubercle bacillus.

Diagnosis.—The diagnosis is easy as a rule. The prevalence of an epidemic or existence of other cases, escape from attack hitherto, are points in circumstantial evidence. Measles is differentiated from a simple catarrh or a coryza by its higher temperature, by the enanthem on the second or third day, and by the exanthem on the fourth day; from hay fever by the period of occurrence and the history of repeated attacks of hay fever, as well as by

the eruptions of measles; from simulating drug eruptions, as from copaiba, quinine, and the various antipyretics, by the history of the case and the immediate supervention of these eruptions without previous coryza; from roseola by the more uniform redness, of lighter color, more limited range, but shorter duration, with the absence of fever characteristic of this affection, if this affection may indeed be specialized. Papular erythema, which may coarsely resemble measles in the face, is distinguished by its localizations elsewhere, upon the forearms and backs of the hands and feet, as well as by the absence of fever, catarrh, and bronchitis.

Measles must be separated from typhus fever. The distinction seldom comes in question, because typhus occurs only in certain places, and is, in general, on the road to extinction. Typhus fever in itself closely resembles a bad case of measles, in that the disease is so contagious, the liability so universal, and in that the eruptions may be, at first at least, much alike. Typhus fever, like measles, begins suddenly, often in the midst of perfect health. There is from the start more profound prostration in typhus, and, with the very inception of the disease, overshadowing symptoms of mental dulness, drowsiness, sopor deepening into stupor, which readily passes over into coma. This is the cloud about the brain which has given the name to the disease. It is present in only the worst cases of measles. Typhus has no exanthem and no catarrh. The eruption of typhus appears on the third day, first upon the chest, to extend thence over the entire body, but to spare always or nearly always the face. A peculiarity in the eruption of typhus fever is the fact that by the third day the spots, which may have hitherto resembled measles, aggregate themselves into points of pin-head size, filled with black blood, the so-called petechiæ. Another very distinctive peculiarity is the fact that the temperature does not fall with the full appearance of the eruption. Disregarding diurnal variations and accidental complications, the temperature of typhus maintains itself at about the level at which it began up to the twelfth or fifteenth day—a duration which is never seen in measles except as the result of obvious complications.

Morbid Anatomy.—The morbid anatomy of measles does not differ much from that of the other exanthematous diseases. What studies have been made concern chiefly the changes found in the skin and the condition of the lungs in pulmonary complications. Neumann found the vessels of the skin dilated and hyperæmic, crowded in the upper portions of the cutis with round cells. Sweat-glands, which were also dilated, were invested in the same way, their coils and ducts packed with thickly-crowded round cells. Round cells accumulated also about the sebaceous glands and insinuated themselves between the muscle-cells in the skin. This inflammatory process distinguishes itself in measles by its more superficial character. The upper layers of the skin were affected rather than the deeper layers, as in scarlatina. Gerhardt and Coyne studied the changes observed in the larynx. They could still discover evidences of catarrhal affection, swelling and thickening, and desquamation of the epithelium, and in some cases suppuration, as in the conjunctiva. Coyne

distinguished the affection of the larynx as an erythematous laryngitis. He found it in connection with capillary hyperæmia and with accumulation of white blood-corpuscles about the glands and vessels. The epithelium had been often more or less denuded, and the interglandular spaces filled with numerous lymph-corpuscles.

Tobeitz, as the result of his investigations of the pneumonic process, observed the disease to start always from the finest bronchi in invasion of the air-cells. The affection differed in no way from the broncho-pneumonia or catarrhal pneumonia originating in the course of any descending bronchitis. The cellular elements exuded are excessively prone to decay. Bartels had made all these observations before. The hyperæmia, with caseous degeneration of the bronchial glands and liberation of their contents, more especially of tubercle bacilli, has already been sufficiently described.

Black measles showed the changes in the blood and parenchyma of organs to be seen in all cases of grave infection, more especially in true typhus.

Prognosis.—The prognosis in general is favorable. The mortality of measles, *per se*, is almost nil. Death seldom or never occurs directly from the disease, but from complications, previous debility, and bad surroundings. Thus, Pott found as the cause of death pneumonia and capillary bronchitis in 21, and croup in 3, of 24 cases. The mortality of the disease in hospital and tenement-house practice is quite different from that of private practice. It is not uncommon to observe a mortality of 30 per cent. under bad surroundings, and the range would be still higher if it included the subsequent cases of tuberculosis which have come to light in consequence of measles. The mortality stands also in quite direct relationship to the age of a child, and diminishes from 50 per cent. under two to 15 above this period. The ravages of the disease among savages, as among our own Indians, were due wholly to lack of sanitation.

Treatment.—Prophylaxis is almost impossible. Sickly, debilitated, more especially tuberculous, children should be removed from infected houses. The liability of infection by third persons and things is by no means so great as in scarlatina; hence the necessity of withholding other members of the family such a length of time from attendance at school and association with others is not so imperative.

The treatment is purely expectant and symptomatic. Full and free ventilation at a temperature of 70° F., a night-gown without under-wear, light but sufficient bed-covers, absolute cleanliness, water and milk *ad libitum*, supply the requisites of treatment for an average case.

Fever above 103° F. is best controlled by warm baths, which may be gradually cooled, or by the occasional administration of phenacetin in doses of from 3 to 5 grains, more especially in relief of associated nervous distress. Burning or itching of the skin is best relieved by warm baths, with subsequent anointment with vaseline or cocoa butter. Photophobia calls for smoked glasses or shading of the eyes in the disposition of the bed or screens, rather than for darkening of the room, an objectionable procedure. A drop or two

of a solution of morphine containing 4 grains to the half ounce or of atropine (1 grain to the ounce) allays any extreme irritation of the eyes; smearing the edges of the lids with an ointment of hydrargyrum oxidum flavum (gr. v. to ℥ss of unguentum petrolei) will usually prevent or cure blepharitis marginalis and keratitis. Simple pure vaseline or boric-acid ointment (gr. xv to ℥ss), snuffed into the nose, will generally relieve the sense of dryness and irritation in the nose and throat. The instillation of hot water or of a drop or two of the solution of atropine (gr. j to ℥j) will often quiet earache. Evaporation from a piece of cotton saturated with chloroform held close to the meatus is often equally effective. Gastric distress and vomiting may require cracked ice, sips of hot water, lime-water, and milk (in proportion of one-third), bismuth (ʒss to ʒj), or chloral (2 to 5 grains), rectal injections of sodium bromide (gr. x–xxx to f℥ij of water), or of chloral (gr. v–x to ℥j of water). Few cases of vomiting from any cause will resist chloral if its absorption can be secured.

Nervous symptoms may call for sodium bromide in doses of 10 to 30 grains largely diluted, or from 5 to 10 grains of chloral or phenacetin suffice for a lighter case. Hæmorrhage and prostration demand alcohol, best given in the form of brandy; black coffee; turpentine, in doses of 5 to 15 drops, briskly stirred in a wineglass of milk; or nitro-glycerin, in doses of 1 drop of a 1 per cent. solution, in whiskey and water; possibly opium (best in the form of the camphorated tincture, in doses of 5 to 40 drops), or codeine, in doses of $\frac{1}{8}$ to $\frac{1}{4}$ of a grain, may substitute morphine for more continued use; carbonate of ammonium, in doses of 5 to 10 grains, in milk; ergotin or preferably sclerotinic acid, in doses of from $\frac{1}{4}$ to $\frac{1}{2}$ a syringeful, may be required. The syrup or wine of ipecac, to which may be added, if necessary, a small quantity of Dover's powder, preferably in the form of a syrup, suffices to restrain any excess of cough. The following is a good prescription for a child in relief of cough:

℞. Apomorphin. hydrochlorat., gr. ss;
Acid. hydrochlor. dil., gtt. x;
Syrup., ℥ss;
Aquæ menthæ piper., ℥jss.—M.
Sig. Teaspoonful every two or three hours.

Diarrhœa requires at first no control. Later, as the discharges become more abundant or colliquative, it may be restrained by bismuth, to which may be added if necessary a drop or two of tincture of opium. An improvement on a time-honored remedy may be written as follows:

℞. Tinct. opii, gtt. xl–ʒj;
Acid. hydrochlor. dilut., gtt. xl;
Aquæ camphoræ, ad ℥iv.—M.
Sig. A tea- to a dessertspoonful every two to four hours.

Broncho-pneumonia calls for stimulation of the respiratory centres as well as of the heart. These centres are best reached by warm baths with cold affusions to the head. A rapid respiration, a quick pulse, cold surface, somnolence, and delirium call for baths and baths, repeated baths with cold affusions, together with the use of the analeptics—camphor, benzoic acid, ether, musk, nitro-glycerin, caffeine, and brandy.

Gangrene of the skin, noma, ulcerative processes, caries of bone, are best treated with caustics, carbolic acid, solutions of corrosive sublimate, the actual cautery, or applications of iodoform. In all these cases alcohol must be administered abundantly.

Cod-liver oil, pure or with malt extract, iron, arsenic, out-door air, fresh air, for the inlander especially sea-side and mountain air, with good food, pleasure, and peace of mind, are the best reconstructives during and after convalescence.

RUBELLA.

By JAMES T. WHITTAKER.

Definition.—A specific, feebly contagious, acute infection of short duration, characterized by the absence of prodromata, the presence of an eruption simulating that of true measles, faucial catarrh, and enlargement of the lymphatic glands.

Synonyms.—Rubella, diminutive of rubeola, from *ruber*, red; Rötheln, diminutive of *roth*, red; German measles, French measles, because described by German and French observers, really first isolated by an English physician; Hybrid measles; False measles, etc.

The word "rubella," which seems to have been first recommended by Veale (1866), soon met with general acceptance. The Germans still call the disease rubeola, our term for measles, which they still call morbilli. The French distinguish it as rubéole, in distinction from rougeole, true measles. The popular designation in Germany is rötheln, a term recognized by scholars everywhere, and as commonly used by writers in Germany as is measles in our country. Rötheln can never be adopted among English-speaking people. The sound of *o* with the umlaut cannot be translated. The name is therefore too distinctly racial for universal acceptance. Rubella means exactly the same thing. No valid objection can be urged against the name rubella as indicating a diminutive of rubeola, and as permitting, however akin to rubeola, the recognition of an independent malady. The universal acceptance of the term varicella, which has a similar relation to variola, establishes a perfect precedent for rubella and rubeola.

Bergen, who described it among the roseolæ in 1752, first maintained the view that it should be separated from measles and scarlet fever, but it was reserved for an English physician, Maton, in 1815 to establish the individuality of the disease as based chiefly upon the observation that, though self-protective, in that one attack confers future immunity, it does not protect against either measles or scarlatina. Nor do these diseases protect against rubella.

There was almost up to the present time—in fact, there is yet—much lack of harmony regarding the true nature of this affection. Köstlein in 1865 still considered rubella a variety of measles. Strümpell, on the other hand, declares that only they who have never seen it deny the existence of the disease as an independent malady. Heim looked upon it as an anomalous scarlatina. Hildebrandt regarded it as an intermediate or hybrid form of measles

and scarlatina—a view which had singular fascination for many authors, including such close observers as Gintrac and Hebra. Barthez and Rilliet, Emminghaus, Gerhardt, Griffith, Hardaway, Murchison, Roger, Steiner, Thomas, Thierfelder, Trousseau, Wilson, all acknowledge the individuality of rubella, while Fagge, Henoch, and Stewart still deny it. It is certain that epidemics of rubella may prevail apart from epidemics of measles and scarlatina. It is also established, as stated, that an attack of either scarlatina or measles gives no immunity from rubella. Again, an attack of rubella does not exempt the individual from attacks of measles and scarlet fever. While more closely allied to measles than to any other disease, it is not hence to be regarded as a subvariety of measles, but as a distinct and separate affection whose cause is *sui generis*. Rubella stands in relation to rubeola not as varioloid, but as varicella, to variola. It certainly differs from both measles and scarlet fever in its contagiousness, mode of invasion, symptomatology, duration, and decline.

Etiology.—Though much less contagious than measles, and hence much less frequent, the disease is decidedly more prevalent than commonly believed. Many cases are mistaken for measles, and most of the so-called successive or repeated attacks of measles are really rubellæ. Rubella is certainly distinctly contagious, and the contagium increases in virulence with the number of cases and with defective hygiene. As to the intensity of its contagion, authorities differ. Nymann, Picot, and Arnold think it but feebly contagious; Thomas and Bourneville regard it as less contagious than measles; Jacobi and Squire consider it eminently contagious, and maintain that the contagiousness is manifest before the appearance of the eruption and persists for several weeks after its disappearance; Atkinson claimed that it is less contagious than measles, and Edwards concludes that it is one of the most contagious of all the eruptive fevers; Griffith states that 37 of 100 children in a "home" which he attended contracted the disease, notwithstanding the most prompt and careful isolation; and Edwards quotes from Hatfield that 110 of 196 inmates of an asylum suffered from the disease. The disease is propagated also by third persons and by things. The bedding of steerage passengers has been known to conceal and convey contagion for a long time.

From the nature of the disease the cause of rubella must be a micro-organism, but the specific structure has not yet been isolated. Micrococci have been observed in the blood, but without any other evidence of positive relationship.

Rubella occurs at all ages, rarely in infancy; 75 per cent. of cases occur before the age of fifteen. Childhood is thus the period of greatest liability, but susceptibility to it is so much less than to measles that the majority of people escape it throughout life. Sholl saw the eruption in a newborn child; Steiner and Roth report cases in infants under six months. These cases are regarded as exceptions. Attacks in adult life are much more frequent than attacks of measles—first, because the susceptibility is not so universal, so that childhood often escapes it; and secondly, because epidemics prevail at much

longer intervals. Adults have, however, immunity in high degree. Kassowitz observed but five cases in adult life. The range of liability in regard to age is illustrated by the possibility of attack in advanced life. Seitz recorded a case in a woman aged seventy-three.

Symptoms and Course.—The period of incubation, two to three weeks, is uncommonly long, while the stage of invasion or prodromal stage, half a day to one day, is uncommonly short. An initial chill is exceptional; malaise, pain in the head, back, or joints; anorexia, rarely vertigo; very rarely more pronounced distress on the part of the nervous system,—more or less immediately usher in the eruption and affection of the mucosæ and glands. Not infrequently the appearance of the eruption, totally unprefaced by any fever, is the first sign of disease. The eruption appears as minute rose-red maculæ, discrete or confluent, "like dark red ink pen-points in white blotting-paper," on the forehead and temples, spreading quickly over the rest of the face, neck, and trunk, to reach its full efflorescence and begin to fade in twenty-four or thirty-six hours. By the third day, as a rule, all signs of eruption disappear without desquamation. Coincident with the eruption is a rise of temperature to 99°–101° F., very exceptionally to 102°–103° F.

Hyperæmia of the conjunctiva, with photophobia and epiphora, of the nasal mucous membrane, with a sense of dryness and irritation, with sneezing or with increased discharge, more especially hyperæmia or visible enanthem of the fauces and pharynx, may precede the eruption during the stage of invasion when it occurs, to coexist with the eruption and remain after it as late as the fourth day of the disease.

Affection of the glands constitutes a much more distinctive feature of rubella. The cervical submaxillary and occipital glands, more rarely also the glands of the axilla, elbow, and groins, become swollen and tender, limiting the movements of the head at times in the swelling and stiffness of the neck. These adenopathies, which exist in 50 to 75 per cent. of cases, disappear entirely in two or three days. Abnormal cases show only an eruption or only affection of the glands.

The reviewer of the literature of rubella is struck with the variety of opinions encountered regarding every feature of the disease. In this regard rubella differs radically from rubeola. True measles has a distinct history and a singular uniformity of symptoms. All competent observers agree in the main regarding the period of incubation, the stage of invasion, the character of the eruption, etc. The occasional abnormalities and irregularities are to be accounted for by the condition of the patient and the character of his surroundings, rather than by any difference in the nature of the disease, expression, order, or sequence of its symptoms.

In rubella, on the other hand, scarcely two observers agree, and the difference at times is so marked as to lead to the belief that different affections are being observed or described. It is questionable if the disease commonly described as rubella be a distinct or single affection. Competent observers, as stated, still maintain it to be a subvariety or hybrid form of scarlet fever,

or more especially of measles. It certainly most clearly simulates measles. The admission of the disease as an entity depends almost wholly upon its independence of measles or scarlet fever. This fact does not exclude the existence of more than one malady.

Thus the period of incubation has been fixed in its description at two or three weeks. Griffith, Glaister, Sholl put it at five to ten days, Steiner at ten to fourteen days, Jacobi at fourteen to twenty-one days, Cotting at three weeks. Edwards fixes it at certainly between ten and twelve days: the shortest period recorded in his experience was six days, the longest twenty-one.

In the observation of the author the stage of invasion has been always free of symptoms. Grave symptoms have, however, been remarked by others. Smith saw convulsions; Hardaway, delirium; Prioleau, hæmorrhage from the eyes and ears; Nymann, vertigo; and various eruptions have been noticed by others.

The disease is announced generally by the eruption, which appears so quietly at times as to be noticed for the first time in the morning on awakening from sleep. It shows itself first upon the face, and spreads, as a rule, so rapidly over the body and extremities as to seem to show itself everywhere at the same time. Patterson indeed declares that it comes out universally. Occasionally it is very sparse and circumscribed. Edwards says that he has seen it confined to a small part of the brow, face, and neck, and so scanty as to have made a diagnosis impossible had it not been for the presence of other cases. It is usually entirely discrete, but becomes confluent at times on the face or upon surfaces kept warm by apposition, as in the flexures of the joints, about the groin, etc. As with all the eruptions, it is more pronounced under hot applications, poultices, embrocations, etc. Griffith says that he saw it once in circular bands about the leg above the knee, in the line of the garter. Klatsch made a similar remark.

The character of the eruption differs in every particular in the description of different authors. Heim gives it the color of red ink on white paper. Thomas declares that it is not so red as that of scarlet fever, nor so blue as that of measles. It is usually entirely macular. Aitkin declares that it is more elevated than measles. Griffith felt induration like shot under the skin. Cases have been described as so closely simulating measles or scarlet fever as to have justified the designations rubella morbilliforme and rubella scarlatiniforme. Harrison, Copeland, and Goodhardt claim that it may resemble either measles or scarlatina. Byers, Picot, and Henderson saw cases where the eruption was morbilliform in one part and scarlatiniform in another part of the same patient. Dukes and Kassowitz declare that it may resemble measles, and Murchison and Tonge-Smith declare that it simulates scarlet fever. These statements are from Edwards, who made an exhaustive study of the authorities, and adds: "This list could be almost indefinitely prolonged, but to no purpose. Sufficient has been cited to show that the eruption of rubella is indeed multiform in character."

The eruption disappears, as a rule, in twenty-four to thirty-six hours:

Emminghaus says in from two to four days; Klaatash, in from one to five days; Liveing, in from five to seven days. The eruption disappears, as stated as a rule, without, or with but very slight, desquamation. When present, desquamation is always furfuraceous. Sometimes it is best marked in the throat.

Slight fever to 100°–101° F. is the rule. Exceptional cases show high temperatures. Haig-Brown recorded 105° F.; Davis, 106°, with a hæmorrhagic eruption and convulsions; Wunderlich declared that many cases show no fever at all; and Griffith reported a case of extensive eruption marked by the complete absence of fever.

Sore throat, faucial catarrh, is observed in the majority of cases. There is perhaps, more unanimity of opinion upon this symptom than upon any other. Hyperæmia of the throat shows itself sometimes in an enanthem like that of measles. The infection may also involve the larynx, and occasionally the bronchial tubes.

The most characteristic symptom is affection of the lymphatic glands. Few writers—among the most noted Kassowitz—failed to mention its frequency. The distinguishing feature of the adenopathy is the universal involvement of the glands. The cervical, occipital, submaxillary, and sublingual glands are often all involved. Park found distinct adenopathy in the neck and under the tongue in 50 per cent. of his cases. The affection may extend so as to involve, as stated, the axillary and even the inguinal glands.

The diagnosis is made to rest largely upon this extensive implication of the lymphatic glands, measles rarely showing any such affection. Scarlatina shows it as a rule, but the swelling is confined almost exclusively to the glands and interglandular tissues below the jaws. Scarlatina never, or almost never, affects the cervical and post-cervical glands. Few observers would, however, be prepared to go so far as Osborn, who claims as a pathognomonic feature of rubella—one so constant in its occurrence, he says, that when observed there can be no longer doubt—"an enlargement of the small glands just about the edge of the hair on the postero-lateral sides of the neck." This feature was never absent in any case which he saw.

Gastro-intestinal disturbance corresponds in severity rather with the fever than with the eruption. It is usually absent or, if present, but trivial and transitory. It is a rare case which shows the intensity of disturbance not infrequently seen in measles and observed as a rule in the inception of scarlatina. The "strawberry tongue" of scarlet fever is never seen.

It is commonly said that rubella has neither complications nor sequelæ. This is true, however, only of the average or milder case, especially as observed in private practice under favorable hygienic conditions. In hospital and tenement-house practice complications are not so rare, though they are by no means so common as in measles.

Bronchitis may become excessive. Edwards saw pneumonia three times, Griffith twice in 150 cases. Stomatitis, intestinal catarrh, icterus, rheumatism, various eruptions, including pemphigus, have been remarked in individual

cases. Sequels of diphtheria, mumps, blepharitis, keratitis, and otitis, to be found in the records, must be looked upon as accidental. The light disturbance produced in the lungs is evidenced by the rarity of subsequent tuberculosis as compared with the history of measles.

Relapses and recurrences are very rare.

Diagnosis.—As a rule, the physician is summoned to distinguish the eruption from that of measles. The eruption of this disease, as stated, appears earlier, often without any previous disorder; is lighter in color—a rose not a raspberry red; is more frequently discrete, or when confluent more diffuse, not aggregated into patches; disappears completely without or with but slight desquamation in one to three days. These features, in connection with the more pronounced implication of the throat and the glandular affections, sufficiently distinguish the disease.

Rubella is distinguished from measles, the only affection with which it is likely to be confounded, by the history or absence of a previous attack of measles, by the existence of other cases, by its feebler contagiousness, longer incubation, shorter invasion, hence earlier appearance of the eruption, absent or but light or limited affection of the mucosæ, more frequent and extensive adenopathies, more trivial fever, and shorter duration.

Rubella is distinguished from scarlatina by the history of the individual, as stated above; by the longer incubation—two to three weeks in rubella, one day to one week in scarlet fever; by the characteristic intense sore throat of scarlet fever in contrast with the trivial catarrh of rubella; by the violence of the invasion of scarlatina—vomiting, hyperpyrexia, often delirium and convulsions, in scarlet fever, all absent in rubella; by the more universal affection of glands in rubella, more intense inflammation and tumefaction of the submaxillary glands only in scarlet fever; by the appearance of the eruption, first upon the face or universally in rubella, first on the chest and neck with slower spread in scarlet fever; by the disappearance of the eruption in one to four days in rubella, in four to six days in scarlet fever; by the disappearance of symptoms with the appearance of the eruption in rubella, by the persistence of symptoms during the eruption of scarlet fever; by the strawberry tongue of scarlet fever, absent in rubella; by the albuminuria and affections of the kidney in scarlet fever, absent in rubella; by the desquamation, membranous in scarlet fever, absent or furfuraceous in rubella.

The roseola, adenopathies, and sore throat of syphilis could not, on account of their persistence, be long mistaken for rubella, even in the absence of all history of primary infection.

The diffuse erythemata of drug eruptions—antipyretics, copaiba, chloral, etc.—have the history of their use, and are unattended by fever, sore throat, or affections of the glands.

Prophylaxis.—Inasmuch as most people escape rubella, isolation of cases in a separate room or story of the house is, when practicable, advisable.

The **mortality** is almost *nil*. In this regard the disease has, however, the same history as measles. Bad surroundings may impart great gravity. Hos-

pital and tenement-house practice furnishes a mortality of 3 to 10 per cent., due almost wholly to complications, chief among which are capillary bronchitis and broncho-pneumonia.

Treatment, which is for the most part superfluous, does not differ, when necessary, from that of measles.

SMALL-POX.

By JAMES T. WHITTAKER.

Synonyms and Definition.—Small-pox or pocks (pock, a bag or sac—*i. e.* small sacs); Variola, from *varus*, a pimple, a term applied in ancient times to many eruptions, first limited to small-pox in the epidemics of France and Italy, 570 A. D. (Hirsch), first used by Constantinus Africanus, 1080 A. D. (Curschfeld); German, Pocken, Blätter (blister); French, Petit vérole,—is a highly contagious, extremely dangerous, literally dreaded disease, characterized by violent onset with severe chill, excruciating pain in the back and head, by an eruption of papules, subsequently converted into vesicles and pustules, which leave in drying disfiguring pits or scars, and by a fever which remits at the period of papular efflorescence to increase in the stage of suppuration.

Small-pox has existed from time immemorial in India, where temples were built and a goddess worshipped, and where, more to the purpose, the Brahmins practised inoculation in protection against it. Accounts of it in Africa date also from the most remote antiquity, and the great susceptibility of the negro race lends color to the view that the disease may have originated in these lands. It was imported into China probably about 200 A. D. Galen speaks of the prevalence of it in Rome, 160 A. D.; Marius, of its invasion of France and Italy, 570 A. D.; Gregory of Tours, of its epidemic occurrence in a large part of the south of Europe, 580 A. D.; and Rhazes wrote his famous work concerning it in 900 A. D. Rhazes declared that while the disease had received frequent mention in antiquity, up to his time "there had not appeared either among the ancients or the moderns an accurate and satisfactory account of it," and therefore he composed his discourse. Rhazes certainly saw small-pox and described its most striking features, especially in distinction from measles.

Small-pox entered England in 1241, Iceland in 1306, but did not reach Germany and Sweden until toward the close of the fifteenth century. It was imported to America first in the West Indies in 1507, exterminating whole races of natives; next by Spanish troops into Mexico in 1520, where it carried off three and a half millions of people. In the United States it reached Boston from Europe in 1649, and, though decimating the Indians in every direction, made but slow progress and limited ravage among the white races because of the introduction of vaccination in 1799, the period of commencing Western migration. Thus it did not reach Kansas until 1837 and California until 1850. Epidemics in South America, first in 1554, corresponded with the intro-

duction of slaves from Africa. Certain islands of Polynesia remain as yet exempt.

Small-pox has now only historic interest. It is on the road to extinction, and may occur in our day in epidemic proportion only in uncivilized lands. The most modern text-books of medicine, if they describe it at all, dispose of it, as of the pest and other plagues of ancient times, in but few words. Small-pox, as we see it, occurs in the modified form known as varioloid. Cases of true variola become rarer and rarer every year. Since the general introduction of vaccination small-pox has lost all its terrors for those who recognize its absolute protection. In many parts of Europe small-pox patients are no longer isolated in pest-houses, but are received into the general wards of hospitals, other inmates being protected by, if necessary, fresh vaccination. The dreadful character of the disease in former times is evidenced in our day in no way better than by the fear inspired, the panic created, by the knowledge of the existence of a case in a community. Watson said of it—the disease may not be studied without reference to the old masters—"The horrible aspect, disfiguring consequences, and fatal tendency are so strongly marked that its appearance has always been watched with affright by mankind in general, and with intense interest by the philosophic physician.".

The havoc which the disease has made in the past is apparent in the holocaust effected in Mexico and in the veritable slaughters in India. In the two years as late as 1874–75 half a million people in the presidencies of Bombay and Calcutta alone fell victims to small-pox. In 1865, 7000 natives died in less than two months. It constituted 7 to 9 per cent. of the total mortality in England in the seventeenth and eighteenth centuries, and nearly 9 per cent. of that of the city of Berlin in 1783–87. In France during the whole of the eighteenth century 3000 people died annually of small-pox. Whole races of men were carried off in Brazil, one-third of the population in Iceland in 1707, two-thirds of that in Greenland in 1734. It is computed of the century preceding vaccination that fifty millions of people died in Europe of small-pox. The human race was beaten down until men became resigned to the disease. Macaulay called it the most terrible of all the ministers of death. The danger to life and disfiguration of the living, especially loss of sight, made it, to a degree of which we can have now no conception, the most dreaded of all diseases. "There is no contagion so strong and sure as that of small-pox," Watson writes, "and none that operates at so great a distance."

Etiology.—Susceptibility to small-pox is almost, though not quite, universal. The extent of immunity is difficult to establish in our day because of the protection of vaccination, but it was recognized in ancient times that certain individuals who came in close or repeated contact with the disease remained exempt from attack. Three distinguished physicians, Morgagni, Boerhaave, and Diemerbroeck, were said to have enjoyed this immunity, and Diemerbroeck was so struck by it in his own person as to have been led to believe that the disease was but feebly contagious. The common English name is said by one writer to have been derived from the fact that it attacks the

small. This is true, but it is rather evidence of universal susceptibility. Though it spares no age, small-pox is essentially a disease of childhood, "interrupted and postponed by vaccination." Of the newborn, one-third died before their first, one-half before their fifth year of life (Werner). Old synonyms of the disease (Kinderpocken, Barnkoppen) attest this fact. Of 622 persons who died of small-pox in Kilmarnock in 1728–64, 508—*i. e.* 92.2 per cent.—were five years of age and under; 7 only were over ten years of age, and the oldest was but twenty-six.

This exemption of maturity and age was, however, due, in some degree at least, to immunity secured by former attack. Accurate statistics disclose the fact that the disease occurs at all periods of life, even up to the advanced age of sixty and seventy, and in proportions at these times which nearly correspond to the number of people alive at this period of life.

Sucklings enjoy some immunity. Liability grows intense at the end of the first year and continues up to forty, when it becomes less marked. Pregnancy and the puerperium rather invite than repel the disease. It may certainly attack the fœtus in utero after the fourth month, and children have been born in every stage of the disease. The greater liability of these periods is counterbalanced in man by his more frequent exposure, so that sex shows no real difference.

Allusion has been made to the frequency and severity of the disease in negroes. This fact has been noticed not only in their own country, but in all lands to which they have been carried. The more frequent disfiguration among the colored race, which may be seen upon the streets, is due partly to this cause, but chiefly to neglect of vaccination.

One attack confers immunity for the future, with occasional rare exceptions, as does also one successful vaccination, with more frequent exceptions. The lightest attack protects, as a rule, for life. This fact was proven by the results of inoculation, "the mother-progenitor of the beneficent vaccination." A second attack, if it occur, is usually, but not always, milder. Louis XV. of France survived an attack at the age of fourteen, but died of one at sixty-four. Aitken quotes a case reported by Roupel of three attacks, a lady of M. Guinnet, who had it five times, a case by Matson of seven attacks, and one by Baring, a surgeon attacked on every attendance upon a case.

The existence of other infections gives comparative immunity only during their course. The chronic diseases of the heart, lungs, kidneys, etc. do not diminish liability. It has been found to coexist with other infections—scarlatina, measles, and pertussis. Epidemics occur more frequently in the colder seasons, partly because of the closer contact of people at this time, partly because of the concentration of the contagium in less-ventilated rooms. Boerhaave, who himself never contracted the disease, first established its development by contagion.

The contagious principle of small-pox certainly exists in the skin, whence it is disseminated about the body of the patient. Inoculation was formerly practised wholly by the matter of the disease in the skin. It was the custom

in China in the most ancient times to introduce the crusts of small-pox matter into the nose in the process of inoculation, and in India to rub the matter on an abraded skin. The fact of infection of the fœtus, which is undeniable, proves that the poison exists in the blood. There is, however, no proof of the existence of the poison in any of the various secretions or excretions of the body. Experiments made upon man date altogether from ancient times. These experiments with the secretions gave negative results. Doubt even had been thrown upon the infectiousness of the blood until Zülzer succeeded in communicating the disease to a monkey with the blood of a variolous patient.

The contagious principle has singular tenacity of life. It sticks especially to bedding and clothing, which, if kept secluded at a warm temperature, may remain infectious for months and even years. The body and bedding of a patient affected with small-pox is surrounded by the infectious matter as by a cloud or halo. In a large, well-ventilated apartment the danger of infection on account of dilution and diffusion of the poison is much reduced. It is certain that the disease has been contracted by an individual who has approached a patient no nearer than three feet, and it is well established that the disease may be conveyed by third persons and by things. The contagion is given off from the body at all periods of the disease, and also for some time after death, at least up to decomposition, but not so long as to account for the cases recorded by Dr. Franklin, when he relates that "several medical men who assisted in London at the dissection of a mummy died of a malignant fever, which it was supposed they caught from the dried and spiced Egyptian."

The contagious principle or cause of the disease has not yet been isolated, the micro-organisms discovered being only those of pus. We have to remember in this connection that the same statements were made for a long time regarding erysipelas and influenza, whose micro-organisms turned out to be quite different or to have different properties from those of pus. To speak only of the latest studies, Weigert found in the pustules (see Fig. 18) the streptococcus pyogenes, which Garré ascribed to mixed infection, and Guttmann found in cultures from pustules the staphylococcus pyogenes aureus and the staphylococcus albus. V. Loeff claims to have developed in sterilized tubes from fresh matter amœboid proteids, and Pfeiffer claims to have discovered as constantly present in the exanthem of variola a parasite of the species protozoa, which runs its whole course of development in the body of man or other mammal. This parasite is a cell of round or oval form, 33 μ long by 24 μ broad, with-

Fig. 18.

Capillary of Skin, stuffed with Micrococci (Zuelzer).

out cilia or means of attachment or opening, and enveloped in a smooth membrane. Motion is present only in its early amœba-like stage, and reproduction occurs in the budding of spores resembling micrococci. Pfeiffer found this parasite in the small-pox of man as well as in genuine cow-pox, also in that of the hog, cow, horse, pig, and goat. Vaccine matter, especially animal matter, contains fully-developed protozoa as well as spores. Judgment is reserved as to the relation to the disease of this parasite, which is studied best in hanging drops. Smaller structures, proteids and amœbæ, were found by V. der Loeff, in great number and much variety of form, in matter from pustules as well as from fresh animal matter examined in hanging drops. Colored with fuchsin, they may be studied also in cover-glass preparations. Garré thinks he discovered the cause of failure of detection of characteristic micro-organisms in the blood in the fact that investigations had been made at too late a period of the disease. Bowen states that he discovered nodules of reticular structure, with subsequent surface pits like those of the skin, in the internal organs—liver, kidneys, lungs, and testes—but without a trace of any organisms. Weigert made the same observations, and Chiari found similar forms in the testes. Bérard long ago pointed out an orchitis, and more rarely an oöphoritis, as complications of small-pox. Protopopoff examined this lesion microscopically, and discovered in it three zones—a central total necrosis, a middle zone with small-cell infiltration, and a peripheric zone with exudation. He hoped to be able to isolate the variolous principle in these studies. He examined 6 cases in boys and made cultures in glycerin agar, finding in all 6 cases a streptococcus whose macroscopic and microscopic appearance resembled that of the streptococcus pyogenes. Bowen, Garré, and Hlava reached the same conclusion. Inoculation of this streptococcus in animals showed in the case of rabbits that it possessed no pathogenic properties—an additional confirmation of the view of Koch and Schultze that our present methods will not suffice to discover the virus of variola.

The contagious principle or cause of the disease is disseminated, as stated, from the surface, not from the secretions, throughout its whole course, including the period of incubation, also for some time after death, in greatest intensity with the maturation of the vesicles, so that infection is brought about both by direct and indirect contact, and the contagion may remain active, more especially in clothing, bedding, etc., as stated, for a long time.

Proof of the transference of the disease during incubation was offered by Schaper in the case of an individual who had particles of skin engrafted upon an ulcer. The particles were taken from the amputated arm of a man during an unsuspected period of incubation of small-pox. The patient who received the grafts was attacked by variola on the sixth day after the operation.

Course of the Disease.—The period of incubation varies from ten to fourteen days. The fact that the disease occurs at such intervals and announces itself with such marked symptoms renders observation of this period easier in small-pox than in almost any other disease. It is usually

easy to fix the exact period of incubation of a case by recall of the exact moment of exposure. To be of value in fixing this period the exposure must have occurred, of course, but once and for a very short time. Exact results acquired in this way fix the period of incubation for ordinary exposure at from ten to fourteen days. Thus, Bärensprung saw seven cases all infected from the same source on the same day. In every one of them the outbreak occurred between the thirteenth and fourteenth day: some of them had been vaccinated and some had not. The introduction of the poison directly into the blood is followed by symptoms sooner, as the period of incubation after inoculation is but six to seven days. There is during this period, as a rule, no disturbance in the general health. The individual is unconscious of the fact that he has become the victim of a loathsome disease. In very exceptional cases there has been noticed malaise, a sense of languor, and sometimes pharyngeal catarrh. But Curschmann, with the most painstaking investigations, could discover these signs in but 11 of 1000 cases—*i. e.* less than 1 per cent. The character of the symptoms which may show themselves in the incubation has no prognostic value.

Invasion is ushered in by a chill, which is, as a rule, violent, with rise of temperature to 103°–104° F. on the first day. Prostration may be pronounced from the start. The patient is put to bed, or if on his feet staggers as if drunk. Anorexia, vomiting, jactitation, insomnia, and severe headache set in at once. Above all other signs, pain in the loins assumes prominence. It accompanies the fever from the start, and subsides only with its fall at the appearance of the eruption on the third day. Persistent pain in the back (sacrum), peculiar in its intensity, constitutes the most characteristic symptom of this stage; unfortunately, it is present in but little more than one-half of all cases.

The initial stage of invasion—*i. e.* the period from the chill to the outbreak of the eruption—lasts, as stated, three days. If there be any variation from this duration, the stage is rather shorter than longer. It may be very much longer in the most grave form, known as variola hæmorrhagica or purpura variolosa. The chill which announces the invasion has been characterized as violent. In this regard small-pox associates itself with malaria, pneumonia, and meningitis (epidemic). There may be, however, every variety of intensity of rigor, or the single severe shock, which is marked by a chill, may distribute itself over a longer time in several or a succession of chills of lighter intensity. The temperature, which reaches, as a rule, 103°–104° F. on the evening of the first day, may continue to rise to reach 106° or even 107° F. by the time of the appearance of the eruption. The pulse, which runs up to 100–120, in women 130–140, and in children 150–160, usually corresponds pretty closely with the temperature. Respiration increases in ratio more frequently than the pulse—to such degree in some cases as to constitute dyspnœa, probably from direct action upon the respiratory centres. Gastric distress is usually a prominent feature in the onset of small-pox. Vomiting may be so severe, especially in grave or hæmorrhagic forms, as to constitute a very

serious symptom. Constipation is the rule, though diarrhœa is not infrequent in childhood.

All these symptoms indicate the onset of a grave infectious disease. There is, however, in no one of them anything especially or absolutely characteristic. Stress must be laid now upon the two symptoms which early assume prominence, and which more distinctly bespeak the character of the disease. The most frequent, if not the most distinctive, of these signs is headache, which is, as a rule, present from the start. It sets in with the chill or occurs in a few hours in the course of the subsequent fever. It distinguishes itself not only by its frequency, but by its severity. It persists also throughout the whole period of invasion, to become milder only with the outbreak of the eruption. The physiognomy of the patient, the flushed, bloated face, bounding vessels in the neck, suffused conjunctivæ, expression of pain, make the headache manifest.

Pain in the loins is common to all acute infections, and assumes prominence in correspondence with the gravity of the disease. With the first records of small-pox the oldest writers laid stress upon pain in the back. Rhazes certainly appreciated the import of pain in the back. Thus: "I have found the peculiar symptoms of the small-pox to be a continued fever, pain in the throat, and in the beginning of the fever pain in the back." Again: "If a person has a pain in the back without any other symptoms of the disease, ... he is going to have the small-pox, and, in short, there is no more characteristic symptom of the small-pox than pain in the back with fever, so that when you see this pain . . . . you may be sure that the small-pox is about to appear rather than the measles, for the measles are not attended with pain in the back." Always severe, it is milder in the lighter cases, as in varioloid, and assumes especial intensity, to become at times excessive in severity, in the worst, hæmorrhagic, cases. Excruciating pain in the back, with hæmorrhage free or subcutaneous, should at once excite suspicion of hæmorrhagic small-pox. It usually sets in early, persists, and like the headache remains up to the period of eruption. It is, however, not so universally present as the pain in the head.

Severe symptoms on the part of the nervous system belong to bad cases, and occur with especial frequency in childhood. The disease is sometimes announced by convulsions, attacks of syncope, and occasionally by coma. Adults as well as children may actually succumb to the force of the initial shock, though fulminant forms are not so common in small-pox as in scarlet fever.

Evidence of infection of the mucous membranes shows itself early. There may be sometimes seen spots upon the fauces, especially upon the soft palate. Coryza with photophobia and epiphora may be so marked as to simulate measles. Bronchitis is not so frequent.

Inasmuch as the eruption proper does not appear until the third day, especial value is attached to two rashes of earlier occurrence in certain cases or in certain epidemics. One is petechial, the other erythematous.

Petechiæ may appear on the second day in the form of a fine macular or spotted eruption in the space known as "Simon's triangle," whose base is at the umbilicus, apex at the knees. It may occur elsewhere, especially in the space under the axillæ. The erythematous eruption has its favorite spot on the sides and inner surfaces of the legs from the ankles up, sometimes in women about the nipples. This eruption indicates a mild case of the disease, whereas petechiæ have no such prognostic value.

Petechiæ should never be mistaken for the true hæmorrhagic eruption, which may stamp the disease from the start or occur at any period later. Both these eruptions disappear, as a rule, in twelve to twenty-four hours. They may last longer, and they may, especially the petechiæ, leave behind them slight brownish discolorations.

The older writers, more familiar with the symptomatology of small-pox, admitted the possibility of termination of the disease at this stage. These are the cases of lightest possible infection, either by reason of natural insusceptibility or acquired immunity, as by inoculation or vaccination. These are the cases of so-called "variola sine eruptione." Absolute proof of the character of the infection is offered in the universally quoted cases—one is enough for proof—of the birth of a fœtus in any stage of eruption from a mother who showed signs only of the stage of invasion. Additional evidence is offered in the fatal hæmorrhagic form which steps in to shut out the true eruption. Subsequent eruption is final proof.

The distinctive feature of small-pox is the true eruption. The symptoms hitherto described, the severity of the chill, the rapid and profound prostration, the vomiting, the pain in the head and back, should excite the suspicion of the development of the disease; and these symptoms present themselves in the nature of almost absolute evidence in the presence of an epidemic. They may, however, any or all of them, be present in many of the grave acute affections. Occurring in an isolated and individual case, they could not in their *ensemble* be relied upon to declare the diagnosis of small-pox. The initial rashes furnish more convincing proof. This fact is not so true of the erythematous as of the petechial form. Erythema is too often an index of mere reflex disturbance. Petechial eruption, or that particular petechial eruption which early in the history of disease shows itself in, and is confined to, the base of the abdomen and the inner aspects of the thighs (Simon's triangle), is surer testimony. Diagnoses have been made upon these symptoms alone, and cases have been recorded where the disease, as stated, cut itself short at this period, and subsequent exemption was secured. Petechiæ elsewhere furnish no necessary evidence of small-pox. These eruptions, both the erythematous and the petechial, are often entirely absent. They occur only in certain individuals and in certain epidemics. When present they are often overlooked. The nature of the disease is therefore only finally and fully declared by the true eruption which shows itself on the third day of the disease.

The eruption of small-pox is peculiar. It differs from that of all the acute infections. While it may show resemblance at first to the eruption of other

diseases, it soon assumes changes which distinguish it. The eruption of small-pox runs through successive phases of development. It is at first papular, then vesicular, then pustular. The pustules dry to form crusts, which fall to leave most characteristic scars. These phases of development may be simulated to some extent by varicella or by syphilis, but there is always something in the character, conduct, or course of the eruption over the body which enables even the superficial observer to separate them as a rule.

In its very first appearance the eruption is purely macular—that is, not elevated above the surface. In the course of the very first day, however, so intense is the inflammation, the macule is thickened to become a papule, so that, as a rule, with its first recognition it seems lifted above the general surface. It shows itself first on the face and scalp, where it is unfortunately always worst; over the forehead and temples, then upon the sides of the nose, about the lips, over the chin, and spreads thence downward in quite regular progression over the body. Surfaces rendered hyperæmic, as by poultices or mustard plasters, show more profuse eruption. The hands and fingers furnish the next most favored surfaces. The eruption disappears upon pressure, yielding to palpation a sense of hardness as of shot under the skin. By the end of the first day, as stated, it becomes elevated, and by the third day is distinctly papular. It is always discrete at first. By the sixth day the papules contain fluid; they become vesicles and protrude like half peas. These vesicles are peculiar in showing later a central depression or umbilicus, which is most marked just before the vesicles change into pustules. The depression is explained in this way: The vesicle is not a single sac. It is reticulated—*i. e.* many-celled—in structure, so that puncture evacuates only part of its contents, and the bands which form the reticula hold down the surface more firmly at one point, perhaps the site of a hair-follicle, sweat-gland, or firmer strip of connective tissue. Effusion takes place between the upper and lower layers of the epidermis with the dissolution of these bands. In three days more the umbilicus disappears, the vesicle becomes a pustule, which is full, round, and large; the half becomes a whole pea. With the coalescence of pustules dividing walls are broken down, dissolved, and eroded. The eruption becomes confluent. The contents of the pustules now escape, and, becoming inspissated and decomposed, cause the peculiarly repulsive appearance and odor of a small-pox patient. Desiccation of unliberated pus to form crusts begins in three days more. In this process the more fluid central portions evaporate first, to reproduce the umbilicus. The crusts fall in about fifteen days, leaving scars or pits the result of necrosis of epidermic cells. Hyperæmic at first, the scars grow gradually lighter in color and more contracted in circumference than the surrounding skin, until finally they remain as disfiguring white spots with radiating lines for years in childhood or for life in adults. A peculiar deformity ensues at times about the alæ of the nose, with notching of the free borders, and distortions as from extensive burns are occasionally seen about the face. Every possible lesion of the eye up to complete blindness and destruction of the globe is also seen. A stroll upon the streets in pre-vaccination

days, when these accidents were to be observed at every step, would, were it possible, do more to dissipate the folly of the opponents of vaccination than mortuary statistics. These "anti-vaccinationists" are not as wise as the pirates who knew that dead men tell no tales.

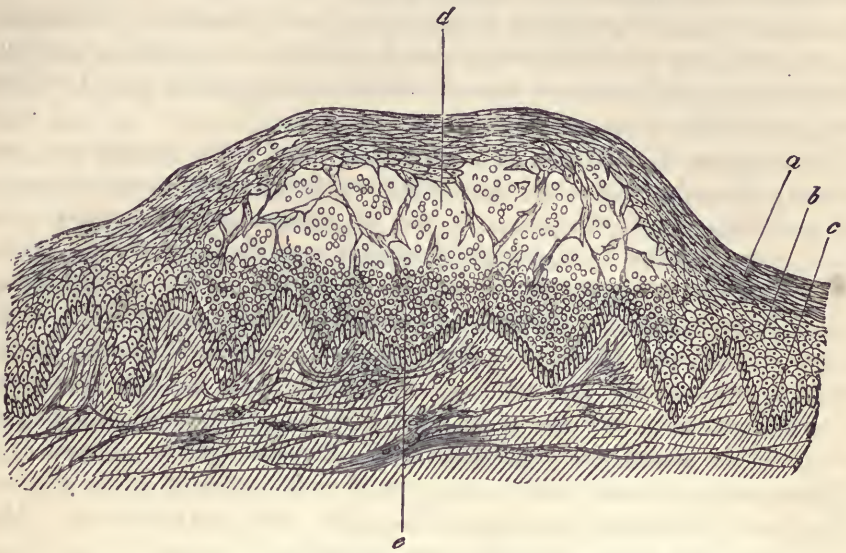
The greater or less abundance of the eruption distinguishes certain forms of small-pox. Where the pustules stand apart the attack is known as discrete, where they coalesce, as confluent. There is in no case coalescence at the start. The confluent form is the result of such abundant eruption as in the growth of vesicles to more than cover the skin. Vesicles break into each other as their surfaces extend. The disease distinguishes itself in modified form by showing the eruption always discrete—*i. e.* less abundant. It may be so much further modified as to disturb the regular course of other features of the disease. This modification is observed more especially in cases of partial immunity secured by previous attack or vaccination, and this much-modified form is known as varioloid.

In the very gravest form of the disease, a form which is fatal from the start, the eruption distinguishes itself by its absolute absence. The peculiar eruption of small-pox is substituted by hæmorrhage, to constitute the variety known as purpura variolosa. Quantitative varieties exist, therefore, in modified (varioloid), discrete, and confluent forms, and qualitative in hæmorrhagic forms.

Returning now to the more detailed study of the eruption, it is observed that it appears first on the uppermost part of the body, on the scalp, about the roots of the hair, on the forehead. The hair conceals it, so that, as a rule, the eruption is seen first on the forehead. It passes down thence over the face in regular progression, invades next the neck and upper extremities, then the chest and trunk, and lastly the lower extremities. Aitken declares that it appears in these different parts of the body in successive crops, the first upon the face, the second upon the neck and upper extremities, the third upon the trunk and lower extremities, and that there is something of an interval in the outbreak of these eruptions. A distinctive feature of the disease is the regularity of its march, so that while it is pustular upon the face it may be only vesicular on the trunk, and at the end of the papular stage on the lower extremities. There is observed also regular progress in the stage of the eruption, so that papules, vesicles, and pustules are not to be found intermingled on the same parts of the body. The eruption, of whatever form, is always less marked upon the body than the face. As the papules develop they become more and more conical, to finally show at their extreme apices a clear opaline fluid, which gradually invades the substance of the papule to convert it into a vesicle. The reticulated structure of the vesicle accounts for the fact that when punctured it does not collapse, but allows to exude from its interior only a small quantity of its contents. The vesicle is, as stated, many-celled. (See Fig. 19.) The walls of these cells are composed in part of sweat-glands or hair-follicles—structures which resist the erosive action of pus or of the poison of the disease, so that, while the vesicle expands in every

direction, it is held down by these firmer tissues. This fact accounts, as stated, for the central or eccentric depression which is regarded as such a characteristic feature of small-pox. It must be said, however, that many vesicles and pustules which show no umbilication or depression may always be encountered. It must be further admitted that this same central depres-

FIG. 19.



Section of Variolous Lesion of the Skin: *a*, outer layer of epidermis; *b*, middle layer; *c*, cylindrical cells of the rete Malpighii resting immediately upon the papillæ; *d*, reticulated cavity of the pock, containing pus-corpuscles, with the epithelial framework; *e*, purulent infiltration of the middle layer of the epidermis (Curschmann).

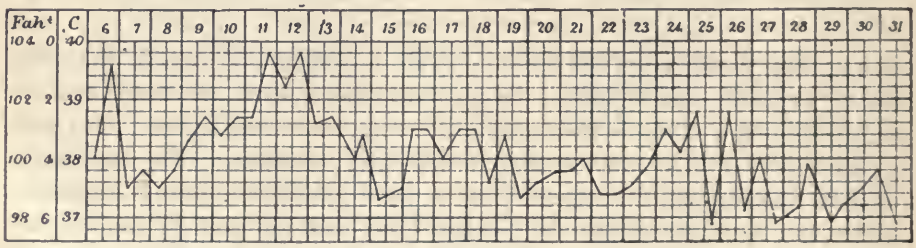
sion is also occasionally, but by no means so commonly, seen in the vesicles of varicella and syphilis. So soon as the vesicle has become thoroughly distended it loses its clarity, to become more and more turbid and opaque—that is, the number of pus-corpuscles increases. The inflammation has become now so extensive as to have affected subjacent structures, so that the base of the pustule becomes dark and the pustule itself seems surrounded by a halo. The whole skin is now infiltrated and thickened, and where the eruption is abundant, as upon the face, the eyes and ears are swollen to closure, the face bloated to distortion. The distension may be so great, especially in unyielding structures, as to give rise to extreme pain. Pain is felt more especially in the fingers, where the eruption is always so abundant, even in pronounced discrete forms of the disease, as to show some degree of confluence. The pain of this distension about the fingers may be so great as to overshadow all other subjective sensations and require especial treatment in its relief. One may only realize the severity of the pain in the fingers by recalling the amount of suffering which is occasioned by a single paronychia. In small-pox there are paronychiæ upon every finger. The old writers speak of the œdemato-phlegmonous inflammation of the extremities. A few pustules

in the eye may destroy the sight. Van Swieten records a case where a single pustule on the prepuce produced a painful phimosis and dysuria.

Curschmann claims that the more abundant eruption observed under heat or moisture, under poultices, plasters, etc., shows itself only when these applications are made before infection or in the stage of incubation. When he produced hyperæmia later, as in the initial stage, with mustard plasters, iodine, etc., the eruption was not thicker here than elsewhere. In one case in his experience an individual presented himself with long lines of eczema, the result of numerous scratchings of the skin for relief of the irritation of pediculi. The eruption when it occurred showed itself in these lines and seemed studded with pustules like strings of pearls.

The eruption of small-pox does not confine itself to the outside skin, but appears also on the inside skin, the mucous membrane of the mouth, pharynx, and sometimes deeper structures. It may be nearly always discovered in the fauces, over the palate and tonsils, and sometimes on the inner surfaces of the lips and cheeks. Occasionally it invades the larynx, to alter or abolish the voice. It may show also deeper lesion than hyperæmia and swelling of the mucous membrane. Ulcers may form in the larynx, with affection of the cartilage, perichondritis, and œdema of the glottis. In a bad case the tongue, which seldom shows any sign of eruption, is swollen to such extent as to protrude from the mouth, and in confluent cases salivation may be profuse. The affection may also extend from the throat to the nose, which it may block from behind, and subsequently involve the Eustachian tubes and middle ear. Mucosæ of other parts of the body are rarely invaded.

Fig. 20.



Temperature-chart of Case of Variola (Wunderlich).

The course of the temperature in variola is characteristic. (See Fig. 20.) The fever reaches its height, as stated, with the period of eruption. As soon as the eruption has covered the body the temperature begins to subside, and falls often nearly to the normal grade within thirty-six hours. With the subsidence of the temperature the pain in the back, the nausea, and vomiting disappear, and the patient seems on the road to recovery. As soon, however, as the vesicles become converted into pustules, about the sixth or ninth day of the disease, the fever is renewed, sometimes with shivering fits, always with a rise of temperature to 102° or 103° F., but rarely to the elevations reached during the stage of invasion. This is the secondary fever, the fever of suppuration, caused

entirely by the micro-organisms of pus. Strictly speaking, it does not belong to the small-pox process. It is only a secondary effect, but is none the less characteristic of the course of the disease. With the stage of desiccation the fever again gradually subsides, to terminate by lysis in the course of the subsequent week. This subsidence, however, may be at any time interrupted and the fever aggravated by complications.

In confluent small-pox the eruption is more abundant from the start. It shows itself, instead of in distinct maculæ or papules, as a more profuse redness from coalescence. Sometimes the sense of hardness or unevenness of the surface may not be recognized on account of the more uniform elevation of the whole surface. The individual papules are always smaller than in the discrete form, but they are much more abundant. The stage of vesiculation is represented by an accumulation of a more or less milky fluid over flat surfaces, often in irregular or zigzag shapes. The swelling is greater, as is also the corresponding deformity at the period of suppuration. The eyes and the ears are swollen, the face is enormously bloated. The scalp is lifted from the head, and the face has the appearance as if covered with a mask or heavily coated with coarse sand (*pergamenæ speciem visu horrendam* (*cutis faciet*) *exhibet*) (Morton). With the rupture of this parchment-like coat masses of decomposing fluid ooze out to stream down over the face and make of the patient an object so loathsome as to be repulsive even to intimate relatives.

Chief among the *varieties* of small-pox are the abortive and hæmorrhagic forms. The abortive is that variety in which the course of the disease is altered from the start. The period of incubation is sometimes shortened, the invasion may be brief, the eruption changed in various ways, the duration cut short. This form is best described under the rather unfortunate denomination of "varioloid."

Hæmorrhage may occur in the course of small-pox in no less than four distinct varieties. Blood is not unfrequently effused in the vesicles or pustules of patients who do not remain recumbent, who leave the bed and get up too soon in the period of convalescence. In these cases the hæmorrhage is confined almost exclusively to the lower extremities, and shows itself as petechiæ or purpura, not unlike the common form of this affection. Such hæmorrhage is purely local, due to escape of blood through paretic vessels. It speedily subsides by absorption with rest in bed; it has no prognostic gravity.

Reference has already been made to that petechial eruption which occurs as an initial rash on the lower surfaces of the abdomen and inner aspect of the thighs. This eruption has also a hæmorrhagic foundation. It is of diagnostic value, but, as stated, has no prognostic significance.

Aside from these eruptions, blood may be poured out into the true eruptions of small-pox at any part of the course of the disease. This accident occurs most frequently in cases debilitated by previous disease or bad surroundings, but sometimes, fortunately exceptionally, under totally unaccountable circumstances. The blood is effused at times into the papules, more frequently into vesicles, at the period of full maturation—*i. e.* at the height of

the disease. The clear serum becomes turbid, sero-sanguinolent, and finally the vesicle is filled with blood. Sheets of blood, diffuse and black, fill the interior of confluent vesicles and pustules, and blood appears under the skin as livid patches, vibices, and ecchymoses in various parts of the body, to constitute what is called hæmorrhagic small-pox, black small-pox, "variola nigra." With this effusion of blood there is a corresponding collapse. Free hæmorrhages—metrorrhagia, hæmaturia, enterorrhagia, least frequently hæmatemesis—may occur also from the various mucous surfaces, under which the patient rapidly succumbs. Should he survive the prostration caused by the hæmorrhage itself, he may have to face other and worse dangers. Diphtheritic deposits form in the pharynx, a scorbutic condition of the gums develops, or nephritis ensues and the patient may perish from uræmia. Recovery from this condition is rare; convalescence is slow and tedious.

Last among the hæmorrhagic eruptions remains to be described that particular variety in which the hæmorrhage assumes prominence over all other signs. This variety presents such distinctive features, so different from all other forms of small-pox, as to have led competent observers to consider it a special malady. The fact that the disease, in any of its forms, may be communicated from this form, and that the body remains infectious also after death, establishes its true nature. That this hæmorrhagic form may be distinguished from those just described, especially from the variola hæmorrhagica pustulosa, it has been given the separate name of "purpura variolosa"—a term which fixes in the foreground the hæmorrhagic character which literally dominates the disease. In this variety of small-pox the initial rash and the true eruption are alike wanting. Although this is the fulminant form of small-pox, it does not necessarily commence with violent signs. It attacks, by preference, the young, healthy, and strong, but does not spare the weak and debilitated. Drinkers and pregnant and parturient women are among its preferred victims.

The disease begins in the ordinary way—with chill, vomiting, and rapid prostration. The stage of invasion (if it differ in any way from the ordinary cases of small-pox) is distinguished by the severity of pain in the back. In the experience of the author patients have complained of excruciating pain in the back when there was no other symptom, not even fever. Another distinctive feature is the rapidity of appearance of hæmorrhage. Should the disease occur during menstruation, metrorrhagia ensues, and the nature of the disease may be thus overlooked, as the pain and the hæmorrhage may be both connected with menstruation. Hæmorrhage now shows itself under the skin—first upon the trunk, later upon the extremities, but never upon the face. The surface assumes a blood-red hue, like that of scarlet fever, and in this redness points and patches of blood appear. The eruption is usually petechial upon the extremities and confluent as irregular ecchymotic patches on the chest and trunk. The face is swollen, the eyes suffused and sunken and surrounded with black rings. The tongue is thick and heavily coated. The breath is exceedingly fœtid. There may be elevation of temperature; sometimes there

is no fever, and often the temperature is subnormal. The tendency is steadily downward, and death occurs by the end of the first week. Fortunately, this form occurs in but 5 per cent. of cases.

A peculiar subvariety or disposition of eruption is that described by Marson as "corymbose." In these cases the eruption shows itself in patches or clusters the size of the hand, as thickly set as possible, while the surrounding skin remains often entirely free. The patches are often symmetrically distributed upon the extremities. The variety is very rare, but, contrary to what might be expected, is very dangerous. Marson found that the appearance of but a single cluster gave gravity to the attack, and rendered it much more liable to complications and greatly protracted convalescence. The mortality was over 40 per cent. Other singularities are verrucose, pemphigose, or miliary eruptions. They are, however, more commonly met with in varioloid.

Varioloid is a misnomer, for the affection is not like variola; it is variola itself. Varioloid does not stand in the same relation to variola as typhoid to typhus fever; varioloid is variola in modified form—is, in fact, the lightest form of small-pox. The disease occurs in this form on account of natural insusceptibility, on account of previous attack, formerly on account of inoculation; but the great majority of cases seen in our day are due to incomplete protection from vaccination. The immunity secured by vaccination has run out, and the severity of the attack will, to a certain extent, depend upon the remaining degree of protection. A case of unmodified variola in our day is a rarity; that modified or mitigated variola known as varioloid is still frequently seen. A knowledge of the nature of varioloid and its differences from other simulating affections is necessary, that the disease be recognized at once in protection of others. From what has been stated it is needless to add that varioloid, mild as it may be in itself, may transmit true variola in any, even its most fulminant, form. Most of the cases encountered in our day are so mild that the question of diagnosis concerns differentiation of varioloid from varicella as much as the recognition of variola itself.

As already stated, small-pox is a very uniform disease. In modified form, however, it presents many irregularities. Varioloid distinguishes itself by abnormalities in every stage of the disease. As most of the cases are due to incomplete protection by vaccination, the various irregularities are mentioned by Morrow when he says that "vaccination denaturalizes small-pox, deranges the original order of the disease, and effaces its most distinctive features."

It is generally assumed that the modification of symptoms is apparent in the initial stage of the disease. This view, however, is by no means correct. The disease begins with its usual train of symptoms, and as a rule with its original violence. The difference concerns duration rather than degree. The initial stage is often cut short a day or two, so that the eruption may appear by the end of the first or second day. The various initial eruptions occur also in varioloid—the petechial as an exception, the erythematous as a rule. It is a common observation that a pronounced erythematous eruption or scarlatiniform rash betokens varioloid rather than variola. Curschmann declares that

we may predict, in spite of the severe depression of the general system, that the form of the disease, if erythematous, will be mild, while petechiæ will nearly always be followed by variola vera, which is not infrequently confluent. For what comfort it may bring, the author may state that the three cases of petechial eruptions in Simon's triangle which have occurred in his experience have preceded, without exception, mild, abortive attacks of the disease.

With regard to the real eruption, varioloid presents the greatest variations. It may begin on the scalp, forehead, and temples, as in an ordinary case, and progress in regular or irregular course. It may, on the other hand, show itself first on the neck and chest or elsewhere over the trunk, to appear later on the extremities or face. As a rule it is much less abundant, so that it is nearly always discrete. There are, however, exceptions to this rule, and marked cases may show isolated patches of confluence on the face and hands. On its first appearance the eruption differs in no way from that of the more pronounced form of the disease. It comes out in spots, which are elevated into papules in the course of the first day. The papules slowly show fluid at their accuminated apices, and become thus entirely converted into vesicles as before. Here, now, the change is usually observed: the eruption usually stops at this stage, and the vesicles, which may have become umbilicated, begin to dry up and disappear. They may fill out as in the course of severer forms; their contents may become turbid and opaque, and the vesicle may be transformed into a pustule, but it is plain to see that the force of the disease is spent. Certain pustules may rupture, but the process is limited, and the secondary fever of suppuration is reduced or is entirely absent. In consequence of the fact that pustulation does occur in places with erosion and destruction of tissue, pits may be left, but they are few and far between as compared with the lesions of ordinary small-pox.

The disturbance of the general progress of the disease is marked also by irregularity. It is more common to find pustules and vesicles or vesicles and papules in a closer proximity in varioloid than in variola. Moreover, the eruption does not last so long. Desiccation begins on the fifth or seventh day, and most of the papules dry up into crusts without rupture. These crusts, as a rule, leave only pigmented traces without scars. There is often also disproportion between the severity of the fever and the eruption. There may be high fever in the presence of but ten or twenty vesicles or pustules over the body, or, *per contra*, the eruption may be almost, or in places entirely, confluent, with but little elevation of temperature. It is plain to see that vaccination has at every point put a muzzle upon the disease.

The various transformations of vesicles and papules which may occur during the process of modification or abortion may convert vesicles or pustules into warty masses or bullæ, or ruptured vesicles may fill with air to constitute varieties known as variola verrucosa, pemphigosa, miliaris, ventosa, or cellulosa, etc. So of the various affections of the mucous membrane. While they may be present, or in individual cases more or less pronounced, in initial stages they rarely assume prominence or give rise to serious complications.

Complications which occur in the course of small-pox do not differ much from those of equally grave acute infections. Sufficient mention has already been made of the lighter affections of the pharynx and larynx. It remains to be said that gangrenous processes, œdema of the glottis, and perichondritis occur in exceptional cases. Stenosis from either of these causes may necessitate intubation or tracheotomy.

Bronchitis belongs to variola as to most of the exanthemata. It is very liable to extend in childhood, to infect the finer bronchial tubes, and to result in broncho-pneumonia. Pleurisy is by no means so common, but is by no means rare. Pericarditis, endocarditis, endometritis, meningitis, are not uncommon complications in grave cases. Affections of the joints, arthritis, pyæmia, septicæmia are much more frequent.

Small-pox occasionally affects the eye. Conjunctivitis, keratitis, affections of the lids, are the most common lesions. Disease of the choroid and retina occurs in exceptional cases. Panophthalmitis, with destruction of the globe, was not uncommon in ancient times. Ocular complications in our day are neither frequent nor severe. In all his remarkable experience Hebra saw them in only 1 per cent. of 5000 cases of small-pox.

By extension of the inflammation of the fauces and pharynx the middle ear may be attacked, to result in otitis or otorrhœa, with subsequent ankylosis of bones and deafness. Phlegmonous inflammations, gangrene of the skin, and furunculosis occur frequently in confluent cases; and local and diffused inflammation of the brain and cord, paralysis, and bed-sores may nearly complete the possible complications.

Diagnosis.—The diagnosis of the disease rests upon the following points: the possible existence of other cases, the history of sufficiently recent protection by vaccination. The mere existence of a scar is no evidence of protection. The worst case of purpura variola encountered in the experience of the author had three well-marked cicatrices upon the arm as evidence of previous vaccination. Then it is observed that the illness sets in suddenly, and is usually severe from the start. Strong men stagger as if drunk. The temperature rises rapidly. Pain in the back is peculiar in its intensity; initial eruptions may be characteristic. The true eruption appears upon the third day after the initial chill. It is maculated, not punctate like that of scarlatina, but darker than the scarlet of scarlatina and lighter than the dusky hue of measles. It is seen first upon the scalp and upper part of the face, spreading downward regularly and rapidly; it does not spare the nose or region of the mouth. It yields a peculiar feeling of hardness as of shot under the skin. Elevation into papules occurs during the first day. The diagnosis becomes nearly certain when the papules by the third day change into vesicles, some of which subsequently become umbilicated.

Small-pox is one of the most grave of the acute infections which survives from the pre-sanitary period of civilization. We see it, for the most part, as a mere relic or rudiment of its former self. There is lacking with us that element of multitudinous infection which gives volume and virulence to the

disease. Nevertheless, even in its modified form, it preserves its character as a grave infection, and it may hence be confounded with any of the infections of equal gravity, especially any of those that are attended with an eruption.

Disregarding the eruption for the present, because not present at the start, mistakes have thus arisen in connection with meningitis, pneumonia, and typhus fever. Meningitis, especially the cerebro-spinal form, pneumonia, and typhus fever begin, like small-pox, in the midst of health, with violent chill, rise of temperature, and rapid prostration. Gastric symptoms, vomiting or nervous shock, especially in children, and convulsions, may announce the onset of any of these infections. In the absence of an epidemic or the history of exposure, in the absence also of adequate protection by vaccination, the diagnosis must sometimes be held in abeyance for twenty-four or forty-eight hours until distinctive signs of one or the other of these diseases are manifest. Meningitis distinguishes itself by hyperæsthesia, opisthotonos, and herpes, as well as by its irregular temperature curve. Pneumonia is early characterized by pain in the side, cough with glutinous and rusty sputum, and increase of respiration out of proportion to the pulse. The diseases which are, however, most frequently confounded with small-pox are those which are attended with an eruption, and chief among these is typhus fever. Typhus fever has, however, a history of importation which may be traced or prevalence which may be known. It begins often, like small-pox, suddenly, with a severe chill in the midst of health, and shows an eruption on the third day. The eruption of typhus, however, appears first upon the body, chest, and abdomen in the form of maculæ which soon become petechial. The eruption of small-pox appears first upon the scalp and forehead, and progresses over the face before it appears upon the body. It shows itself in the form of maculæ, which soon become papular, vesicular, etc. The petechiæ which may occur in small-pox occur on the legs or thighs or in the course of a hæmorrhagic form. Vesicles, especially umbilicated vesicles, are never seen in typhus fever. There is also characteristic difference in temperature, which subsides with the appearance of the eruption in small-pox, but persists unaffected for several days or as long as a week in typhus fever.

Confusion with typhoid fever is less pardonable. Typhoid fever begins insidiously, requiring the time of a week to reach the temperature attained by small-pox in a day or two. The cloud about the brain which belongs both to typhoid and typhus fever from the start is not present in small-pox until the last stages of the disease. Typhoid fever shows abdominal symptoms, roseola on the seventh to tenth day, meteorism, gurgling, diarrhœa, etc., absent in small-pox.

A light case of small-pox may be regarded as measles, and a bad case of measles as small-pox. Consequently, the separation of small-pox from measles is the most frequent problem submitted to the practitioner. The future of the case, the safety of the community, the reputation of the physician, depend upon the proper solution of this problem. Here, too, help may be had by a knowledge of the history of the case as to the existence or absence of an attack of

measles or small-pox, the period of the last successful vaccination, the prevalence of either disease in the community. As for measles, it is always present in cities, and, thanks to the popular fear of small-pox, knowledge of its existence is early promulgated by the health authorities. Nevertheless, sporadic cases steal in at times unannounced. In the experience of the author with the management of a large dispensary practice small-pox was twice introduced into Cincinnati by peripatetic philosophers commonly called "tramps." These cases formed centres of infection. Knowledge of the period of exposure—*i. e.* the period of incubation—is of little value. The stage of invasion is much milder in measles than in even modified forms of small-pox, for, as has been stated, varioloid may be announced with symptoms as severe as those which distinguish the onset of variola vera. The chill is less severe, the fever is less high, the prostration is less profound, in measles as a rule. There are, of course, exceptions on both sides. The eruption appears on the third day of small-pox, on the fourth day of measles. The maculæ of measles are bigger than those of small-pox. They appear also upon the back almost at the same time as upon the face, whereas the eruption of small-pox much more uniformly appears upon the face, and reaches the back only later in its advance over the body. The maculæ of measles are softer than those of small-pox. Rhazes said nearly a thousand years ago: "The difference between the two he found to be that measles are red and appear only on the surface of the skin without rising above it, while the small-pox consists of round eminences. When these eminences appear fix your attention on them, and if you are in doubt as to the disease, do not express any opinion about it for a day or two; but when there are no eminences you must not give as your opinion that the disease is small-pox." Collie declares: "A case of small-pox severe enough to simulate measles imparts to the hand in passing it over the surface a hardness and furrowed roughness, as that produced in passing the hand over a piece of corduroy; whereas in raised, confluent measles it is that of passing the hand over a piece of velvet." Moore gives the "grisolle sign" as a certain means of diagnosis: "If upon stretching an affected portion of the skin the papule becomes impalpable to the touch, the eruption is caused by measles; if, on the contrary, the papule is still felt when the skin is drawn out, the eruption is the result of small-pox." The catarrhal symptoms, more especially the coryza, which may exist in both affections, are wont to be more prominent in measles at the start, but persist longer in small-pox. The course of the temperature is characteristic in the two diseases. The fever falls in small-pox with the appearance of the eruption, whereas in measles it remains unaffected or may rise higher. The appearance of papules or vesicles soon dissipates all idea of measles.

The severity of the sore throat, the backache, and the scarlet color of the rash, which appears as minute points as early as the second day after the initial chill, distinguish scarlet fever. The grave hæmorrhagic form, "purpura variolosa," is recognized by the extreme severity of pain in the back, as well as by the petechial character of the eruption, free hæmorrhages, etc.

Papular eczema is irregular in its distribution, unattended with fever or involvement of the mucous membranes. The same exceptions apply to erythema, acne, and herpes. Only the most superficial observer could consider these eruptions variolous.

Syphilis may show pustules to closely resemble discrete variola, including even the process of umbilication, but the absence of the initial signs, chill, fever, pain in the back, etc., the history of syphilis or associated evidence elsewhere, render the diagnosis easy as a rule.

All cases concerning which there is any doubt should at least be isolated for a time until sufficient protection can be offered to others by vaccination. Marson says of his experience in the London Small-pox Hospital: "Upward of twenty diseases have been mistaken within the last few years, in the early stage of the disease, for small-pox, and the patients have been sent, as having small-pox, to the small-pox hospital." The separation of variola and varicella will be discussed under *Varicella.*

The **prognosis** is largely determined by the last successful vaccination. The next most important factor is the determination of the form of the disease. The third is the age of the patient. Small-pox in infancy has a mortality which is put at 90 per cent. Almost equally grave are the cases which occur in pregnancy and the puerperium. The greater danger which is thus imparted to the female sex is counterbalanced in the male sex by the mortality of the disease among drinkers. The percentage runs high again in advanced age: nearly 75 per cent. of old people unprotected by vaccination or previous attack succumb to the disease.

Severe symptoms on the part of the nervous system are of evil omen, but to a less degree in children than adults. Trousseau laid great stress upon tumefaction of the extremities—what he called "red œdema"—which should set in at the end of the ninth day with acute pain; with Sydenham, Morton, Van Swieten, Borsieri, he attached great importance to it in a prognostic way. He says: "Swelling of the hands and feet is such a necessary phenomenon in confluent small-pox that patients almost invariably succumb where it is absent unless there be a great critical discharge by the kidneys or bowels." Profuse suppuration in the skin is a sign of danger. Hæmorrhagic small-pox is very serious; less than one-half the cases recover. The prognosis is not, however, unfavorable because of initial petechiæ, which may show later upon the legs of patients who try to get about too soon. Purpura variolosa is always fatal. The mortality of the unvaccinated ranges, even in our day, at 20–40 per cent.

Prophylaxis.—Vaccination, if it could be enforced, would render superfluous all other prophylaxis, including isolation. Inoculation, which it substitutes, has only historic interest. Vaccination and revaccination, if they could be made compulsory, would eventually eradicate the disease; thus but a single fatal case of small-pox has occurred in the German army during the past fifteen years. Unfortunately, however, vaccination cannot be made compulsory in our country, "where the cry of infringement of personal liberty is the shibboleth of the demagogue" (Foster), so that patients must still be

isolated and sick-rooms disinfected. A temperature of 400° F. is fatal to small-pox. The organisms of the disease are destroyed by sulphur in sufficient concentration. That this process may be properly brought about, it must be done by health authorities. All combustible material should be consumed if it may not be subjected to the antimycotic action of live steam; walls should be rubbed down with bread, and floors scrubbed with a solution of corrosive sublimate, 1 : 1000; doors and windows should be closed, and sulphur, 4 pounds to every 1000 cubic feet of air, should be burnt to bring about perfect fumigation: at the end of two days the chamber may be thrown open and thoroughly ventilated for two weeks. Bedding, clothing, curtains, etc., after subjection to superheated steam, should be suspended in the air day and night for a week. The dead body should be subjected to immediate interment, as infection is disseminated from its surface up to the period of decomposition. In the interval between death and burial the body should be enveloped in a sheet saturated in the solution of corrosive sublimate, 1 : 1000. Transportation should be permitted only when a body is put in an air-tight metal case. In the experience of the author an endemic was once developed at a distance in a country town by neglect of this precaution.

Treatment.—If seen early the patient should be vaccinated at once. Vaccination in the early stage of the disease modifies variola. After the fourth day vaccination is useless. Marson puts it positively: "Suppose an unvaccinated person be exposed to small-pox on Monday; if he be vaccinated as late as Wednesday, the vaccination will be in time to prevent small-pox being developed; if it be put off until Thursday, small-pox will appear, but will be modified; if the vaccination be deferred until Friday, it will be useless: it will not have had time to reach the stage of areola, the index of safety, before the illness of small-pox begins." Curschmann does not subscribe to these views. He declares that he has seen in cases in which vaccination was practised that infection with vaccinia and small-pox pustules developed side by side. He doubts whether vaccination can render the disease even milder in its course. Nevertheless, so long as there is doubt the patient should have the possible benefit of early vaccination.

Treatment in the absence of a specific is wholly symptomatic: rest in bed in a thoroughly ventilated room at a temperature of 65° F., as determined by a thermometer at the head of the bed; light but sufficient covering; cool drinks, water, lemonade, seltzer-water, in sufficient quantities; fever diet, milk, soups, gruels. This much we owe to Sydenham. What it must have effected may be learned by the results from that which it substituted. The contrast is shown in a chapter from the practice of Diemerbroek. "Keep the patient," says Diemerbroeck, "in a chamber close shut; if it be winter let the air be corrected by large fires; take care that no cold air gets to the patient's bed; cover him over with blankets. Never shift the patient's linen till after the fourteenth day, for fear of striking in the pock, to the irrecoverable ruin of the patient. Far better it is to let the patient bear with the stench than

thus be the cause of his own death." Trousseau is right when he says: "If the second epoch in small-pox was introduced with inoculation, and the third with vaccination, the first was introduced with the treatment of Sydenham."

Fever above 103° F. can best be combated by frequent baths or by phenacetin, gr. x, or antifebrin or antipyrine, gr. v, or in half of these doses in childhood. For throat complications steam from an atomizer, simple or medicated with boric acid, gr. xv to f℥iv; thymol, gr. xv, alcohol and water, āā. f℥ij; or carbolic acid or creasote, ʒss, glycerin, f℥j, water, ℥iij, or with less efficacy gargles of the same strength. Inhalations may substitute all local applications in very young or refractory children. Chloral, gr. ij–x, becomes a necessity in periods of unrest, nervousness, insomnia. It has no equal in the relief of nausea and vomiting. For jactitation or extreme nervous distress it may have to be substituted by Dover's powder, gr. ij–v. Frequent ablutions of tepid water, ointments, diachylon ointment, plasters, mercurial plaster, or opening pustules after the manner of the Arabs and touching them with nitrate of silver, or better carbolic acid and glycerin āā., or touching the tops of *beginning* pustules—*i. e.* mature vesicles—with a camel's-hair brush dipped in carbolic acid, best prevents or limits pitting. Xylol internally is said by Zülzer to have the power of coagulating the contents of pustules, but the claim was not at all substantiated by subsequent trial. Where tissue is destroyed, cicatrization must result, and, in consequence of it, pits and scars. Means to prevent deformity to be effectual must therefore be brought into use before the stage of suppuration is complete. Nothing can prevent pitting in an established confluent small-pox. The best clinicians are content with frequently renewed water-dressings made antiseptic as much as may be with sublimate solutions, 1 : 5000 or 1 : 10,000. The whole question, with all the other horrible evils of small-pox, sinks into insignificance and slinks away like the devil at sight of the cross when brought face to face with vaccination.

VACCINATION.

By JAMES T. WHITTAKER.

Vaccination (from *vacca*, a cow; vaccinia, cow-pox) is a term introduced from France to express the inoculation of man with cow-pox in prevention of small-pox, and to substitute the awkward word "cow-poxing." The promulgation of vaccination by Edward Jenner in 1798 constitutes one of the great epochs in the history of mankind, in that from this period the terrible tropical plague variola, which overran and literally ruined Europe and the rest of the world, was reduced to the trivial malady varioloid, which is, uncomplicated, never fatal.

Jenner was a medical apprentice at Sodbury when he became acquainted with the popular belief in the protective influence of cow-pox; and, though he was unable to interest his preceptor, the celebrated John Hunter, in whose house he subsequently lived for two years, or to convince any of his medical brethren of any relation or antagonism between the affections, he could not dismiss the subject from his mind. He visited dairies in Gloucestershire and made observations and prosecuted investigations for himself. He found that there existed a widespread belief among the dairymen that certain individuals, who had contracted sores upon their hands from contact with sores on the udders of cows, were never attacked with small-pox. Much contradictory testimony presented itself at first, and many disheartening exceptions were found. If genius be patience, it found in Jenner an example, for Jenner worked with this subject for more than twenty years before all the mighty truth of it was clear to his mind.

May 14, 1796, was the memorable day when Edward Jenner transferred cow-pox from vesicles on the hands of Sarah Nelmes, a dairymaid, by means of two superficial incisions, into the arms of James Phipps, a healthy boy eight years of age. The cow-pox ran its ordinary course, and a subsequent inoculation with small-pox on the first of the following July failed to produce the disease. In the same month Jenner wrote to his intimate friend Gardner: "The boy has since been inoculated for the small-pox, which, as I ventured to predict, produced no effect. I shall now pursue my investigations with redoubled ardor."

This was the first attempt of a simple practice which has, within less than a century and without radical correction or real improvement, afforded to all mankind protection from the ravages of small-pox. Jenner was led to make this experiment by the observation that individuals accidentally infected with

cow-pox, to use his own words, "resisted every effort to give them small-pox." A number of children, subsequently vaccinated in succession, "one from the other," were after several months exposed to the infection of small-pox, "some by inoculation, others by variolous effluvia, and some in both ways, but they all resisted it." Though Jenner was himself now thoroughly convinced, he determined to withhold his conclusions from publication until, by frequent repetition and fortification in every direction against any possible error, he might establish them without doubt. He repeated his inoculations with every precaution, and finally prepared his pamphlet. Hereupon he visited London to obtain the assent and support of his medical friends, but was unable for nearly three months to find any person in London who would submit to the operation. Finally, on his return home, the distinguished surgeon Cline introduced vaccine matter over the diseased hip-joint of a child as a means of securing counter-irritation. When he found later that this child had thus secured immunity against small-pox, he became an earnest advocate of the operation, and a supporter of Jenner at a time when the latter stood in need of one. There is evidence that Jenner worked with this subject, encountering and overcoming obstacles and opposition on every hand, for over twenty years before he announced his results to the world, and it is known that fully two years elapsed—a delay which might be considered culpable in our day—between the first vaccination and the publication of his paper. The paper was modestly entitled an "*Inquiry into the Causes and Effects of the Variolæ Vaccinæ, a Disease discovered in some of the Western Counties of England, particularly Gloucestershire, and known by the name of Cow-Pox*, London, 1798, 4; 1800, 8; 1801, 8."

Jenner lived to see all opposition overcome and the procedure adopted all over the world, and to receive, with universal honors and emoluments, from Parliament in 1802 an award of ten thousand pounds (nearly all of which was lost in fees), and later, in 1807, a second allowance of twenty thousand pounds. In 1857 a statue was erected to him in Trafalgar Square in London. The most consummate cynic must admit that up to the present time Edward Jenner has been the greatest benefactor that the world has known.

Intimations of the protective influence of cow-pox had been made here and there in various parts of the world, especially in connection with dairies. People in different places had believed in the influence of this protection, and certain individuals had actually practised it upon themselves and in their families. Such statements have been handed down from Persia, Scotland, and Holstein, but they made no impression of the virtue and secured no adoption of the practice of vaccination up to the time when a country milkmaid said to Jenner during his student-life at Sodbury, "I cannot have the small-pox, for I have had cow-pox." This statement repeated itself and rang in his ears for over twenty years. It left him no rest until it resulted in the discovery of the protection of mankind, and but for the stupidity of men would have long since led to the extermination of the disease.

Vaccine matter was soon carried all over the world. The Spanish govern-

ment sent ships and surgeons to all its possessions in the Old and New Worlds. The expedition made circuit of the entire globe in the course of three years.

The operation was first performed in this country by Prof. Waterhouse of Cambridge, and in the same year (1801) was practised in his own family by Jefferson, then President of the United States. The Empress baptized the first child vaccinated in Russia with the name "Vaccinoff," and gave it governernment endowment. The nations of the earth vied with each other in tributes to Jenner and demonstrations of joy. Napoleon Bonaparte took his signature as a passport. The anniversaries of the first operation and that of the first vaccination were celebrated in Germany—in a special temple at Brunn, Moravia—as the Church celebrates its saints, with holidays, and our own Indians sent with belt and wampum a declaration that "we shall not fail to teach our children to speak the name of Jenner and to thank the Great Spirit for bestowing upon him so much wisdom and benevolence."

Jenner spent the rest of his life in the perfection of his discovery. His practical conclusions remain impregnable. In theory, however, he fell into two slight errors: one, the belief that cow-pox would protect for life—a belief that led later to some doubt regarding its protection in general; the other, that the disease was conveyed to the cow from the horse by individuals engaged in the double duty of hostlers and milkmen. A disease of the horse's hoofs, commonly known as "the grease," when conveyed to the bag of a cow produces an eruption which simulates, but which subsequent investigation has shown to be not identical with, the true cow-pox. The belief that cow-pox is modified human small-pox found much wider acceptance, and has few opponents in the present day. These opponents maintain, however, that the doctrine is dangerous, and that it has been the cause of insufficient protection, and therefore injury to vaccination, as well as directly of death by the propagation of small-pox itself.

Cow-pox is an infectious disease which appears in dairies from time to time, often at wide intervals of both time and space, and shows itself first in some particular cow, usually a young cow, a heifer in her first milk. It never appears first in other cattle than milk cows, and never shows itself elsewhere than on the teats or at adjoining parts of the bag, as they may be infected by direct pressure or contact. It appears in the form of scattered papules, which in the course of a few days show fluid at their apices, to become transformed into distinct vesicles. These vesicles are broken by the hands of the milkers, and the disease is thus disseminated in the course of a few weeks, sometimes months, throughout the entire dairy. After rupture the fluid of the vesicle thickens to form a crust, under which the eroded tissue or ulcer cicatrizes, producing a scar with indurated margins and puckered surface. Uncleanly dairymen often infect other parts of their own bodies with their hands. Jenner, Ceely, and Pearson described such cases of infection of the lips, side of the nose, temple, etc.

In January, 1799, Woodville of the London Small-pox Hospital succeeded in storing a supply of pure material, from which source Jenner, with several

hundred practitioners, got their vaccine matter. This Woodville stock was then used all over the world up to 1836. At about this time matter began to be introduced from other sources: first, from the Passy cow in 1836, and here, again, from the accidental infection of the hands of a milker. Material from these vesicles started a new stock, which was subsequently used in France. By 1838 the new disease, vaccinia, was so far forgotten in Jenner's own parish, Berkeley, Gloucestershire, that the milkers were ignorant of the cause of the appearance of vesicles upon their own hands. From these vesicles Estlin of Bristol established a new geniture. Next Ceely of Aylesbury (1838–41) discovered half a dozen cases of cow-pox in dairy-farms of his district, and cultivated lymph from them. In 1866 the disease was discovered in Beaugency, and this source furnished lymph for the inoculation of calves, which was now practised as a regular business in Dutch, Belgian, and other vaccine farms. Genuine cow-pox has since been discovered and described in Holland, Italy, Bengal, South America, Mexico, New England, Pennsylvania, and California; and there can be no doubt, as Seaton says, that "much more would be found than really is found if only looked for."

The first case of kine-pox in the United States was reported by Dr. John Yale of Ware, Mass., as observed at Torringford, Conn. (1844), and at Ware, Mass. (1855), the true nature of the pox having been established at Ware by inoculation of man as well as by propagation in calves. Martin of Massachusetts established the first well-equipped vaccine farm in the United States in 1870, with the inoculation of a constant succession of heifers. He was followed by Foster of New York and Griffin of Fond du Lac in Wisconsin. Vaccinifers from these farms furnish nearly all the lymph used in this country.

A disease attended with eruption of vesicles and pustules occurs in many animals besides the cow, as in the horse, sheep, goat, dog, etc. Sometimes the eruption is general, sometimes local. In some cases the disease is marked by ulcers, in others by glandular enlargements, etc. In some animals the disease is trivial, in others dangerous and often fatal. Cow-pox differs from all other kinds of pox in that the disease, as stated, is confined almost exclusively to female animals at the time of lactation, and the eruption is confined to the bag. After a period of incubation of three or four days the eruption appears as red spots, which speedily swell to assume the form of papules, become converted into vesicles by the fifth or sixth day, to be transformed into pustules by the tenth day. The pustules in full development are present to the number of twenty or thirty as fully-rounded bodies, slightly depressed in the centre, and often umbilicated. Sometimes they remain flat, with no central depression, their presence being then easily overlooked. A vesicle or pustule is not a single sac, but a set of chambers, puncture of which does not permit the escape of all the contents, and the full discharge of which can be secured only by pressure. The pustules dry to form crusts, which fall on the twelfth to the fifteenth day, to leave oval or rounded scars which persist for years.

It is characteristic of cow-pox to appear also in successive eruptions. Not

infrequently vesicles and pustules appear side by side with dry crusts. Individual vesicles run their course in five or six days, but the whole disease is a subacute and chronic process, lasting often for several months. The disease causes in the cow, as a rule, no sign of general distress, but sometimes there are fever and loss of appetite; occasionally there are quantitative and qualitative alterations in the milk.

The discovery of the origin of genuine cow-pox has always been a fascinating study. The characteristic course of the disease unmistakably gives it place among the infections, so that there can be no question whatever of spontaneous origin or generation. Inasmuch as the disease occurs only at intervals, it cannot be sustained by continuous succession, so far as the cow alone is concerned. It must therefore arise from some other animal or from man. Jenner considered it to be derived, as stated, from the horse, but this origin is now no longer considered tenable, as it breaks out in dairies where there are no horses, and occurs in places where horse-pox, as in Germany, is almost unknown.

The accumulated observations of a century reveal the fact that there are but two chief forms of small-pox—to wit, human pox and sheep-pox. They both attack the multitude, they both assume pandemic range: one is a genuine epidemic, the other a genuine epizootic. All other varieties of pox—that of the horse, cattle (including cow-pox), of swine, goats, and dogs—constitute, as Bollinger proves, no distinct individual disease. They are to be regarded only as irregular forms of the primary human or sheep-pox modified in different animals. Whether man got the pox from sheep or sheep from man is a question that may never be determined, but the best authorities (Bohn and Bollinger) unreservedly maintain that the virus is identical in the two cases—identical and interchangeable. The corollary of this fact determines the origin of cow-pox. Cow-pox is in its essence variola vera. It is variola modified in the body of the cow. If vaccinia be but an attenuated or modified variola, its protective action ceases to be a mystery. It protects by the immunity of previous attack. It protects by the immunity of inoculation—*i. e.* variolation; for vaccination is variolation with virus robbed only of its virulence.

On the other hand, it is alleged that, first, the processes are not similar—vaccinia remains always a local, variola a general disease; second, one process never produces the other—vaccinia always produces vaccinia, as variola always produces variola. Fatal cases have ensued in man after the use of matter from the cow inoculated with the small-pox of man (Chauveau).

According to Bohn, Gassner of Günzburg (1807) was the first to inoculate the cow with the small-pox of man. He introduced small-pox matter from the vesicles of children in a number of cattle. The operation succeeded eleven times. With the contents of vesicles so formed he vaccinated four children. They developed, without exception, perfect vaccinia pustules. This operation was subsequently practised on a large scale by Thiele of Kasan and Ceely of England (1838). They inoculated cows on the bag and vulva with human variola, with the development in every case of cow-pox, which was always localized at the point of inoculation, and which was never

followed by general eruption. Matter derived from these pustules in the cow, introduced upon the arms in children, produced the same result as matter from the common cow-pox. Thiele carried his new matter through seventy-five human generations in more than three thousand persons, and proved its thoroughness by subsequent inoculation with genuine variola humana in twenty-one cases. Ceely carried his matter through more than sixty generations in over two thousand people, and tested its value in the same way, with numerous inoculations with small-pox matter. These experiments were multiplied by Badcock, Senfft, and others until they seemed to have established, beyond doubt, the identity of variola and vaccinia. These conclusions were, however, controverted by the committee of Lyons, consisting of Chauveau, Viennois, and Meynet, who maintained (1865) that the cow was incapable of changing variola into vaccinia, and that the inoculation of variola only reproduced itself. The Turin committee (1874) reached the same conclusions.

As Bollinger states, however, any number of negative results cannot overthrow positive results. Much depends upon the mode of inoculation and the character of the animal operated upon. How else could it happen that Senfft got only positive results with all his calves? While, from occasional mistakes, some accident might occur in the reinoculation of man with matter thus derived from the cow, when ordinary precautions were observed the operation was always successful (B. Reiter, Kranz).

These cases do not, however, entirely explain the origin of cow-pox, for this disease may prevail in cattle independently of variola humana. Examples of coincidences have been noticed (Dinter, Saxony, 1860; Bollinger, Holzstein, 1871), but they are rare, and cow-pox, since the practice of vaccination, is much more frequent than small-pox. The frequency of cow-pox is explained by the fact that the disease is given back to the cow by humanized matter. Scarcely two decades of years had lapsed after its discovery until vaccination became universal, so that the frequency of genuine cow-pox in our day is explained by accidental inoculation with humanized vaccinia. Under these circumstances it is a matter of secondary importance, so far as the protective efficacy of the virus is concerned, whether so-called animal or human lymph be employed in vaccinating, for it is the same virus in every case. Animal lymph "takes" slower and harder, but compensates for these objections by freedom from any possible infection with tuberculosis, syphilis, or other disease, excepting, possibly, erysipelas.

No fact is better attested than this protection in the case of small-pox. The proof of the degree of it is seen at a glance by observation of statistics in countries and cities where they are most accurately kept. Thus in Sweden the mortality from small-pox in the twenty-four years before the introduction of vaccinia (1801) was 2050 per 1,000,000 annually; after vaccination, 158 per 1,000,000. Drysdale says of Berlin that the mortality in that city during the epidemic of 1872–73 rose to 243 and 262 respectively per 100,000 inhabitants. Thereupon vaccination in the first year of life was made compulsory, and revaccination in the twelfth

year of life, with the result that in the first year of enforcement (1875) the mortality fell to 3.6 per 100,000; to 3.1 in the year 1876; to 0.3 in 1877; and so on for succeeding years, down to 1883, with an average of 1.7 per 100,000. (See Fig. 21.) The nearly absolute protection of vaccination is shown, again, by comparison of cities in which vaccination and revaccination are obligatory and optional. Thus, according to the recent reports of the Berlin Health Office the mortality of small-pox per 100,000 inhabitants in 1888 was in Dresden, 0; in Berlin, 0.07; in London, 0.6; in Munich, 0.75; in Hamburg, 3.58; in Paris, 9; in St. Petersburg, 15.30; in Vienna, 26.15; in Prague, 55.49. Corbally reports that the vaccinated children of Sheffield (1887–88) had, as compared with the unvaccinated children, a twenty-fold immunity from attack, and a four-hundred-and-eighty-fold security against death by small-pox. These facts render further statements superfluous, but a few points may be added from army life. Army statistics are especially valuable from their accuracy. Thus, Schultze shows that since the operation of the German law the annual average cases of small-pox per 100,000 was in the army of Germany, 4.94; of France, 169.72; of Austria, 374.0. During the Franco-German War (1870–71) the mortality of small-pox in the unvaccinated French army was 23,469, while that of the vaccinated German army was but 261. As already stated elsewhere, there has been reported but one case of death from small-pox in the German army since 1874. Morbidity shows the same results as mortality statistics: for since the enactment of the compulsory law in Germany (Schultze) there have been attacked with small-pox annually:

In the German army, up to 1887, 4.2.
In the French army, " 1886, 169.0.
In the Austrian army, " 1881, 317.5.

The immunity conferred by vaccination does not, as Jenner hoped, last for life. Therefore, revaccination becomes a necessity after a lapse of years. The best proof of this necessity is furnished in the fact that revaccination "takes" as a rule; thus, among the soldiers of Prussia, Russia, and Denmark it was successful in 50–70 per cent. of cases. Heim found in five years but 1 case of varioloid among 14,384 revaccinated soldiers, and but 1 case among 30,000 civilians, small-pox meanwhile prevailing in three hundred and forty-four places in which these people lived.

Moreover, the number of epidemics has diminished from 71.4 per century previous to vaccination, increased to 84 during inoculation, to fall to 24 since vaccination. Protection begins on the fourth day after the introduction of the virus, and is perfect on the ninth day. The degree of protection, independent of revaccination, is determined to a considerable extent by the success of the operation and by the quantity of matter introduced—*i. e.* by the number of places vaccinated. Thus, according to Marson, the average mortality of small-pox among all vaccinated persons is 5.24 per cent., while that of individuals showing perfect cicatrices is about .5 per cent. In 6000 cases of small-pox after vaccination observed by Simon in twenty-five years the per-

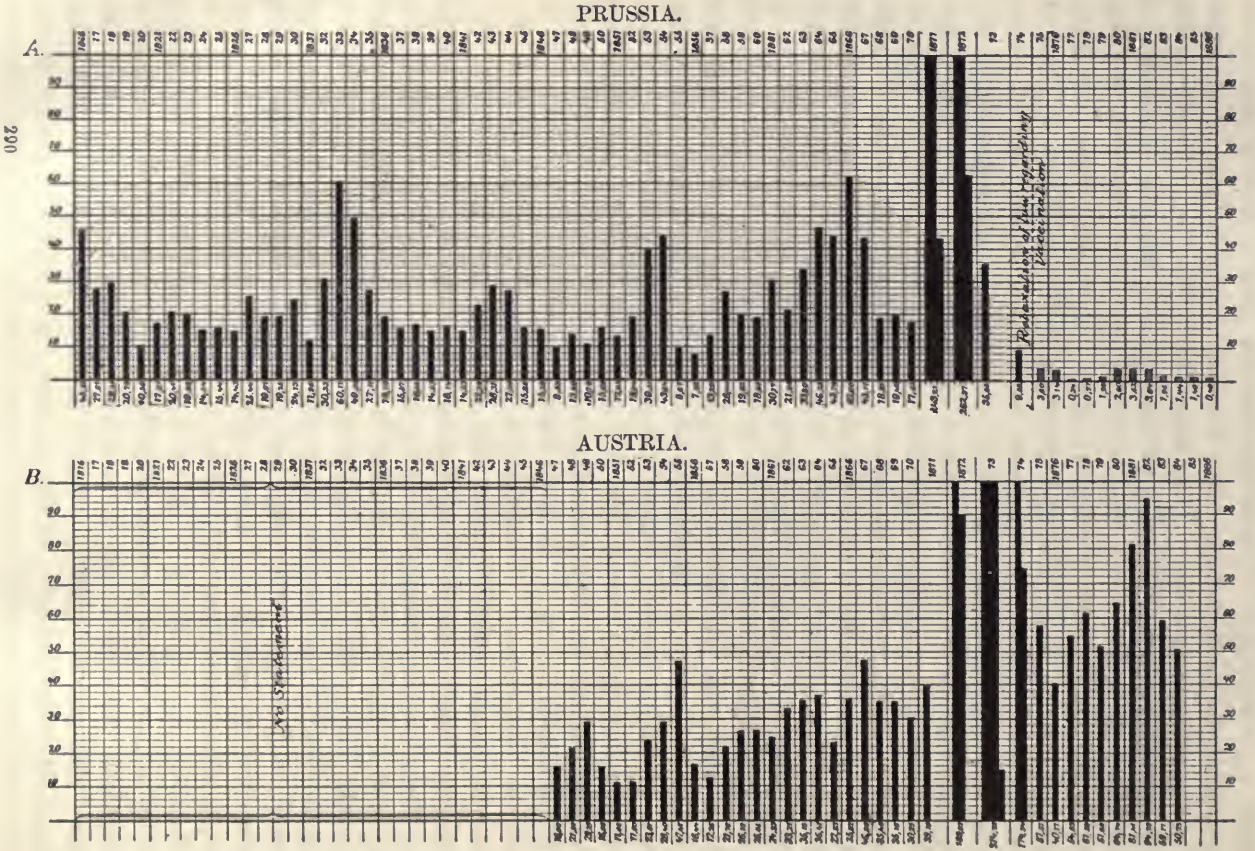


FIG. 21.—Deaths from Small-pox in Prussia and Austria in seventy years (1816–1886), showing number of deaths in every 100,000 inhabitants.

centage of deaths among individuals without cicatrices was 21.75 per cent.; with one indefinite cicatrix, 12 per cent.; with one typical cicatrix, 4.25 per cent.; with two cicatrices, 4.125 per cent.; with three cicatrices, 75 per cent.; with four or more cicatrices, .25 per cent. This fact finds additional proof in the observation that the protection of vaccination, however great, is not so thorough and sustained as that offered by an attack of small-pox itself, whether contracted by ordinary exposure or by inoculation. Vaccination and revaccination once or twice in later life, as at puberty and maturity, protect for life absolutely. The exact duration of protection by a single vaccination cannot be definitely established. It varies in different cases. If revaccination "takes," the individual was certainly liable to take small-pox. The operation is so simple and inexpensive as to justify its practice at stated intervals, and, inasmuch as no case of small-pox contracted within seven years after a successful vaccination—twelve in Germany, according to report of the health office—stands upon authentic record, this period may be put down as the proper interval for absolute protection, with the injunction in all cases that revaccination be performed with every exposure seven years after the last successful vaccination.

The history of vaccination would be incomplete without at least mention of its forerunner, inoculation, which has now closer connection with or relation to vaccination than ever before. No fact could better exemplify the lack of intercourse with the nations of the East than the ignorance of Europe of the practice of inoculation in China and India for three thousand years. The first knowledge of its protective effects was introduced into Europe by letters from the literary celebrity, Lady Mary Wortley Montagu, wife of the English ambassador at Constantinople. She was a personal witness of the method of procedure as well as of its effects, and became an enthusiastic advocate of its protective virtue. On her return to England she had her own daughter inoculated with small-pox matter. As the practice was opposed by men of high repute, she succeeded in interesting the government in her defence to the extent that a promise of pardon was granted to six criminals condemned to death if they would consent to the operation. It need hardly be stated that consent was readily given, and that the men thereby secured double release. In the following year the two daughters of the princess of Wales were protected in the same way. Meanwhile (1721) small-pox was raging in Boston, where inoculation was speedily introduced. It has already been stated that inoculators were most successful when they practised the operation at periods exempt from the prevalence of the disease. The history of inoculation in Boston relates the disadvantage of inoculation during the period of prevalency, for it proved fatal in six cases. Several deaths of prominent persons in England occurred at about the same time, to check precipitately the spread of the new practice. A quarter of a century now elapsed before the operation was practised in any systematic way in public institutions where the poor as well as the rich might receive its benefits. The London Small-pox and Inoculation Hospital was not founded until 1746. During the following half cen-

tury it was the fashion to be inoculated. People made engagements with the inoculators as they now do with the dentists. The wife of General Washington during a visit to Philadelphia took advantage of her visit to undergo inoculation, in which process she had "a very favorable time" (Plant).

There was no doubt of the protection of the individual by inoculation. The proportion of deaths was reduced from 20 or 40 per cent. to 3 in 1000. There was, however, another side. The disease which was introduced was the true small-pox, and each inoculated person was a centre for infection. By the end of the eighteenth century, when the practice of inoculation had become general, the proportion of deaths from small-pox to deaths from all causes had increased from one-fourteenth to one-tenth. With the recognition of this fact the process was interdicted by law both in England and France. This was in 1841, but inoculation was still secretly practised, with the continued production of new centres of small-pox, until the government affixed to it as late as 1860 a penalty of fifty pounds.

Something of the nature of vaccinia-lymph may be learned by study of this history of inoculation. As has been stated already, it was the custom in China and India three thousand years ago to directly produce the disease by inoculation of small-pox matter itself, and secure protection by immunity thus conferred by previous attack. It was the custom in Europe, as also in the early history of our own country, to isolate people in a period of health in pest-houses, and directly inoculate or engraft them with the disease itself, in the hope that the introduction of the poison at a period free from the presence of an epidemic would produce a milder form of the disease. Some of the older variolators became exceedingly expert in this operation. Gatti, the "Jenner of inoculation," brought it to a grade of perfection worthy of being called a science. He was certainly able in a large percentage of his cases to bring about a variola so modified as to be distinguished at times by the absence of any general eruption, and sometimes by the absence of all eruption. He selected his subjects and season of the year as well as his stock of matter. He learned to make the the smallest possible wound without drawing blood, and to introduce his matter in minimum quantity. He selected it also from the oldest case, convinced of the fact that the poison was mitigated by continuous generation. This mitigation of the intensity of the poison is the clue and the key of the whole process of securing immunity against the various infections in modern times, the results of which promise to soon eradicate or modify the forms of these diseases. It was learnt by accident that the mitigation of small-pox virus was precipitated by passing it through the body of a cow. At the present time there is no doubt that the original cow-pox is human pox modified in this way. It is well known also that variola virus may be attenuated in other ways. Thiele of Kasan in the systematic desiccation of genuine small-pox matter, dilution with milk, inoculation, propagation, redilution, etc., through several generations, succeeded in producing a virus whose properties were absolutely identical in every way with the vaccinia-lymph in common use. In fact, the process of generation

can be continued so far as to destroy all infectious properties. This fact was observed long ago in bovine virus, which, when continuously transmitted from calf to calf, finally loses its protective property. To sustain the virtue of bovine lymph occasional resort to inoculation of the animal with human matter became a necessity. The antiseptic fluids, carbolic acid, salicylates, solutions of boric acid, thymol, etc., added to vaccine virus for preservation, gradually reduce its active properties; so too chemically pure glycerin, which is added for dilution, has the same effect of weakening and finally destroying the active principle.

The best lymph from the cow is that which exudes from perfect vesicles before they begin to point. The presence of slight incrustation in the centre of the vesicle indicates the period of greatest virtue. The puncture should be made with a sharp lancet as near the centre of the vesicle as possible, and the fluid collected, as it exudes spontaneously or under slight pressure, in capillary tubes or upon the surface of bone points. Puncture of the margin of the vesicle secures only blood, which is worthless. "Vesicles on which the central crust has begun to form are the most productive, particularly if the crust be small and the margin of the vesicle tender, hot, and tumid: the small superficial vesicles are often more yielding than contiguous larger vesicles, which are more deeply seated or confluent" (Aitken).

Supply is now so abundant and is furnished from so many sources as to secure mankind against any accident by extinction of the natural disease. No place is so distant from a vaccine farm that it may not be supplied in the course of a few days or a week with effective material. In emergency, where a pure lymph cannot be secured, that which has undergone some degree of incrustation, as aggregated lymph found in the immediate vicinity of broken vesicles, may be used as a substitute. It should be clear and colorless like crystals of white sugar candy, or, if colored, but lightly tinged with amber. Central crusts which represent a mass or mould of vesicles, dark brown, but nearly translucent, may also be used. The crust should be pulverized in a clean mortar and preserved in glycerin.

Where human lymph is used vaccination is best practised from arm to arm. The vesicle is punctured at the period of full maturity on the eighth day, never later, and the clear fluid which exudes is collected and utilized as before. Before the general use of bovine virus the material mostly employed was the crust which fell spontaneously from a healthy child. This crust was treated in the same way as that from bovine lymph. Protected from the air, enveloped in rubber cloth and absorbent cotton, and enclosed in well-stopped vials, it retains its efficacy for almost indefinite periods. Thus, Müller of Berlin (1869) made use of cow's lymph which had been originally sent from Holland for the purpose of experimentation, and had been kept dry between apposed glass plates for ten years, with perfect success. The virtue of the lymph is preserved also under considerable dilution. Lymph is usually kept diluted in two parts glycerin and two parts distilled water, the mixture being made by means of a small brush and a watch-glass. Müller found that this

fluid could be further diluted to one part to eight without sacrifice of virtue. In greater dilutions it begins to lose protective property, but vaccination with one part lymph and two hundred of distilled water, if used in abundance, "took" as a rule. Much depends also upon the extent of surface inoculated. Thus, Reiter found the use of a dilution 1 : 1600 successful after introduction upon the surface of an extensive abrasion. Charpie saturated in this solution and applied to the surface of a blister produces definite results. These facts account for the diverse statements of different observers. Thus, Hillier found a dilution of 1 : 10 in glycerin inactive, while Chauveau succeeded with a dilution of 1 : 150 of water.

Failures to secure results diminish in every decade. Thus, up to 1872 the proportion of failures to success was 1 to 120; up to 1876, 1 to 129; up to 1880, 1 to 280 (Cless).

The discovery of the nature of vaccine virus and the mode of infection of the cow affords satisfactory explanation of the first development of the eruption in the cow upon the teats and udder. The disease is conveyed, as a rule, by recently-vaccinated milkers. It explains also, as stated, the protection of vaccination by immunity of a benign attack, and completely takes the ground from under the feet of the opponents of vaccination, who are left as ignorant, but none the less dangerous, possible lepers in a community. Proof of protection may be thus written with the pen, but it has come true, as Jenner said it would, that "the keenest of all arguments for or against the practice of vaccination *will be those engraved with the point of a knife.*"

As already stated, it is a matter of indifference, so far as protection is concerned, whether use be made of human or bovine lymph. Objection was raised against human lymph on the ground that it had undergone degradation. Hebra declared, however, that the lymph used in Vienna produced the same effect as when first introduced. Chapin of Rhode Island made the same observation in regard to matter that had been employed for twenty-six years upon nearly forty-seven thousand persons. With proper care in the selection and preservation of lymph it undergoes no diminution in potency and powers of protection.

A valid objection to the use of human lymph is, however, the possibility of the conveyance of other diseases. Thus, it has been asserted that tuberculosis, syphilis, and erysipelas have been transmitted in this way. The possibility of introducing these diseases with vaccination is unquestioned, although, as a matter of fact, tuberculosis has never been transmitted in this way. The few apparent cases recorded meet with truer interpretation as localizations of bacilli of tuberculosis previously latent in lymphatic glands (scrofula). As to syphilis, there is no doubt. It is admitted that the disease has been introduced in this way by the use of virus from syphilitic infants. It was for a long time maintained that this disease could not be thus conveyed unless blood, pus, or other matter than pure lymph itself had been introduced with the lymph itself. It is, however, now determined that the virus of syphilis may be conveyed with the pure lymph of vaccinia virus. Robert Cory, chief of the Natural Vaccine

Establishment, England, settled this question with a self-sacrifice that finds but too frequent following in other fields. He selected only clear, pure lymph from children who showed unmistakable evidence of the disease in the stage of active eruption. With this lymph he vaccinated himself on several occasions. After repeated failures he succeeded in producing , in the course of three weeks after a last inoculation, a distinct eruption, followed in regular course by sore throat and other unmistakable evidence of syphilis (Plant). The difficulty, as well as the possibility, of transmitting syphilis in this way is proven in this experiment. The smallest precaution as to the selection of subjects suffices to procure protection against this disaster. The transmission of syphilis is easily avoidable by taking matter only from healthy children at least six months old, the ultimate limit of "tardy" inherited syphilis, and all possibility is absolutely excluded by the use of animal matter, as syphilis is an exclusively human disease.

The streptococcus of erysipelas may be introduced with vaccination or may fall later upon the broken surface. The accident is rare in any event, occurring in the practice of the author but twice in twenty-five years, but has been sufficiently frequent during the prevalence of an epidemic of erysipelas, as in Boston in 1854, to justify the suspension or postponement of vaccination.

Vaccination should be done at the age of three to six months, or, in the presence of an epidemic, at any time, even at birth. In case of failure the operation should be repeated at intervals until it is crowned with complete success. Whatever diversity of opinion may prevail as to the relative value of human and animal matter, it is now established that either confers relative immunity for life, and absolute immunity for at least seven to ten years. Animal virus, as stated, takes later by one or two days, and takes harder as a rule—*i. e.* with more inflammation—but its readier supply (a single heifer may furnish two thousand to ten thousand effective ivory or bone points) and freedom from any possible taint of syphilis soon secured for it general adoption. Revaccination at stated intervals—at puberty, maturity, or at any time during an epidemic—robs the question of the value of the kind of virus or the number of simultaneous vaccinations of practical interest.

Points of selection for the operation are about the insertion of the deltoid or the junction of the heads of the gastrocnemii muscles. As a protection against future carelessness regarding revaccination the matter may be introduced in three places, at the angles of a triangle—horizontal insertions at the shoulders permit concealment by a narrow sleeve—at least half an inch distant from each other. Six or eight parallel tracings or strokes, with as many cross-strokes, with the point of a knife so light as to expose the superficial lymphatics and draw little or no blood, afford the best wound, upon which the moistened bone surfaces may be gently rubbed.

Susceptibility is universal. There is no such thing as insusceptibility to vaccination. Seaton never saw it in more than nine thousand cases at the Black Friars National Vaccine Station. Cory confirms this statement with reference later to bovine lymph, and Robertson declares that so-called consti-

tutional insusceptibility is usually a confession on the part of the operator that he has not ascertained the cause of his failures. This fact proves also that there is no real insusceptibility to true variola: escape is due to accident.

The true lesion of vaccination shows all the characteristics of a single typical small-pox pustule. At the end of forty-eight hours the surface of insertion is marked by slight redness and swelling to the size of a large papule, upon the summit of which develops by the third or fourth day a small vesicle filled with a clear fluid. This vesicle is a reticulated sac, the puncture of which—as for the collection of lymph—discharges its fluid contents by slow oozing. It reaches its maximum size by the seventh or eighth day, at which time it is umbilicated and surrounded by a ring of inflamed tissue—the areola—which continues to enlarge for two days, to attain in full development a diameter of one to three inches. The contents of the vesicle now begin to grow somewhat opaque (pus)—to present the appearance on its inflamed base quaintly described by Jenner as "the pearl on the rose." The areola is the evidence of a successful vaccination. By the tenth day the serum is changed into pus, the vesicle has become opaque, and its centre shows yellow inspissation in the form of a crust, which by the fourteenth day extends to convert the whole pustule into a hard, dry mass. The crust falls spontaneously by the twentieth to twenty-fifth day, to leave as a result of the destruction of tissue a characteristic scar. The cicatrix of vaccinia is a more or less circular depression marked by minute pits and radiating lines. It should measure in its diameter fully one-third of an inch. Red or pink at first, its color gradually fades to the bleached appearance of cicatricial tissue, to remain as a mark for life or to gradually disappear in the course of adolescence to the faintest trace. However pronounced, a cicatrix, it is needless to state, is evidence only of destruction of tissue, not of permanent protection against small-pox. The writer recalls a malignant case of purpura variolosa in a young woman whose arms were marked by two typical cicatrices, relics of successful vaccination in early childhood. Slight fever, fretfulness, headache, insomnia, restlessness, disturbance of digestion, lymphangitis as evidenced by swelling of the axillary glands, may be present for a few days at about the time of maturation of the vesicle, to subside rapidly during the period of incrustation. More extensive inflammation, dermatitis, or destruction indicates mixed infection. The constitutional signs are mildest in infancy and increase in severity with advancing years.

Delay in the appearance of the vesicle even to the end of a week does not preclude success, provided the subsequent phenomena appear in course. Accelerated, abortive, so-called "spurious" vaccinations differ in various ways, and furnish only partial, limited, or no protection.

It is estimated that at the present time twenty-two million people are vaccinated every year.

VARICELLA.

By JAMES T. WHITTAKER.

Varicella or varicellæ—diminutive of varus, pimple, pock; chicken- (French, chiche; Latin, cicer, insignificant) pocks or pox; water-pock, wind-pox; variola notha, spuria; false pox—is a trivial acute infection of childhood, distinguished by a long period of incubation, absence of prodromata, slight fever, a vesicular eruption varied in size and short in duration; as a rule, without complications or sequelæ.

Chicken-pox was first described under the term crystalli by the Italian anatomists—Igrassias in 1575, Guido Guidi in 1585—and received its present unfortunate name from Vogel in 1764.

History.—Fuller (1730) and Heberden (1767) made the first attempts to separate this affection from variola (varioloid), with which it had been formerly confounded, and has been so since by many authors (Hebra, Thompson) "with inconceivable persistence" (Thomas)—a mistake which resulted in complete confusion regarding the nature of both affections, and in reproach and disrepute of vaccination in the early years of its practice.

As early as 1690, Morton, who introduced later the term "chicken-pox," is said to have described a case of varicella under the title "*variola maxime benigna*," and Jennings declares that at this time the disease was distinguished by the people from small-pox. Opposition to inoculation toward the close of the last century concentrated attention upon the milder forms of small-pox, and the practice of inoculation—which was, by the way, a very lucrative procedure—depended upon the separation of varioloid and simulating affections. Willan (1798) discussed the eruption in detail, describing the vesicles as acuminate, conoidal, and globate. With the introduction of vaccination it again became necessary in its defence to separate cases of varicella, but, notwithstanding all the study that has been put upon these infections, the difference between varicella and varioloid has been at times so little marked as to have led certain eminent authorities to regard them as identical, or to look upon chicken-pox, as Morton put it, as the most benign variola. Kaposi and Bruyelle still support this view.

Nature and Etiology.—Gee declares that there is not upon record a single authentic instance where varicella resulted from variola, or *vice versa*. So, too, epidemics prevail entirely independently of each other. Mohl emphasized this fact by the statement that small-pox was entirely absent in Copenhagen from 1809 to 1823, while chicken-pox was frequent every year. Successive epidemics are very rare in varicella, but very frequent in small-pox. Inoculated

small-pox produces at times a very light form of variola, but the form is never so light but that it may be distinguished from varicella. Both variola and vaccinia protect against variola, but not against varicella. It is impossible to conceive of the occurrence of a case of varioloid, however benign, immediately or shortly after an attack of small-pox of any kind or shortly after a successful vaccination, but infinite are the cases in which chicken-pox has followed upon the heels of variola or has occurred in the course of or soon after vaccinia. Varicella is a disease of childhood almost absolutely. Variola is a disease of chilhood by preference, but does not spare the adult unprotected by previous attack or by vaccination.

If varicella is but a modified variola, there should be upon record at least one authentic case of communication of this disease to some adult member of a family. Hochsinger thought that he had seen such a case when a boy, æt. 10, affected with varicella (together with fourteen of his school-mates), communicated chicken-pox to his brother, æt. 14, who had not been at school, and small-pox to his mother, æt. 40, who had not been away from her home. The small-pox case ran a typical course, and the author concluded from this observation that the old Hebra-Kaposi doctrine of identity was thus re-established. Close study of these cases convinced Thomas, however, that they were all of them mild cases of varioloid. Varicella very rarely attacks a whole family, and still more rarely those of the ages mentioned. Henoch says that he never saw an undoubted case in an adult. It is known, moreover, that individuals who have been vaccinated, or even revaccinated, may, after a certain indefinite period, suffer attack of the lightest possible true variola, which may run its course without or with almost no fever, and be marked by an eruption of papules or vesicles, but no pustules at all. Thomas looked upon these cases, therefore, as the very mildest possible forms of true small-pox, the so-called "variola vesiculosa."

Inoculation of varicella, when it succeeds, invariably produces varicella, never varioloid, while inoculation of varioloid invariably reproduces itself or variola, and never varicella.

The study of the points of resemblance and difference of these two affections is very interesting, not only from the standpoint of differential diagnosis, but also because it throws a side light upon the all-important relation of vaccinia to variola. Bollinger says "the small-pox which has been described in dogs has a much stronger resemblance to varicella than variola." Dogs do not contract small-pox.

The recognition of the fact that an attack of one disease secures future immunity from itself, but does not protect against the other, finally led to a distinct separation of the two affections. Confirmation of this view was also obtained in the fact, as stated, that vaccinia does not prevent varicella, nor varicella vaccinia. Czakert, after three failures in the ordinary way, succeeded in vaccinating a boy æt. 4 by introducing lymph into the interior of vesicles during an attack of varicella.

Varicella appears in sporadic and endemic (rarely epidemic) form, and epi-

demics never assume the range nor show the intervals of measles and smallpox. The disease does not die out entirely in large cities, but assumes somewhat of epidemic proportion once or twice a year on the opening of schools and kindergärten. It is confined exclusively to childhood (exceptions having been noted by Heberden, Gregory, and Seitz) up to the age of twelve, and is rare after ten. The short-lived contagious principle, probably from the vesicles, is believed to be inhaled (contagium halituosum). Infants are never born with it.

Inoculation experiments fail oftener than they succeed. Thus, Hesse failed in 87 cases, succeeded in causing a local eruption in 17, and a general eruption in 9. Steiner claims to have succeeded eight times in ten trials, but was unable to propagate the disease from any case. Tenholdt found in the contents of vesicles a micrococcus which, inoculated in man, produced light redness and swelling like that of spurious vaccinia, and in one case a vesicle smaller than a sudamen, the affection remaining local. Pfeiffer found in fresh vesicles of thirty cases, without exception, a parasite showing an amœboid stage, a cystic stage with spore-formation, and, after the development of numerous spores, a return to the amœboid stage. Inoculation with the contents of vesicles showed three times in five days a localized, circumscribed varicellar exanthem, recurring in a scattered manner up to the eighth day. The parasite could not be cultivated upon any culture soil.

Symptoms.—The incubation period varies from eight to seventeen days. Prodromata, in some form of light malaise, occur only very exceptionally. In rare cases they may assume prominence, and there may be headache, vomiting, and high fever. Henoch once saw a case begin with convulsions.

The disease is announced, as a rule, by the eruption, which shows itself in the form of spots of hyperæmia, in the centre of which appear, in the course of a few hours, distinct but slightly elevated vesicles, which attain their greatest circumference in the course of from three to twenty-four hours. The vesicles contain a clear, sticky serum of neutral or alkaline (never acid, as in sudamina) reaction, which fully distends the vesicle, and which exudes slowly, but not wholly, on puncture of the sac. The serum shows under the microscope a few pus-cells, which, when exceptionally present in greater quantity, may make the vesicles appear like drops of wax. In lighter cases, without halo, the patient looks as if sprinkled with drops of water.

The eruption shows itself first upon the neck and chest (face, according to Thomas), to spread subsequently over the face and scalp, trunk, and extremities, and shows itself always in successive crops, to the number of ten to fifty, or as many as two hundred to eight hundred, over the whole body, irregularly, never uniformly or at once.

The vesicles vary also in size, usually from a pin-head to a pea, exceptionally from a dime even to a dollar. These large vesicles are, however, always lax, never full, as is the case in the blebs of burns, blisters, and pemphigus. Distinct, isolate, and irregular elsewhere, they may show aggregation like zoster upon the extremities, but are very rarely confluent at any part. They

are very superficial, lifting only the upper layers of the epidermis, and penetrate to the rete Malpighi in only exceptional cases. Hence they but rarely show an umbilicus and seldom leave a scar.

A peculiarity of the eruption may be its simultaneous appearance in different parts of the body. It does not begin definitely; it does not show any regular grouping or course in its progress over the body. In places the vesicles may stand apart, while elsewhere they may be closely grouped. Closer contact is most frequently observed in parts subjected to pressure or heat, as on the back or about the tuberosities of the ischia, where the eruption may be grouped to resemble zoster. Decubitus on one side may determine the eruption to that region. Any surface of local hyperæmia or surface subject to tension, as from an underlying abscess, may be thickly covered.

The eruption is at first, as stated, macular. It occurs as rounded red spots, like the rose spots of typhoid fever (Trousseau), in the centre of which appear, unlike the roseola, a vesicle of about the size of a pin's head. The vesicle appears so rapidly as at times to seem to form at once, or assumes magnitude in the course of an hour or two sufficient to soon cover the base or hide it, so that it is not surrounded with a halo as in the case of small-pox.

The eruptions of varicella and varioloid present morphological differences. Varicella is a vesicle from the start: it does, in reality, appear as a macule, but the vesicle forms upon its surface so soon as to cover it at times in the course of an hour or two, so that the macular state, as a rule, is not seen at all, and chicken-pox is said to appear with vesicles. Such rapid change into vesicles is never the case in small-pox. Variola begins always with papules. It also really begins with maculæ, which become papular in the course of a few hours or by the time of the first observation; the papules are converted into vesicles in the course of a few days, and vesicles grow so slowly over the base as to be surrounded with a more or less distinct halo of hyperæmia. The vesicle of chicken-pox is a single sac which nearly collapses upon puncture; that of variola is multilocular and collapses only partly upon puncture. It must, however, be admitted that occasionally vesicles in chicken-pox are not so simple—that among many will be found a few which may undergo pustulation, and may show umbilicated surfaces and actually leave cicatricial deformities. The fact must also be recalled here that many cases of variola are maxime-benigna, that the eruption may stop at the papular stage, and that it is very frequently aborted before the pustular stage. These are cases which Thomas calls the "*variolosa vesiculosa.*"

The eruption may last two to five days, when the residue desiccates to leave a light pigmentation, very exceptionally ulceration (Hesse), which gradually fades to leave no trace. Through premature rupture air may enter a vesicle to produce a condition known as varicella ventosa or emphysematosa or wind-pock. Occasionally, as stated, a few vesicles undergo pustular change, in which case they become opaque by the end of the second day. The pustule then begins

to desiccate by the third day, to collapse later into a brownish crust and leave pigmented reddish spots for eight to fourteen days. The crusts disappear, as a rule, to leave no scars. Now and then a bigger pustule will have invaded the deeper structure of the skin to produce a permanent lesion, so that a few well-marked cicatrices about the forehead or eyelids may cause as much deformity as a case of varioloid. The isolated, soft, chalk-white, superficial scars seen upon the forehead, side of the face, or about the mouth in children are generally relics of varicella.

The eruption of varicella is irregular in every regard. It does not all come at once: it does not all disappear at once. New groups may appear in irregular succession, and vesicles may show themselves in one part of the body side by side with traces of previous eruption.

The eruption may also show itself on various mucosæ, as in the eyes, to produce conjunctivitis or keratitis; in the mouth and palate, to cause stomatitis; in the pharynx, to lead to more or less dysphagia, and induce, at times, swelling of the cervical glands; on the vulva and prepuce, where it may show itself as a string of vesicles on the inner aspect of the labia majora; or at the frenum, to give rise to pain in micturition. Vesicles which appear in the mouth, especially on the tongue, are readily broken to show irregular ragged abrasions, sometimes with aphtha-like surfaces.

A slight rise of temperature, maximum 102° F. (exceptionally 106°, Heberden), with associated symptoms of fever, headache, insomnia, anorexia, nausea, etc., attends or may attend the eruption, to continue with it two or three, or exceptionally as long as five, days. Defervescence is by crisis, without subsequent elevation or interruption. Very light cases may show no fever at all. Relapses and recurrences are possible, but not probable.

Hutchinson described a grave varicella which occurs most commonly in weakly, ill-nourished children. The vesicles, instead of drying up in the ordinary way, grow blacker and larger, to present the appearence of round black spots, of the diameter of an inch or more, scattered over the body. These crusts cover underlying ulcers, which sometimes extend through the skin and subjacent muscular tissue. These cases are said to be very fatal. They may be attended with eye complications—irido-choroiditis and loss of sight. This variety must be exceedingly rare, as it is not mentioned by other authors than Eustace Smith, who connects it with the curious tendency to gangrene seen in certain children. It is probably the result of a mixed infection, and has no more to do with genuine varicella than a coincident erysipelas or other dermatitis. Hæmorrhagic varicella has been observed (Andrew) as a special complication in cachectic cases. Varicella may occur in connection with other infections—with measles, scarlet fever, diphtheria, pertussis, and even with variola (Sharkey).

Diagnosis.—Inasmuch as varicella was so long, and is often yet, mistaken for variola (varioloid), the question of differential diagnosis assumes supreme importance. The diagnosis demands, first, a knowledge of the existence of either disease in the vicinity or community, and a definite history of the pre-

existence or absence of either in the individual, together with the period of the last successful vaccination; second, the age of the patient, as variola occurs at all ages, and varicella is almost confined to childhood. Variola is preceded by prodromata—malaise, fever, headache, backache, sometimes by initial rashes—and is attended by a characteristic eruption on the third day; varicella announces itself by its eruption without prodromata. The most anxious mothers seldom notice illness of any kind until the eruption appears. The physician is called to decipher the eruption.

Varicella appears, as a rule, first upon the back, neck, and chest, or, if upon the face, irregularly over it and irregularly over the body. Variola appears, as a rule, first upon the face, forehead, to extend over it regularly from above downward, thence to spread uniformly over the neck, chest, etc.

The superficial vesicles of varicella contain only serum; the deeper-seated vesicles of variola contain serum, and later pus.

The eruption of variola is much more uniform in size; that of varicella varies greatly.

Varicella is rarely confluent anywhere, and its vesicles are only exceptionally umbilicated. By the end of the third day spots of hyperæmia, fully-developed vesicles, and crusts may be perceived simultaneously and side by side in varicella, whereas the variations in the age of the eruption would be observed only at points distant from each other in variola.

The eruption of varicella may be abundant anywhere over the body, the face, trunk, or extremities; the eruption of variola is most abundant upon the face and fingers. A thick eruption upon the fingers has often established the presence of variola.

Fever precedes by several days the eruption of variola, to fall with its appearance, whereas fever occurs only with the eruption of varicella, to increase with its development. Variola shows in its further course secondary fever, this being absent in varicella.

There are exceptions to all these rules, but these form in their ensemble almost unimpeachable evidence. The cases about which may still hover any doubt or uncertainty should be considered as variola to secure proper protection of others by vaccination.

Prophylaxis and Treatment.—The mortality of varicella is practically *nil*. Trousseau says that no physician has ever seen a patient die of chicken-pox alone; yet, inasmuch as complications, fatal hæmorrhages, catarrhal pneumonia (Meigs and Pepper), nephritis (Hutchinson and Henoch), have been recorded as coincidences or complications, delicate children may be protected by removal from the area of infection or isolation of patients in separate rooms. Patients should remain indoors, if not in bed, during the existence of the eruption, and should not be permitted to return to school until all signs of it have disappeared. Vesicles, especially when extensive, or pustules on exposed surfaces should be treated with consideration to prevent or limit subsequent lesion. It is advisable to touch the surface of such vesicles with carbolic acid and glycerin, *āā*, to secure, if possible, speedy coagulation of their contents and destruction

of pus-producing micro-organisms. Where the eruption is unusually abundant, as in the face, the whole surface may be bathed in sublimate solutions, 1 : 1000–5000.

Other treatment is superfluous, or does not differ, if called for by complications, from that discussed in considering varioloid.

MUMPS.

By JAMES T. WHITTAKER.

Mumps (from Danish mompen, whence our words mum, mumble); Parotitis epidemica; Fr. Oreillons; Ger. Schafskopf, Ziegenpeter,—is an acute, contagious infection, of short duration and little gravity, distinguished by painful inflammation of the parotid gland and vicinity, sometimes also by orchitis.

Mumps was known in the remotest antiquity. Hippocrates mentions the disease, and the older physicians associated it with measles, scarlet fever, whooping cough, etc. as an affection of childhood. It was observed then, as frequently since, that the disease prevails in epidemic form, and that epidemics are wont to precede or follow outbreaks of some of the exanthemata or other affections of childhood.

A special affection or infection of the parotid gland which occurs in individuals in the course of, or as a sequel to, many of the graver infections, septicæmia, typhus, typhoid, or puerperal fever, etc., is set apart and distinguished from the epidemic parotitis as a metastatic inflammation. A subvariety of this form may follow intestinal or pelvic lesions. Paget collected a number of cases of parotitis apparently independent of septic infection—*i. e.* unattended with suppuration after injury or disease of various abdominal and pelvic organs

The older writers, Fourcroy and Portal, attributed the flow of saliva which has been observed in certain cases of inflammation of the pancreas to a kind of connection, or "sympathy," between the buccal and so-called abdominal salivary glands. Canstatt speaks of metastasis of mumps to the pancreas in the same sense as to the testes, ovaries, and mammæ, and Andral and Mondière do not hesitate to declare that the parotid gland swells in disease of the pancreas. Schmackpfeffer actually reported a case wherein an autopsy revealed a pancreatitis in explanation of a parotitis. In most of the cases, however, inflammation of the parotid was assumed to account for a ptyalism which was oftener due to gastric catarrh, or, as in the last case mentioned, to mercury given for syphilis. Yet after a full review of this subject Friedreich is not willing to dismiss the possibility of such metastasis—a question which must be decided in some future extensive epidemic of mumps or fortunate opportunity at autopsies.

Etiology.—Parotitis epidemica, our common mumps, shows certain peculiarities of resemblance to and difference from other ordinary infections. There is, in the first place, remarkable predilection for the colder seasons of the year.

Of 117 epidemics tabulated by Hirsch, 51 occurred in winter, and of 99 studied by Leichtenstern, 42 were in the first quarter of the year. The disease shows also a preference for certain localities, in which it may prevail continuously or recur with every accumulation of fresh material. Great variation is shown also in its extent or range. It remains confined to certain institutions, boarding-schools, orphan asylums, barracks, etc., or, again, extends over or is circumscribed to a certain quarter of a city, or ranges over the entire city and surrounding country. Epidemics may be extinguished in the course of a few weeks or prevail throughout the greater part of a year. The disease shows some predilection for soldiers, probably on account of close association in barrack-life. Some of the best reports are furnished by the military surgeons (Bruns). It attacks males always more frequently than females, and is at times limited to children, or, again, spares no individual unprotected by previous attack except sucklings and old people, who almost universally escape. The age of preference is from two to ten. In a house full of children mumps usually begins with the youngest first, successively seizes the older children, and may afterward attack adults. Liability of males is nearly universal. The disease has often been known to attack 90 per cent. of the residents or inhabitants of public institutions, schools, barracks, etc.

Mumps is undoubtedly contagious, and probably, as no other explanation seems possible, through matter expectorated from the mouth to contaminate the atmosphere in the vicinity of the patient. It may attack animals (dogs) with active salivary glands. Poore declares that a boy aged seventeen, affected with mumps and five days later with inflammation of the testicle, which suffered atrophy, communicated the disease to a dog, his constant companion and bedfellow. The dog began to show symptoms in fourteen days exactly like those of his master, including subsequent infection of the testicles, which likewise suffered atrophy. Thenceforth the dog took no pleasure in the society of other dogs, which he seemed to shun, and in his disgust forsook his old master for a new one. Roth declares that third parties may carry the disease.

Whether mumps be a general or local process is a question difficult to decide, though it has been nearly definitely decided in what way the poison of the disease penetrates to the parotid gland. With the older writers the view prevailed that the disease was a general infection with localization in the salivary glands, as measles, scarlet fever, and small-pox were taken to be general diseases with localizations in the skin. From the nature of the disease there can be little doubt that the cause is a micro-organism which infects the blood, or which, from its nidus in the parotid gland, evolves toxines to produce fever and the other general symptoms of the disease. The evidence actually in our possession goes to show that mumps originates in the mouth, and not in the blood—that the poison of the disease is conveyed to the parotid gland through the duct of Steno, and not through the blood-vessels. This fact seems to have been proven of metastatic parotitis, where *a priori* reasoning would certainly indicate derivation of the poison from the blood. What lends special support to this view in epidemic parotitis is the fact that the disease is

found associated almost universally with stomatitis or some form of sore mouth or sore throat. Ziem thinks that it is from the nose, as he finds it always in connection with, or as a sequel to, nasal catarrh. This subject is discussed later under Metastatic Parotitis.

Soltmann attributes the exemption from mumps of infancy and old age to the fact that the duct of Steno is too small in infancy, and too atrophic in old age, to permit the entrance of noxious matter.

Morbid Anatomy.—As the disease has in itself no mortality, and as all signs of hyperæmia disappear after death, study of the lesion of mumps is difficult. It is rare to find a case upon the post-mortem table, and opportunity for observation may be furnished only in cases of death from accident or intercurrent disease. Intercurrent disease is not common. Pure mumps has no lesion. Foerster states that the most manifest condition is hyperæmia. The gland appears red on its cut surface. Serum exudes freely, so that the granular aspect is lost. Virchow considers the affection as a catarrh of the epithelial structure. The succulence imparted to the gland by the excessive hyperæmia and exudation of serum gives to it the doughy feeling different from the fluctuation of pus.

These processes subside entirely with the process of resolution, to leave no trace. The disease may extend to involve also other salivary glands, the submaxillary and sublingual, and always in marked cases the interglandular connective tissue, to produce the distensions and deformities characteristic of mumps.

Metastatic parotitis shows much graver lesions. The tubes and acini are swollen and reddened. There is the same extensive infiltration of the connective tissue, but the disease is soon distinguished by the accumulation of pus, some of which may by pressure be forced out through the duct of Steno into the mouth. The pus accumulates in the form of multiple abscesses, which break into each other to finally reach the surface of the skin or penetrate deeply into the tissues of the throat. Pus may thus burrow toward the external auditory meatus and break the surface to discharge from the external ear, or an abscess may penetrate the anterior mediastinum or force its way inward and upward through the base of the skull to involve the periosteum, attack the bones, and reach the membranes of the brain. Involvement of the middle and internal ear, with destruction of the bones of the ear and lifelong deafness, is a deplorable catastrophe of occasional occurrence. The course of the facial nerve seems especially fitted to carry infectious matter to the auditory apparatus, as the twigs of the trifacial favor its transportation to the brain (Vogel).

The cause of mumps, necessarily a micro-organism, although not yet isolated, penetrates to the parotid either by means of its ducts through the mouth or through the blood after inhalation to the tissues of the gland. The first is the most probable mode of genesis, but whether the process be chiefly catarrhal or interstitial is not yet quite clear. The contagious principle or cause of the disease finds a nidus also in certain cases in the other salivary glands and in

an organ as remote as the testicle. Baginsky saw in the same family, in one boy aged seven years, the parotid alone swollen, and in another, aged ten months, the submaxillary alone. The point of interest in both cases was the fact that they both coincided with varicella. Penzoldt and Soltmann both speak of affection of the other salivary glands, and Henoch declares that he had charge of a case in which both submaxillary glands were affected with subsequent orchitis, but without any involvement whatever of the parotids.

One attack secures future immunity as a rule.

The period of incubation ranges from ten to fourteen days. It may be as short as seven or as long as twenty-one days. English writers put it at a fortnight, and prove it by a fine illustration offered by Harley: A medical student had mumps in London at a time when his mother was staying with him. Mother and son remained in London until all swelling disappeared, and then went a hundred miles into the country, home. There was no mumps in that neighborhood, but a *fortnight after the arrival* one of the children was taken with the disease, and it afterward successively affected, *at regular intervals of a fortnight*, each member of a large family. The story is told as taken verbatim from Hooper's *Physician's Vade Mecum*, 7th ed. p. 558 (Aitken). The period of incubation is usually entirely free of symptoms.

Symptoms.—The disease sets in with chill or shivering fits, followed by fever of 102° to 105° F., and may often be preceded for a few days by malaise, anorexia, headache, and neuralgic pains. Coincident with the elevation of temperature is the pain, the localization of which distinguishes the disease. Shoots and stabs of pain are felt at the angle of the jaw, radiating to the temple and the ear. The parotid gland swells: it fills up the space between the mastoid process and the angle of the jaw, mounting over the side of the face and extending over the cheek and down the neck, with such a degree of tumefaction as at times to obliterate the natural outlines. By this time there is such interference with the action of muscles and the excursions of the jaw as to close it, so that often the handle of a spoon cannot be inserted between the teeth. The pain, on account of the tumefaction, tension, and interference with the circulation, always severe, is sometimes excruciating, and is of course greatly aggravated by every attempt at motion of the jaw and deglutition, or even at times by the sight or odor of food, which may stimulate the salivary glands. The inflammation extends also through all the tissues of the neck, and is manifest often in the throat and mouth by marked redness and swelling, sometimes by actual displacement or partial occlusion of the palate, pharynx, and larynx. The lobe of the ear is lifted and carried forward. The whole head may be pushed over to one side. The swelling reaches its height, as a rule, by the fourth day, when with the fall of temperature it begins to subside, and subsides so rapidly as to have almost entirely disappeared by the sixth to the eighth day, unless, which is not infrequently the case, the opposite side takes on the same swelling to repeat the same process. Much more rarely the affection is bilateral from the start. In such a case deformity is most pronounced. The cheeks, the jaws, the neck form a vast, tumefied, œdematous,

indurated mass, and the suffering from distension becomes correspondingly great. A peculiar, characteristic, and not the less striking because somewhat comical, picture is thus presented by an individual affected by mumps.

The inflammation or infection of the testicle is the most interesting complication of mumps. The organ is usually affected after the process in the parotid has subsided, sometimes coincidently, still more rarely alone as the sole sign of the infection. It is the testicle itself which is invaded (orchitis), very rarely the epididymis or the cord, and then only after puberty. In double mumps the right testicle, in single the organ on the side of the affected parotid, is most frequently affected. Double orchitis is rare. Affection of the testicle is revealed by a sensation of weight and pain in the gland and along the cord, by fever, and sometimes by vomiting. The testicle soon becomes swollen and tender, and the scrotum is often reddened and œdematous. Strange to relate, the existence of a gonorrhœa during an attack of mumps rather repels than invites attack. Liability is not increased by the severity of the mumps. Orchitis may occur in the lighest case.

Frequency of attack varies greatly. Granier saw orchitis develop 115 times—*i. e.* 23 per cent.—in 495 cases from military life, while Luehe saw it but once in 116 cases, and then in a youth of sixteen in a school of young cadets. Brown records orchitis 10 times after 20 cases of mumps in a military school, 9 times on the side affected, and once on the opposite side, with subsequent affection of the same side. Homén speaks of cases of orchitis at the early age of twelve and fourteen, followed by atrophy.

The process usually subsides without damage, though it sometimes results in atrophy, a catastrophe that may be prevented at times by faradization of the testicle on the subsidence of acute inflammation. Urethritis with blennorrhœa has been also noticed with œdema of the scrotum, and in women, very exceptionally, oöphoritis with leucorrhœa and swelling of the external labia and the mammary glands.

Mumps, though considered a light infection, is liable to certain very grave complications. Sudden deafness may set in from labyrinthine disease, and serious affection of the brain ensue from interference with the circulation or poisoning by toxines.

The disease may announce itself with deafness, due usually to catarrh of the middle ear. The catheter may in these cases reveal the presence of fluid in the cavity of the drum, and inspection disclose hyperæmia of the membrana tympani. Menière and Moure reported cases of permanent deafness after mumps, and Kosegarten claimed to have prevented grave lesion by the administration of the infusion of the leaves of jaborandi. Deafness may also occur in the course of mumps from transmission of infectious matter to the labyrinth through the fissura Glaseri (Roosa).

Musgrove (Austinville, Texas) reported in a very old lady, aged 84, a case of parotitis acuta duplex which ran a regular course up to the sixth day, when she suddenly fell into stupor, with jactitation and stertorous respiration. She roused from the stupor sufficiently to swallow fluids, but died on the following

day. Percy Smith (London) reported two cases of mental alienation, one in a young merchant, and one in a medical student, who suffered also with orchitis. In the first case there set in after the eighth day insomnia, which developed into acute mania that lasted for four months; the second case developed melancholia and suicidal mania, which, however, also entirely disappeared. Both cases experienced extreme prostration during the mental malady.

Other complications recorded are hyperæmia of the brain from pressure on the jugulars, meningitis, amblyopia and color-blindness, conjunctivitis, laryngeal stenosis, albuminuria, hæmaturia, nephritis. Eichhorst quotes with an interrogation point a case of endocarditis reported by Isham, and from Colin a case of uræmia and death. Michaelski saw a death in convulsions. Palsy of accommodation was seen once in an extensive epidemic (Boas), and paralysis of the limbs was reported once by Joffroy. Gowers thinks that diphtheria may have been the disease in both cases, and mixed infection might account for many other complications mentioned. With all this list it must not be inferred that mumps is a grave disease. The author, in the practice of a quarter of a century, has never seen any complication other than a trivial and transitory orchitis.

The **diagnosis** is usually easy, and is helped in any doubt by the existence of the disease elsewhere. The extreme swelling and pain, with closure of the mouth, lifting of the lobe of the ear, and torsion of the head, distinguish the affection. Lesser swelling with less pain may necessitate inspection of the throat in elimination of scarlet fever, diphtheria, or quinsy. Digital examination would detect a retropharyngeal abscess, which might extend to involve the connective tissue about the jaw. A lymphangitis or simple adenopathy from infection of the throat, a very common affection, may be nearly as extensive and painful as mumps. It is usually seated or arises lower on the neck, has no definite duration, and is much more prone to suppuration.

Metastatic Parotitis.

Metastatic parotitis occurs, as stated, in connection with, or in the course of, the more grave infections, such as typhus, typhoid, and relapsing fever, yellow fever, pyæmia, measles, scarlet fever and small-pox, pneumonia and dysentery. The disease has hitherto been regarded strictly as a metastatic process due to transfer of diseased products from a distant seat. Recent investigations, however, go to prove that the affection begins in all cases in the mouth. Hanau made a special study of the genesis of five cases of suppurative parotitis which occurred as a secondary process in consequence of septic infection. These studies were made especially to determine the question whether the disease was due to the migration of the micro-organisms from the mouth or as a result of metastasis from the blood. The micro-organisms encountered were in all cases micrococci, which in their arrangement were clearly staphylococci. They were always found in the excretory ducts or in the abscesses which had arisen in connection with them, while the blood-vessels, lymph-vessels, and acini were entirely free. Dittrich discovered in fatal suppurative

parotitis the staphylococcus pyogenes aureus as the sole cause of the disease. The micro-organism was found only in the ducts, never in the vessels.

The process is thus carried from the orifices through the tubes to the substance of the gland. The mouth has always been recognized as a reservoir for all kinds of micro-organisms. In disease, especially in fever, conditions accumulate to secure their retention and multiplication, and the discovery of this origin of parotitis is of great value as showing the necessity of regular and thorough disinfection of the mouth in disease as well as in health. It is of interest to note in this connection that Testi found in a fatal case of pneumonia, which developed in its course unilateral pleurisy, multiple abscesses of the skin, and a bilateral parotitis, the diplococcus of Fränkel in the contents of the abscesses in the gland. The finding was verified by cultivation and inoculation.

The **symptoms** of metastatic parotitis do not differ at first from those already described of the more benign but contagious form of the disease. The gland is harder; the inflammation is more circumscribed. The doughy sensation produced by the intense hyperæmia and the excessive exudation of serum are substituted by the characteristic induration and subsequent fluctuation of the suppurative process. Resolution almost never occurs in metastatic parotitis, and suppuration shows itself in the course of a few days.

Mumps requires but little **treatment**. Confinement to the house, if not to bed, applies of course to every case attended with fever. Light diet from necessity, as thin milk, soups, soft-boiled egg, custard, suffices to secure nourishment without strain upon the inflamed tissue or disturbance of the stomach. Hot emollient applications, hot oils, olive oil, cod-liver oil, cocoa butter, vaseline, etc., the lead-and-opium wash bound in with oiled silk, flannels wrung out of hot water, hot, not too heavy, poultices of flaxseed or slippery elm, best relieve the tension and pain. Gargles of hot water with salt, repeated every half hour or hour, serve as poultices applied nearer to the seat of the disease. A saline laxative, a dose of calomel, an antipyretic, quinine 2 to 5 grains, phenacetin 3 to 10 grains, broken doses of Dover's powder 2 to 3 grains every two to six hours, best protect the patient during the short duration of the disease. Some mode of light suspension, especially in the recumbent posture, gives great relief to a developing orchitis. Faradization, as stated, may prevent atrophy. Affections of the ear and brain call for special treatment, though little hope of relief of a deafness which sets in over night may be entertained.

Metastatic parotitis is treated in the same way, with address to the remote origin of the complication and speedy evacuation of accumulated pus.

WHOOPING COUGH.

By JAMES T. WHITTAKER.

Pertussis (*per-*, intensive; *tussis*, cough); Tussis convulsiva; German, Keuchhusten, Stick-husten, Kinn, Kink, Kind-husten (panting, suffocating, child's cough); French, Coqueluche (used also for influenza), from *coqueluchon*, a cape worn by patients; Italian, Tosse asenine,—an acute infection of childhood, distinguished by paroxysms of cough in rapid series threatening suffocation, terminated by a long-drawn, audible inspiration.

The name is derived from the fact that the cough is distinguished by a prolonged, forcible, and audible inspiration through a spasmodically contracted glottis. Many cases of whooping cough, however, exist without this characteristic sound, and where different stages of the affection may be recognized the sound is absent during the whole of the first and most of the last stage. The cough consists of a series of short, sharp explosions, spasmodic in their character, a series of expiratory efforts without stop to catch the breath, until finally, after the lapse of from fifteen to sixty seconds, at the point of exhaustion occurs this prolonged, audible inspiration. It is the series of explosive coughs in quick and uninterrupted sequence, the short, sudden cough, the staccato cough, which marks a case of whooping cough.

History.—The origin and home of whooping cough are involved in obscurity. According to Mason Good, the disease was known to the Greeks; but their descriptions, as well as those of subsequent writers, do not distinguish it, strange to say, from other spasmodic or catarrhal affections. All authors agree that the disease was certainly definitely described by Baillou (Paris, 1578) as an epidemic of a cough "qui tussis quinta seu quintana quod certis horis repetit." Baillou spoke of it as a well-known malady. For the most part, however, it was still confounded with bronchitis, croup, and influenza up to and even into the eighteenth century. Danz published the first compilation in a monograph in 1791. Cullen accurately and graphically described the disease as a contagious malady attended by cough, convulsive and strangulating, with sonorous inspiration, frequently repeated, and often followed by vomiting—"morbus contagiosus, tussis convulsiva, strangulans, cum inspiratione sonora, iterata, sæpe vomitus." By the end of the eighteenth century reports of epidemics began to multiply. It seemed to spread from different centres in Europe, prevailing with greatest frequency and intensity in the colder climates. The largest number of deaths were reported from Norway, Sweden, and Denmark, though in Ireland it ranked, as it still ranks,

fifth among the causes of death. By the end of the eighteenth and the beginning of the nineteenth century the disease began to be distinctly separated from allied affections. Thus the first period in the history of whooping cough was distinguished by a more perfect conception of the nature of the disease, a more accurate description of its symptoms, as well as by considerable strife as to the age of the disease, its causes, and, according to Wunderlich, the recommendation of infinite drugs in its relief. The lapse of nearly a century has not entirely cleared up these obscurities as to nature and cause, nor relieved the practice of medicine of the odium of polypharmacy in treatment.

Etiology.—Broussais looked upon whooping cough as a variety of bronchitis; Beau regarded it as a laryngitis; Copeland, Webster, treated it as a neurosis; Friedleben considered it an effect of pressure of swollen tracheal or bronchial glands upon the vagus nerve. Canstatt and Lebert first proclaimed the view that whooping cough was a zymotic affection, with localization in the respiratory organs. Baginsky calls attention to the fact that physiological experiment demonstrates the superior laryngeal nerve as the nerve of cough, and the regions, the irritation of which discharge the most intense cough, as the posterior laryngeal wall close under the vocal cords and the bifurcation of the bronchi. Hence, whooping cough could be regarded as an inflammation of the larynx and trachea.

The view that whooping cough depends upon catarrh of the larynx and bronchi found support in the investigations of Marcus, Loeschner, and Oppolzer. Herff more especially had the almost incredible fortitude to study the condition in himself. Watching his opportunities to investigate the larynx even during an attack, he found marked hyperæmia of the interarytenoid folds, the under surface of the epiglottis, with mucous deposits especially on the posterior laryngeal wall. He maintained that removal of these deposits jugulated the attack. Rehn in his studies found the posterior wall of the larynx perfectly sound, the anterior wall showing the most change; while Rossbach found all parts of the larynx and trachea without any pathological alteration. So that whooping cough has really no morbid anatomy except in its complications.

The disease, considered first as an affection of the stomach, next as a catarrh, then as a neurosis, has finally taken its definite place among the acute infections. Gerhardt goes so far as to remove it altogether from the category of pulmonary diseases and give it a place between meningitis and cholera. Strümpell discusses it along with maladies of the bronchi. Fleischer more properly puts it between croup and mumps. Whooping cough is certainly an infectious disease, because it is contagious and prevails as an endemic and epidemic—because also of the absolute immunity which one attack confers. Rare as are second attacks of scarlet fever, measles, or small-pox, still more rare are second attacks of whooping cough. With the other infections it attacks preferably the age of childhood. Facts which have been taken to militate against the views of its infectious nature are absence of fever and indefiniteness of duration. Facts which refute the idea that pertussis is a neurosis are, first, origin

and dissemination by contagion; second, appearance as an epidemic; third, immunity conferred by single attack. Neuroses belong to individuals and not to numbers. They show no relation to others and have a constant tendency to recur.

Proof of contagion is furnished by the attack of wet-nurses and nurses generally, instances of which are noticed in every epidemic. The period of preference as regards age is from six months to six years. Sucklings, because of natural immunity, are rarely attacked. Szabó declares that nearly half of all cases occur between birth and the age of two years, and nearly three-fourths of all cases between birth and the age of four years. Of 117 cases, Baginsky found but 6 over the age of four years. Susceptibility diminishes at six and is nearly annulled at ten years, yet cases are on record where the disease has occurred in infancy and advanced life. Barthez and Rilliet observed a case of a newborn child whose mother had been attacked four weeks before its birth. The disease set in on the day following birth. Bouchut recorded a case of a child infected on the second day, with a well-developed whoop upon the eighth day. Berger reported a case in a woman aged fifty-seven, and Heberden in a woman aged seventy and a man aged eighty. In the exceptional cases in which the disease occurs in advanced life it runs a mild or abortive course. From some inexplicable reason the female sex suffers most, in the proportion, according to nearly all authors, of 5 to 4. The disease is not only most frequent, but also more severe, in girls. Measles, pregnancy, and the puerperium predispose to pertussis. The contagion is conveyed directly.

The contagious principle exists probably in the sputum, hardly, but possibly, in expired air, which contains no sputum. It is nevertheless a contagium halituosum. The great botanist, Linnæus, nearly two centuries ago expressed the belief that whooping cough was due to a contagium animatum which he thought would be found in the eggs of insects. The principle is thoroughly accepted in our day. The cause is believed to be a micro-organism, varieties of which have been recognized and described by Hallier, Letzerich, and Bürger, with no other proof of pathogenesis than presence in the sputum. Afanassieff succeeded in isolating from the sputum of a whooping-cough patient a short, thick bacillus which he cultivated upon beef-peptone and agar. The bacillus differs in important particulars from forms hitherto described, and gives rise, when introduced into the trachea or lungs of dogs and rabbits, to symptoms simulating whooping cough and to lobular pneumonia. After the death of the animals these same micro-organisms were discovered in the mucous membrane of the trachea and bronchi, and also in the nose. As it was also found in the lungs and bronchi of children who had died of the disease, the author considers it the true cause of whooping cough, and names it the "bacillus tussis convulsivæ." These investigations were subsequently confirmed by Schwenker and Wenat. Ssemtchenko after considerable experimentation reached the following conclusions: First, the bacillus of Afanassieff is specific. It may be found in the sputum as early as the fourth day of the

disease. It multiplies in the body, and as it increases the disease diminishes in severity. It disappears with the resolution of the disease or when the paroxysms are reduced to two to four daily. In the presence of complications, especially catarrhal pneumonia, it increases in the sputum. Thus the bacillus is of value not only in etiology and diagnosis, but also in prognosis. These conclusions have, however, not yet met with universal acceptance, as the observations have not been sufficiently verified. Renewed interest attaches to this bacillus of Afanassieff with the discovery by Griffiths of a ptomaïne or toxine in the urine of whooping-cough patients. Griffiths claims to have established the fact experimentally that an absolutely identical toxine is developed by this bacillus. The toxine is not found in the urine of any normal individual nor in that of one suffering from any disease other than pertussis.

There is, therefore, scarcely room for doubt that pertussis is a mycosis whose toxines have a special action upon that part of the nervous system which presides over cough—to wit, the centres of the superior laryngeal and vagus nerves. Thus in our day the mycotic has displaced the neurotic theory, and the views of Canstatt and Lebert are substantially re-established. Be this as it may, there is no doubt as to the exquisite hyperæsthesia of the larynx in these cases. Any active movement of the larynx as in coughing or crying, the ingestion of food and drink, any irritation as by exposure to cold air as in a draft or a cloud of dust, or to contaminated air as in a close, hot room or crowded apartment, any external pressure or irritation as by inhalation of tobacco smoke or the drinking of any form of alcohol, may produce an explosion of the disease.

The contagious principle is not often disseminated without direct exposure to the disease. Thus very slight isolation secures exemption from attack. The bacillus has no great tenacity of life. Cases in which the disease has been conveyed after weeks or months by clothing, curtains, or other fomites, so common in measles, and more especially in scarlet fever, are very rare in whooping cough. The disease is spread by direct contact in families, and more especially in kindergärten and schools, to assume endemic proportions and to cease only when the material is exhausted.

Whooping cough occurs with special frequency, as stated, during convalescence from measles. The disease shows itself also in close relation to tuberculosis. It has long been noticed that tuberculosis often follows close upon the heels of whooping cough. It is impossible to say in a given case whether the whooping cough made the soil fertile or merely aroused the latent disease.

Symptomatology.—The disease begins with the signs of an ordinary catarrh of the exposed mucous membranes. There are sometimes conjunctivitis with photophobia, and such catarrh of the nose as to lead to the suspicion of the development of measles. Very shortly, if not simultaneously, there is bronchial catarrh, which may distinguish itself in no way from an ordinary cold. The nature of the disease may, however, be anticipated, especially in the presence or vicinity of other cases, on account of its severity and obstinacy, especially on account of the undue prostration in association with it. Some-

times, even at this early period, there is a peculiar ring or intonation in the cough which excites suspicion. Often, again, the paroxysmal nature of the outbreak thus early defines the disease. The statement of the mother or attendant that the child coughs worse at night is not in accord with an ordinary catarrh. A simple catarrh of the larynx or bronchial tubes may distinguish itself by restlessness and exaggerated cough in the early part of the night; but it usually becomes quieter, and the child sleeps more or less continuously after midnight. Whooping cough intrudes itself at any hour of the night, and rouses the child usually to a sitting posture, with a more or less violent paroxysm. The statement also that the cough is attended with flushing of the face and vomiting lends confirmation to suspicions. If, on inspection, the face, especially the lower eyelids, be swollen and dusky, the disease is probably whooping cough. Throughout the whole period of the catarrhal stage, which lasts from ten to twelve days, there is commonly more or less fever. It is most marked, as a rule, in the evening, and is observed only in those cases where the temperature is taken at bed-time. Fever is often absent throughout the whole course of the disease.

Whooping cough occurs, as stated, in paroxysms or explosions. It would appear as if the nerve-centres suddenly discharged themselves of accumulated force or irritation, as in a case of epilepsy. Close observation of a case gives rise to the impression that the poison accumulates gradually up to a certain point, when it may be no longer stored, and is discharged with the explosion that characterizes a paroxysm of the disease.

Whooping cough is usually divided into three stages—the stages of catarrh, of spasm, and of resolution. The first stage lasts about one week. Sometimes this catarrhal stage is very short, and the spasmodic element manifests itself at the end of the second or third day. A pseudo-croup may precipitate an attack at once, so that the disease may supervene on the day following the night of its occurrence. A premonitory catarrh of five or six weeks' duration is usually a preceding complication, and not a distinct prodrome or stage of whooping cough.

The neurotic element now assumes prominence. The cough becomes more frequent, severe, and harassing. The intervals between the paroxysms are, however, more distinct. Very soon the cough assumes the convulsive character mentioned, and sooner or later occurs the typical staccato cough, with the long-drawn, audible inspiration. The second stage has now set in.

In these attacks the seizure is sudden. Sometimes, though not as a rule, there is a kind of premonition or aura which previous experience has taught the child to recognize. It is usually a sense of impending distress or danger, which leads the child to leave its play and run to its parents or grasp a chair for support. A water-closet, a slop-jar, or a cuspidor is a frequent goal. The aura may be in the form of dyspnœa, præcordial distress, nausea, sometimes actual vomiting. Thereupon ensues the series of expiratory coughs which distinguish the disease. The first inspiration is easily drawn. Expiration is substituted by the attacks of cough. A second inspiration is caught

with difficulty, and is often interrupted by a violent spasm of the glottis and diaphragm. The child tries to force the spasmodic contraction. The face assumes an expression of indescribable anxiety. The breath is lost. The face flushes or becomes livid. The eyes protrude. Saliva flows from the mouth. The look is wild, bewildered. There is for a few moments the appearance of extreme danger. At last the expiratory muscles get the upper hand in the form of a series of breath-taking coughs, with the final long-drawn, audible inspiration whereby the stomach is caught between the diaphragm and the abdominal muscles and its contents expressed in the act of vomiting. The discharge of the contents of the stomach and a quantity of glassy, glutinous mucus from the throat closes the attack. This scene may, however, be repeated once or twice before the last of the spasm yields. Inspiration then becomes quicter; and the child, pale, covered with sweat, exhausted, sometimes almost in a collapse, is released until the next attack. Meanwhile it recovers itself entirely, resumes its play, unmindful of the disease, until it is suddenly seized again.

Paroxysms occur in every grade of severity. They are sometimes so mild as to make the diagnosis difficult, in other cases so severe as to lead to rupture of vessels. Hæmorrhage may occur from the nose and mouth. Subconjunctival hæmorrhage is not uncommon. There are hæmorrhages in this region which do not disappear with the subsidence of the cough. The membrana tympani ruptures at times and free blood appears at the external meatus. Ectatic vessels burst in the skin in the face, in the cheeks, and show, visible even at a distance, subcutaneous extravasated blood. Hæmorrhage from the stomach or intestines or from the kidneys or bladder is much more rare. Hæmorrhage in the brain, which sometimes occurs, is fortunately very much more rare. Hernia is not uncommon. Convulsions are possible. The duration of an attack is usually from half a minute to two minutes, though it seems to anxious, sympathetic relatives four or five times as long.

Paroxysms occur also in every grade of frequency. Attacks may be limited in some cases to ten to twelve daily throughout the whole course of the disease, or they may occur as frequently as twenty, forty, or sixty times in the course of twenty-four hours. As the severity stands in quite close relation with the frequency of attacks, it is important that the number be counted, as by strokes on a piece of paper or a slate, according to the suggestion of Trousseau. Diminution in the number of attacks is the first sign of approaching relief. During the interval the patient is apparently in a state of perfect health. Even when attacks occur frequently during the night, arousing the child to a sitting posture and a state of extreme anxiety with the struggle for air, it falls asleep as soon as the attack is over, to become immediately unconscious of suffering and to awaken in the morning without a sign of fatigue. Burman attributes the frequency of attack at night to the diminished vigilance of the respiratory centres, retarded and more superficial respiration, and greater accumulation of carbonic acid gas, which may at any time explode the disease.

About the fourth decade of the present century attention began to be direct-

ed to the more or less constant appearance of an ulcer on the frænum linguæ. The lesion was observed so constantly as to have given rise to the belief that it caused the disease. So soon as it was seen that the ulcer was absent in attacks which occurred in very early life, and showed itself only after dentition, the occurrence of the lesion was explained by friction of the protruded tongue against the inferior incisors. More extensive destruction of tissue has been observed also on the base of the tongue by the side of the frænum, and a similar break has been seen even upon the dorsum or upper surface of the tongue, where it has been protruded against sharp upper incisors. The lesion even at the frænum is by no means universally present. It is absent altogether where the attacks are very light, or where the frænum is short or the tongue may not be protruded, or where the incisors are dull. It has been seen also independently of whooping cough in cases of cough from ordinary catarrh where the lower teeth have been unusually incisive.

The spasmodic stage lasts, as a rule, during two to four weeks, when the interval between the paroxysms becomes gradually longer and the explosions themselves less severe. A series of milder attacks may be followed by occasional paroxysms of former severity, and the disease may be protracted in exceptional cases over a period of several months. Thus the period of resolution may last from two weeks to two months. Cases which are said to last during six to twelve months and more are usually complicated by chronic bronchitis, bronchiectasis, emphysema, and more especially tuberculosis. Any paroxysmal cough, sufficiently severe, may be followed by an occasional audible inspiration. These are the cases in which the patient is said to have never recovered from the disease. Such an explosion in the observation of the author was attributed by the mother to an attack of whooping cough four years before. It was apparently a bronchiectasis.

Whooping cough is liable to many complications, especially on the part of the respiratory organs. Bronchitis belongs to the disease, and usually drowns all other sounds in the lungs with its râles. Any disease attended with bronchitis is liable also to broncho-pneumonia, and broncho-pneumonia is the most frequent of the serious complications of whooping cough. The spasmodic closure of the glottis and the powerful efforts of the expiratory muscles sometimes develop œdema of the glottis, more frequently emphysema of the lungs. The wonder is that emphysema is not more universal. The occurrence of it is in fact an exception. It is usually slight, marginal or peripheral, and is marked by dilatation only of the air-cells, whose walls are so resilient as to recover themselves entirely with the relief of the strain on cessation of the disease. Sometimes, however, especially in cases of failing nutrition, tuberculosis, syphilis, and rickets, the dividing walls are broken and air-cells are ruptured. Still more rarely air may escape into the pleural sac, to constitute a pneumothorax or break the lung at its hilus, reach the mediastinum, or escape into the subcutaneous connective tissue and inflate—literally blow up—the upper half of the body. There is no better proof of the strength of the heart than the fact that it escapes damage under the spasm and stasis of whoop-

ing cough. In very protracted or extremely severe cases the heart-muscle may be finally weakened and show spots and regions of degeneration. As a rule, however, all the circulatory disturbance of whooping cough is limited to stasis and ectatic dilatation of the vessels, so that in the great majority of cases nothing is seen except puffiness of the eyelids and ectatic vessels about the face.

Complications on the part of the nervous system are very rare. At the height of the attack there are experienced extreme anxiety, a sense of suffocation, a vertiginous bewilderment approaching loss of consciousness, which disappears entirely with the recovery of the breath. The momentary apnœa may be prolonged to the point of danger, and very young children may actually succumb to suffocation. Vomiting, which is usually hailed with pleasure as indicating the end of the attack, may be excessive. It may continue into the interval. It may even produce collapse, or in more protracted form lead to marasmus. More frequently a more or less decided convulsion ensues, and the case may be marked by a series of convulsions, any one of which may prove fatal. Sometimes cerebral symptoms continue during the interval, and a case may bear the aspect of a meningitis. Stupor, coma, and hemiplegia would indicate the occurrence of cerebral hæmorrhage.

Trórtzky reported three cases accompanied by mental disturbance, loss of speech, and monoplegias. In one case a child aged two was seized immediately after a paroxysm with strabismus, dilatation of the pupils, blindness, spasm of the flexors of the arm, delirium, and Cheyne-Stokes respiration. In a second case, in a boy aged three and a half years, there were great headache and difficulty in thinking and speaking during the five weeks' duration of the disease. The case was complicated with pneumonia.

The relation of pertussis to measles has been already remarked, and the coincidence of these diseases intensely aggravates the prognosis of either one. Pertussis stands also in close relation to tuberculosis. It is said to fertilize the soil for the growth of the bacillus tuberculosis. The truth is probably that it awakens into activity latent dépôts. Enlarged tracheo-bronchial glands have been so often remarked in connection with pertussis as to have been at one time considered the cause of the disease (Gueneau du Mussy). Latent tuberculosis is the more modern explanation of these adenopathies.

The **diagnosis** of whooping cough in the convulsive stage is an easy matter. The series of rapid, sudden, explosive, breath-taking coughs, attended by the evidence of venous stasis, cyanosis—whence the old name, blue cough—which ceases only when a quantity of mucus, under the combined efforts of cough, retching, and vomiting, is expelled; the prolonged expiratory efforts, followed by a long-drawn, audible inspiration, which has been not inaptly likened to the bray of an ass; and the gradual cessation of the disease,—sufficiently characterize it.

In the first stage whooping cough is not so easily separated from other forms of catarrhal affections. The age of the patient may throw some light upon the case. The presence or absence of the disease in the history, more

especially the existence of other cases in the family or community; then the obstinacy of the cough, the longer duration, the fewer physical signs to account for it, the more spasmodic character of it, with intervals of more complete exemption,—excite suspicion or confirm the evidence of the existence of disease.

In the last stage there will generally have been a well-marked history of previous whoop in the cough, which may, indeed, be still occasionally heard. Here, too, there is a more marked interval between individual attacks than is common in the ordinary bronchitis; a slight nervous element still prevails. The cough will have lasted unusually long, six to twelve weeks; other cases in other stages of the disease are in the vicinity, etc.

The **prognosis** is for the most part entirely favorable. Notwithstanding the threatened suffocation and tremendous strain upon the heart, recovery is the rule, and that without a trace of lesion. Complications and bad surroundings may, however, intensely aggravate the natural benign prognosis. The prognosis is determined to a considerable extent by the age and sex. The disease is, as stated, from some inexplicable cause, not only more frequent, but more severe, in the female sex. The disease becomes less and less grave with advancing life. Maier declares that 97 per cent. of all the fatal cases occur under the age of five, 58 per cent. in the first year. Biermer made a grand average of the established mortality-rate, based upon the statistics of many authors, at 7.6 per cent.—a figure that certainly entitles the disease to respect.

It is an error to consider whooping cough as a trivial malady. There occurred in England in one year, of 500,341 deaths, 10,318 deaths from whooping cough. In New York in one decade, wherein 4062 deaths occurred from typhoid fever, there were 4094 deaths from whooping cough. Hagenbach says that whooping cough had more victims in Basle in fifty years than any disease except typhoid fever and diphtheria. The general mortality is estimated, as stated, at 3 to 7 per cent. It has reached as high as 48 per cent. in the second year of life.

The most frequent causes of death are (1) suffocation from spasm of the glottis; (2) broncho-pneumonia; (3) hæmorrhage; (4) marasmus. A simple peripheral emphysema disappears, as stated, without trace. Interstitial, mediastinal, and general emphysema, pneumothorax, make the prognosis grave.

The prognosis depends, aside from the condition of the patient himself, upon the severity and frequency of the attacks. A single explosion may last from fifteen seconds to an entire minute, and a series of explosions which constitute an individual attack may last from ten to fifteen minutes. The prognosis is grave where the attacks reach fifty in the course of twenty-four hours; at sixty it assumes special gravity. Individual attacks may do damage also by their intensity; thus hæmorrhage may be copious from mucous surfaces. Blindness occasionally results, probably from œdema of the brain. It is almost always temporary, and disappears with the subsidence of œdema. Then subarachnoid hæmorrhage, unusual as it is, is sometimes fatal. Such excessive

vomiting occurs in certain cases as may not be stilled with the cessation of the attack, so that inanition may result. Psychopathies from the profound mental disturbance, fortunately usually temporary, are occasionally reported. Absolute exophthalmos has been produced by excessive retro-bulbar hæmorrhage. Rupture of the membrana tympani, with subsequent otitis media, has resulted in deafness and, occurring in very early life, in deaf-mutism. The frequency with which the disease is attended with, preceded by, or followed by tuberculosis has been remarked already. Thus it will be seen that whooping cough is by no means a trivial affection.

Prophylaxis.—As the disease has, at least at times, such gravity, prophylaxis assumes importance. The only prophylaxis worthy of the name is isolation. Isolation to be effective must be complete. The patient must be separated not only from children, but from adults who come in contact with unaffected members of the family. As this isolation in a disease which is usually considered so mild is practically impossible, attention should be directed rather to the protection of delicate members of the family; they should be isolated rather than the patient. It is advisable that tuberculous, rachitic, syphilitic, or otherwise diseased or debilitated children should be removed from the house as early as possible. Whooping cough is contagious in all stages of the disease. So long as there is cough, matter is expectorated, to be dried and disseminated, and thus to convey the disease. In the removal of children from the house, warning should be entered at the new place of residence, that the disease may not be developed in new centres.

The most essential element in prophylaxis at all times is the destruction of the sputum. Though the individual is attacked with the suddeness of an explosion, mucus, at least in quantities, is not expelled until the attack has spent itself, so that there is, for the most part, time for the collection of sputum in water. As in tuberculosis, the handkerchief should never be used for the reception of sputum. Perfect prophylaxis implies also the use of separate beds, the separate washing of bed-linen or the subjection of it to steam or dry heat, the use of separate utensils for food, the use of separate clothing, etc.

Treatment.—Until the specific nature of the disease shall have been determined there can be no question of any specific treatment, and remedies may be addressed only to its symptoms. The symptom which assumes prominence, and upon which nearly all the complications of the disease depend, is the cough, and the nature of the remedy which is used against the cough will depend upon the view which the practitioner may take of the nature of the disease; that is, whether it be catarrhal, neurotic, or mycotic. The truth is, the treatment of whooping cough remains still in the stage of empiricism, and as nearly every remedy in the materia medica has been tried to relieve the cough, so appeal is made to every new remedy.

The older writers used the anodynes early. Opium in some form or other was the shield which was soon interposed. In modern times the active principle of opium, morphine, was and is still extensively employed:

℞. Morphinæ sulphatis, gr. ss–j;
Aquæ amygdalæ amar., f℥ss;
Aquæ, f℥iss.—M.
Sig. A teaspoonful every two to six hours.

With the morphine was often combined 5- to 10-grain doses of the bromide of sodium or potassium, or there may be added to the prescription the hydrochlorate of apomorphine, ½ grain to 1½ grains, or for the bitter-almond water or cherry-laurel water may be substituted ½ an ounce of either glycerin or syrup, simple or of orange-peel, raspberry, etc. The remedies commonly employed in the treatment of bronchitis are also frequently resorted to. The syrup, simple or compound, of ipecac, ¼ to ½ teaspoonful; the wine of ipecac in half these doses; minute doses of tartar emetic, $\frac{1}{64}$ to $\frac{1}{32}$ of a grain; belladonna, 1 drop of the tincture for each year of life; or atropine, 1 grain to 1 ounce of water, given in doses of from 1 to 2 drops two or three times a day. The iodide of potassium is a remedy of value. It may be given as follows:

℞. Potass. iodi, ℥ss;
Aquæ menth. piperit., f℥ss.—M.
Sig. Two to five drops in a dessertspoonful of milk three or four times a day.

The iodides are more used in cases in which the chest is full of râles; the ipecac preparations especially in the presence of burning irritation in the throat and chest; belladonna, the bromides, and morphine being addressed more especially to the spasmodic element.

Camphor, valerian, asafœtida, and musk have their advocates in the treatment of whooping cough. Chloral had at one time high laudation in doses of 3 to 10 grains. Chloroform, ether, the bromide and iodide of ethyl, and amyl nitrite (2 to 5 drops) were inhaled with the hope of curtailing the attack; creasote, the salicylates, and carbolic acid were remedies administered internally and by inhalation for the destruction of the assumed mycosis. Various antipyretics, more especially antipyrine, in doses of from 2 to 5 grains every two to four hours, do certainly prolong the intervals and mitigate the severity of the attack. These remedies were recommended indeed as specifics a few years ago, with the later fate of all the specifics, in the treatment of whooping cough. Saturation with bromides, gr. x–xv four times a day, is a plan now much in vogue.

The mere mention of the names of remedies recommended from time to time in the treatment of whooping cough would consume the space allotted to the discussion of the whole subject. One remedy, however, deserves mention, if only because it is the last used. This is bromoform, which was recommended first by Stepp of Nüremburg. Löwenthal used it in Senator's polyclinic in 100 cases, claiming that it made the attacks milder in the course of a few days. Bromoform is given in drop doses, 2 to 5, three or four times a day. Children one year of age receive three times daily 2 to 4 drops; children from

two to four years of age receive 3 to 4 drops three to four times daily; children from four to eight should receive three or four times daily 4 to 5 drops, according to the number and frequency of the attacks. The remedy must be protected from the light, hence in dark bottles with good stoppers. It is usually given dropped in water, when care must be taken that the pearly drops floating about in the water are swallowed. If the use of the drug be stopped too soon, relapses occur. Bad effects have been observed, but never from these small doses. One child which received a larger dose than had been prescribed fell into narcosis, but was readily revived. Fischer of New York reports 51 cases, claiming almost specific properties. The duration of the treatment was from ten to thirty days, and cure occurred in 75 per cent. of the cases in from two to three weeks, if there were no complications. Neumann of Berlin is more temperate in his statements. He tried the remedy in 25 cases, and believed that it exerted a favorable influence upon the individual attacks, but had no real effect upon the course or duration of the disease. He was never able to cut an attack short even by early administration of the drug, though he never saw any ill effects. These conclusions represent the results which are generally admitted, so that it may be said that bromoform is the most valuable of the late contributions to the therapy of this disease.

Among the latest remedies recommended the following may be cited: Carbolic acid in aqueous solution, 1 : 120, of which ½ an ounce three or four times a day is advocated by Oltramare; hyoscine hydriodate, by Edelfsen; turpentine, revived by Ringk; pilocarpine to abort the disease, by Albrecht; chloride of gold and sodium, by Magruder; cocaine, by Krimke; peroxide of hydrogen, by Richardson; cyanide of mercury, by Drzewiecki; resorcin, by Concetti; oubäin, by Gemmel; thyme, by Johnson. Among remedies to be inhaled, turpentine, thymol, illuminating gas (carburetted hydrogen), carbolic acid, cocaine, sulphuretted hydrogen, tar, benzole. Ledolier recommends chloral by rectal injection; Goldsmith sprays the nose with mercuric chloride or salicylates; and Rossbach applies the constant current of electricity.

Vomiting may be usually relieved by chloral, grains 2 to 5, with peppermint-water, a dessertspoonful to a tablespoonful, by the mouth or if necessary by the rectum.

Local applications addressed to the mycotic nature of the disease cannot reach it, for the reason, probably, that the mycosis is in the blood, and the symptoms are due to toxines. Applications and inhalations of carbolic acid, chloroform, benzine, turpentine, quinine, resorcin, however highly lauded by individual observers, have all fallen into disuse. May it be mentioned that Naegeli reports that in two children he has succeeded more than five hundred times in apparently arresting the spasms of pertussis by pulling the lower jaw downward and forward?

Cocaine has been tried, but met with no better success. The influence of the drug is too temporary and its cumulative effects too depressing, if not dangerous, to warrant its continued use, so that at the present time the treat-

ment of whooping cough, awaiting the discovery of some specific antitoxine, resolves itself into the extremely cautious use of morphine as a shield against the damage of the disease. Mild cases are let alone, on the principle "*primum non nocere.*" Let it be said, for the honor of medicine in this dearth of therapy, that change of climate does often really "act like magic."

SEPTICÆMIA AND PYÆMIA.

By WILLIAM PEPPER.

From the earliest times the occurrence of febrile disturbance after wounds and in connection with internal suppuration has been noted. The terms "septicæmia" and "pyæmia" were for a long time indiscriminately used to designate the conditions referred to, and it is only since the development of the science of bacteriology that the ultimate etiological differences between the two conditions have been satisfactorily discovered. While they come under the notice of the surgeon more frequently than that of the physician, there are forms of both conditions that are seen by the latter and that arise independently of the occurrence of external wounds.

Septicæmia.

Definition.—A condition caused by the absorption of animal poisons developed in the body, as a rule due to bacterial growth, characterized clinically by febrile reaction and various accompanying symptoms; pathologically, by the absence of purulent foci in the various organs of the body.

After the receipt of any wound there may arise a reactionary fever, whether the solution of continuity be exposed to the air or enclosed by the uninjured parts surrounding the lesion. After simple fractures there is apt to be a rise of temperature, of short duration, and acquiring a variable, but not frequently a considerable, height. This reactionary fever was at one time supposed to be due to reflex causes, but it is more reasonable to suppose that it is produced by the absorption into the blood of some of the materials surrounding the lesion. This is well seen in the febrile condition that often results from the presence of an extravasation of blood into the tissues. In this form of septicæmia the presence of bacteria in the primary focus is not necessary. Usually without preceding chill there is a rapid rise of temperature to 102°, or even 104° F., with corresponding rapidity of the pulse, but without marked constitutional disturbance. This fever continues for a short but variable length of time, from one to three or four days, disappearing spontaneously and leaving no sequelæ. A second form may result from the absorption from wounds of the products of growth of the micro-organisms of putrefaction. This is exemplified in the rise of temperature with constitutional disturbance seen after operation, where there is in the wound either a disorganized blood-clot or a portion of necrotic tissue. The symptoms in this form appear rapidly, and differ from those observed in the form first described merely in their greater intensity and more prolonged duration. Rapid disappearance of all unfavorable symptoms follows the removal of the cause.

In a third and more severe form the symptoms are produced by the absorption of ptomaïnes from a local lesion, and by the ptomaïnes in the circulating blood likewise produced by absorbed micro-organisms. The micro-organisms concerned in this form are usually the pyogenic micrococci. The condition is well seen in the results of such poisoned wounds as are frequently produced in the dissecting-room. After a variable period, twenty-four hours to three or four days, there appears a feeling of malaise, with slight chilly sensations or even a pronounced rigor. Soon the bodily temperature progressively rises, and shows daily remissions or intermissions. With this rise of temperature there are headache of variable intensity, loss of appetite, a moderate amount of thirst, in some cases diarrhœa. The pulse is rapid, and at first full and bounding, but later becomes soft and compressible. The tongue is lightly coated in the beginning, but as the bodily temperature rises becomes thickly coated and dry. Locally, there may be intense redness and swelling of the part affected if on the cutaneous surface, and lymphangitis and lymphadenitis may be found in the lymphatic structures that drain the part. If the cause be not removed or if the poisoning be very intense, the patient sinks into a typhoid condition, with marked irregular elevation of temperature, rapid, feeble pulse, delirium of variable intensity, but usually of muttering character, and marked prostration of all vital power. The tongue now is apt to become dry and glazed, or may be covered, as are the teeth, with brownish sordes, with which may be mingled bloody crusts from the numerous fissures that are present in the organ. If the condition persist, the first sound of the heart becomes weak, bed-sores form, and the patient dies exhausted. In some cases there appear before death petechial spots on the surface or hæmorrhage may occur from various mucous surfaces.

Upon examination after death there are no characteristic lesions to be discovered. The blood is usually dark in color. Upon the serous surfaces points of extravasated blood may be seen, or there may be found hæmorrhagic effusion in the cavities enclosed by them. The spleen is usually enlarged and softened. The liver may show evidences of fatty degeneration or may be merely fuller of blood than is normal. The lungs are apt to be congested, chiefly in the posterior portions. There may be areas of atelectasis or of broncho-pneumonia. The heart is softened, and its cavities contain either adherent ante-mortem coagula or grumous, friable post-mortem clots. There are no hæmorrhagic infarcts or metastatic deposits of pus. Upon microscopic examination of various organs there may be found islets of coagulation-necrosis.

The **diagnosis** is, as a rule, easy, the only difficulty being the discovery of the source of blood-contamination. By careful examination into the history of the case this can usually be discovered even in the cases that do not result from some recent injury or operation. One point should, however, be borne in mind: the frequent dependence of this condition upon the attempt to produce criminal abortion or upon the presence in the uterus of placental remains after an unrecognized miscarriage.

The prognosis is in all cases to be guarded. Death may occur within the first twenty-four hours if the amount of poison absorbed be very large.

In regard to treatment but little can be said. Removal of the cause is of the greatest importance. In addition to the adoption of appropriate surgical measures the patient's strength must be supported by appropriate diet and the judicious use of stimulants. In the lighter forms quinine may be of value in small tonic doses; in the more severe forms its value is very slight. The diet should consist of liquid, easily digestible and nourishing articles, such as milk, raw or lightly boiled eggs, beef- or clam-juice, and liquid peptonoids. Alcoholic stimulants in some form are required in all severe cases. Careful attention must be directed to the condition of the skin, and the urinary bladder must be carefully protected from over-distension. Should danger arise from hyperpyrexia, suitable hydrotherapeutic measures should be adopted.

Pyæmia.

Definition.—A condition arising from the diffusion of the micro-organisms of suppuration throughout the body, characterized clinically by frequently recurring chills, remittent or intermittent fever, profuse sweatings, and various symptoms depending upon the involvement of different organs; pathologically, by the presence in various tissues of multiple metastatic purulent foci, produced by the transference of the pyogenic micro-organisms from a primary focus of suppuration.

As will be seen by the definition given, this condition is dependent upon the presence in the various organs of single or multiple abscesses that have been produced by the transportation of pyogenic bacteria from some primary focus, thereby differing from septicæmia, wherein no such metastasis is present. Among the more frequent sites for these primary abscesses, when not traumatic, may be mentioned the subcutaneous cellular tissue; the pelvic cellular tissue and organs; the subperitoneal connective tissue; the marrow of the long bones; the parts surrounding the middle-ear cavity; and the joints. It frequently happens that wide dissemination of purulent emboli occurs from an ulcerating lesion of the cardiac valves. This form of endocarditis is usually itself secondary to some other lesion, the valves merely offering a good breeding-place from which the bacterial masses may be swept off by the blood-stream and scattered throughout the body. "Idiopathic pyæmia" is the name applied to those examples where no primary purulent focus can be demonstrated.

The essential cause of the condition is one of the forms of pyogenic micrococci. The streptococcus pyogenes is the most frequent form found, but the staphylococcus pyogenes aureus or albus is present in many instances. These micro-organisms, either by their own activity or by means of the materials they produce, cause coagulation-necrosis of the surrounding tissue-cells; by their continued action this area of coagulation-necrosis extends; inflammation of the veins of the part follows, with a similar process in the wall of the vessel; the endothelium of the vein so affected is loosened from its deep attachment, and with its contained micrococci is swept off by the blood-stream.

Arriving at some portion of the circulatory system where the calibre of the vessel diminishes to such an extent as to preclude the passage of the embolus, stasis occurs, and, if the soil be suitable, the transported micrococci repeat the process of pus-formation in their new quarters. In this way are formed numerous abscesses in various parts of an organ or in various and frequently widely-separated regions of the body. If the original focus were in the superficial portions of the body or in the long bones, the secondary abscess will be found in the lungs, or if they pass through these organs without lodgment the heart and kidneys will offer a favorable site for their development. If the primary focus be in the area drained by the portal system, purulent pylephlebitis and secondary abscesses in the liver will result. When malignant endocarditis has been the starting-point of the emboli, the secondary foci may be found in the spleen, kidneys, brain, skin, or intestines.

The **symptoms** of pyæmia vary greatly in different cases, depending upon the organ or organs that are the seat of the secondary abscesses. There are, however, certain symptoms that are present in all forms, and that characterize the condition. The onset of pyæmia is usually announced by the occurrence of a rapid rise in the bodily temperature. With this rise of temperature, or following shortly thereafter, there is a chill that at times merely amounts to a sensation of coldness, at times to a severe rigor. The temperature may shortly sink to near the normal, but soon again rises to a point higher than that previously attained. The fall of temperature may not occur until the following morning, the second elevated point being usually observed upon the evening of the day after the onset. From this time the temperature assumes a peculiar type, with high elevations toward evening, and a fall of oftentimes three, four, or more degrees toward the early morning hours. The rigors are repeated at varying intervals, but they may not be a marked feature of the case. With this peculiar temperature range and the occurrence of rigors there is found to be a marked tendency to profuse sweating. The sweating may be almost continuous, or it may occur in paroxysms that are apparently causeless, but that are more apt to occur toward night-time. The patient rapidly loses strength, and emaciation progresses with constantly increasing celerity. With the symptoms enumerated there are loss of appetite, thirst, a peculiar sweetish, nauseating odor, and usually the signs of involvement of one or more of the internal viscera. The patient sinks into a condition of profound prostration; bed-sores form on parts exposed to pressure; and the patient dies from exhaustion or from involvement of some vital part in the suppurative process.

When the liver is the seat of secondary foci of suppuration, a more or less intense yellow discoloration of the conjunctivæ and skin will be developed, with in many cases a varying amount of diarrhœa, and on percussion there is shown to be enlargement of the organ with tenderness over its site. Involvement of the kidneys may give no sign; more frequently, however, there is albuminuria with granular casts, and at times blood. Splenic infarction is shown by pain in the left hypochondriac region, with progressive enlargement of the area of dulness. Metastasis to the lungs usually is productive of but few changes

in the physical signs, save those of the accompanying inflammatory conditions. Intestinal involvement is accompanied by marked diarrhœa, causing errors to be frequently made by its simulating the diarrhœa of typhoid fever. Metastatic infarctions of the derm produce multiple superficial abscesses. Secondary abscesses may also occur in the parotid gland and in the pancreas, giving rise in the latter to deep-seated pain in the epigastric and umbilical regions. Abscess of the brain may give rise to various forms of paralysis, but the lesions are, as a rule, multiple, and hence give rise to no trustworthy localizing symptoms. One other form must be mentioned, wherein the joints are attacked, giving rise to what is known as pyæmic rheumatism.

The **pathology** of the condition has been already indicated. Upon post-mortem examination it may be difficult to determine the primary source of infection. The abscesses that form in the various organs are multiple, and usually do not attain to large dimensions before death occurs. In some cases, however, owing to the fusion of smaller abscesses or owing to the embolus obstructing a large arterial branch, one large abscess may be present. In the secondary deposits there can be found the pyogenic micro-organisms that are accountable for their production.

The **diagnosis** is usually readily made by observing the peculiar irregularly intermittent fever. The two diseases with which this condition is most apt to be confounded are typhoid fever and malaria. A careful review of the history of the case, due attention to the course of the temperature, the appearance of the countenance, the absence of marked sweating and of rigors, with the presence of the typical eruption, the characteristic stools, the tympany, and splenic enlargement, will usually indicate typhoid fever. Malaria can be readily distinguished by the greater regularity of the fever, the periodic occurrence of the rigors and sweating, the completeness of the intermission, the specific action of quinine, and finally the presence of the plasmodium malariæ in a patient suffering from the malarial infection. The diagnosis is not sufficiently accurate until not only the condition of pyæmia is determined, but the lesion that gave birth to it is discovered.

The **prognosis** is in all but the mildest cases extremely grave. Where the disease is well marked, and where surgery cannot be called to our aid to evacuate the secondary dépôts of pus, recovery is rare. Unfavorable signs are those indicating involvement of the deeply-seated organs.

Unfortunately, but little can be done in the way of **treatment**, save where the secondary abscesses are amenable to surgical interference. All our measures must be directed to keeping up the patient's strength. A nutritious diet, moderate doses of quinine, with suitable amounts of alcohol, are our chief mainstay. Digitalis, caffeine, or strychnine may at times be of use. For the sweating, atropine, aromatic sulphuric acid, or agaricin internally, with sponging of the body with alcohol and alum, may be resorted to. Pain is rarely severe, but may require the use of morphine. The chief indication is to support the patient until surgical interference for the evacuation of secondary foci may become possible.

ACUTE MILIARY TUBERCULOSIS.

By W. GILMAN THOMPSON.

Definition.—Acute miliary tuberculosis is a form of tubercular infection characterized by the general dissemination of minute tubercles throughout the various organs of the body, by pyrexia, constitutional symptoms, and a rapidly-fatal ending.

Etiology.—Acute miliary tuberculosis may occur at any period from infancy to sixty years of age, but it is most common between the age of puberty and middle life.

Acute miliary tuberculosis may follow upon a tubercular pleurisy or various bone and joint diseases of tubercular origin with caries and necroses, or tubercular lymph-glands with cheesy degeneration. It not infrequently occurs in connection with a tubercular psoas abscess or with the scrofulous diathesis; hence it is very often a secondary disease. Reich reports a remarkable occurrence in Neuenburg, a town of 1300 people. The midwifery practice of the town was divided between two women. One of these midwives acquired pulmonary tuberculosis. She was in the habit of resuscitating stillborn infants by applying her mouth to theirs and breathing into their lungs. In the course of two years ten of these infants died of miliary tuberculosis, which affected chiefly the meninges, while in the practice of the healthy midwife no such fatality resulted, and the parents of these children were not tubercular.

Similar infection has occurred among Jewish children after the rite of circumcision when the operator, having pulmonary tuberculosis, has applied his lips to the incision. Occasionally operation upon tubercular joints or upon tubercular bones with incomplete removal has been followed by miliary tuberculosis.

None of these various conditions, however, are necessary forerunners of miliary tuberculosis, and the disease occurs sometimes in persons who are in good health—at least in whom no caseous or tubercular foci or tubercle bacilli have been discoverable. The cause of this sudden infection by miliary tuberculosis of the entire body in persons previously in apparent health is undecided. It is not directly due to the entrance of any virus through the lungs, and it does not follow acute pulmonary inflammations. It was long ago suggested by Buhl that it was owing to the sudden liberation of caseous material from a circumscribed focus by ulceration into a blood-vessel or lymphatic vessel. In support of this theory is the fact that Ponfick discovered caseous infiltration of the walls of the thoracic duct in children who had died of miliary tuber-

culosis. A few years later Weigert found similar appearances in the walls of the pulmonary veins. Under similar circumstances it would be easy for a lymphatic gland to adhere to the wall of a vein, ulcerate into it, and pour its products into the circulating blood.

On the contrary, miliary tuberculosis is a very unusual complication of advanced pulmonary phthisis, and, indeed, of many other conditions in which there are large caseous deposits of long standing.

Since the discovery of the bacillus of tuberculosis by Koch in 1881 the theory has been advanced that miliary tuberculosis is not occasioned by caseous or other infectious material present in the circulation, but by the bacilli themselves, which, having escaped into the blood, find lodgment in the different viscera and serve as foci for the development of countless tubercles. In support of this recent theory is the fact that the injection of Koch's tuberculin into tuberculous subjects occasionally excites acute miliary tuberculosis.

Koch's tuberculin is a glycerin extract prepared from a culture medium in which tubercle bacilli have been growing. It is diluted with distilled water at the moment of use, and one milligram, gradually increased to five or more, constitutes the dose, given by hypodermic injection. According to Koch, the glycerin extract contains peptone, albumose, and other undefined proteids and salts: it is a viscid, thin, syrupy fluid with a neutral reaction, faintly aromatic odor, and the color resembles diluted iodine. When diluted with water it is opalescent and greenish.

The action of tuberculin upon tubercular subjects is both local and constitutional. In from four to five hours after the first inoculation it produces febrile symptoms. There is a chill with nausea and vomiting, headache, malaise, aching of the limbs, and a sharp rise of temperature to 103° or 105° F. A few hours later the local symptoms appear. If there be a tubercular joint or skin or gland lesion, there is local swelling, redness, and pain, accompanied by exudation and infiltration of the tuberculous tissue with leucocytes. Similar changes occur at the site of tubercular processes in the lungs and elsewhere. The tuberculin does not kill the tubercle bacilli, but modifies the nutrition of the tissues that surround them, and it has no influence on necrosed bone or old cheesy material. Living bacilli and caseous detritus have been found in the sputum after tuberculin inoculation. In other instances the bacilli become encapsulated, and are thereby rendered innocuous. In still other cases, however, patients have been found to suddenly develop acute miliary tuberculosis. This is due to the local inflammatory process excited by the tuberculin, resulting in a communication between a tubercular lymph-gland, or other focus of tubercular material, and a vein or lymphatic trunk. In this manner the bacilli pass into the circulation, and are borne to all parts of the body to excite the formation of fresh tubercles. This occurrence is not very frequent, but well-marked cases have been described by Virchow and others. The use of tuberculin, although so disappointing in its benefits, has proved of exceptional interest by adding to the knowledge of this mode of exciting acute miliary tuberculosis.

Morbid Anatomy.—After death the body presents the appearance common to acute febrile disease. The blood is dark and fluid, the spleen is softer than normal, and there may be more or less visceral congestion. The muscles are red and rigor mortis is well marked.

For the detailed structure of tubercles the reader is referred to the article upon *Pulmonary Phthisis.* They present no anatomical peculiarities in miliary tuberculosis, excepting in regard to their extension and uniform distribution through many organs and tissues of the body. The tubercles vary in size from $\frac{1}{500}$ to $\frac{1}{250}$ of an inch in diameter. Submiliary tubercles are also found, and the masses may be as large as a split pea. Such masses are composed of aggregations of the miliary nodules. When newly formed they are gray, translucent, and somewhat firm in consistence, so that they can be picked out with the point of a scalpel. They commonly possess the epithelioid and giant-cell structure which is typical of such growths, or they are merely minute foci of tubercular inflammation. The outer wall of the arterioles is often thickened by infiltration with lymphoid cells. Such structure is often found in the vessels of the pia mater.

Sometimes inflammatory products or granulation tissue are mixed with the tubercular growths. The miliary tubercles, when newly formed, are often found to contain tubercle bacilli. Older tubercles, especially caseous ones, contain few or no bacilli.

The disease usually runs such a brief course that few if any of the tubercles have an opportunity to undergo caseous change, as they do in pulmonary phthisis. In more protracted cases they may become caseous and yellow; sometimes the centre only of the tubercle is caseous and friable.

The lungs are filled throughout with minute tubercles, which can for the most part be seen with the naked eye and distinctly felt between the fingers. They may also involve the pleura. The tubercles are grayish and translucent, and the larger yellow cheesy masses and abscess-cavities, which are irregularly disposed in chronic pulmonary tuberculosis, are wanting, unless the latter disease has previously invaded the lungs, which is not usually the case. Tubercles are found in the parenchyma of the lungs, and they invade the walls of the air-cells. The air-cells between the tubercles contain more or less detritus of granular matter, desquamated epithelium, or inflammatory products, fibrin, and pus in small quantities. The tubercles also occur in the walls of the bronchi and pulmonary blood-vessels. They are frequently discrete, but may be very close together or occasionally gathered in masses as large as a pea. The lesions of extensive acute catarrhal bronchitis may accompany the tubercular extension in the lungs. In children's lungs the miliary tubercles often attain a large size.

In the liver and spleen tubercles also occur in large numbers, but they are of microscopic size. The spleen is not much enlarged, but may be slightly so. In the liver the tubercles are found both within the lobules and in the interlobular connective tissue, the latter being often increased in amount.

The endocardium may contain tubercles. The lymph-glands, peritoneum,

and omentum may be filled with them, and peritonitis may be excited. Tubercular granules are also found in the marrow of the bones, such as the vertebræ, sternum and ribs, and in the kidney. In the latter there may be only infiltration of lymphoid cells.

The tubercles invade the pia mater, especially at the base of the brain. They are found upon the membrane over the pons, optic chiasma, etc. This condition is often present in cases which attack the young.

The invasion of the choroid by tubercles was first observed by Manz and Colnheim. They can be distinguished in the eye during life by an expert ophthalmologist, although they are somewhat difficult to demonstrate. They commonly develop near the yellow spot or the disk, or they may occur throughout the choroid. When present in a doubtful case they make the diagnosis absolutely certain. They vary in diameter from 1 to 2.5 mm. The tubercles may occur anywhere in the body, but they are rare in the skin, mucous membrane, or muscles, and seldom occur in the pancreas or salivary glands.

Symptomatology.—The symptoms depend upon the general infection and the extent of the local lesions.

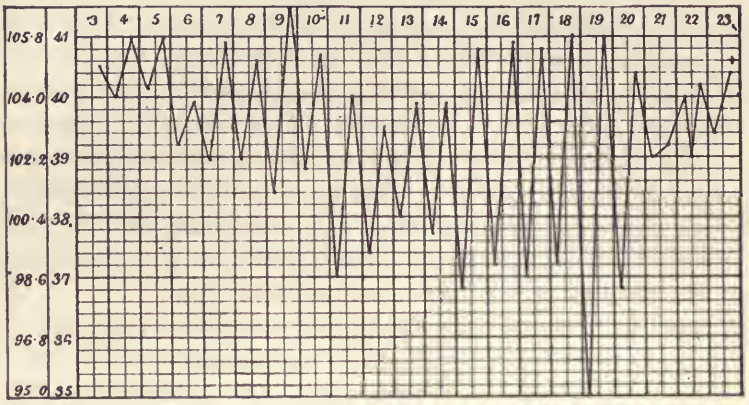
In a typical case of acute miliary tuberculosis the symptoms are as follows: The patient complains of malaise, anorexia, prostration, and fever for two or three days. The pulse is accelerated, and there is a sensation of thoracic oppression. The temperature rises rapidly in a few days to about 104° F., though it presents no regular course. Respiration becomes greatly accelerated, and finally its rate reaches 40 or 50, or even 60, per minute. The pulse may be 140. There is marked pallor and cyanosis, and lips, cheeks, and finger-tips become purple. Without special pain, the patient feels very ill and dull, and prostration becomes more and more marked. There is a cough which may be dry and obstinate, or there may be muco-purulent sputum. The rapid pulse and respiration continue, the temperature is irregular, but often of a remittent type, and the patient passes into a typhoid condition. If the serous membranes are affected, the temperature remains higher. Nervous symptoms develop: there may be muscular twitchings and carphologia. The dulness deepens into coma, or there is a low muttering delirium; the tongue is brown, dry, and fissured; there is herpes labialis; the evacuations become involuntary; and the patient dies in collapse or coma, with pulmonary œdema. There may be uncontrollable perspiration, producing sudamina, and occasionally there is a roseola upon the chest and abdomen. Albuminuria is frequently observed, and sometimes peptonuria is present. The mind may remain clear until the approach of death, but the patient frequently becomes dull and listless, or there may be anxiety, restlessness, and delirium. Acute mania has been observed.

Special Symptoms.—The onset is usually gradual, as in the case described, but it may be acute, with one or more rigors and rapid rise of temperature.

The pulse is always rapid, and it soon becomes very feeble and compressible. It ranges between 120 and 160.

The temperature is very irregular. (See Fig. 22.) It may be intermittent or remittent, or the evening fever may be higher than the morning. Just before death it may rise to 107° or 108° F. In some cases it remains as low as 102° throughout the disease. Not rarely the morning temperature is higher than that of the evening. Some writers divide the disease into two types, based upon the character of the fever—namely, the "typhoid" and the "intermittent" forms.

FIG. 22.



Temperature Chart of Case of Acute Miliary Tuberculosis (Wunderlich).

The temperature depends upon the infection of the blood rather than upon the extent of tubercular deposit.

The respiration is always accelerated. It may be deep at first, but soon becomes shallow. The accessory respiratory muscles are called into action and the nostrils work. The patient does not always complain of shortness of breath. In young children the respiration may exceed 85, and in adults it occasionally rises above 60.

There is usually more or less bronchitis, which excites a hacking, dry cough or gives rise to expectoration, which may be clear and frothy or muco-purulent. Rarely the sputum is streaked with blood. Tubercle bacilli are not found in the sputum unless there was a pre-existent phthisis. Hæmoptysis is not a feature of the disease. There may be a few rigors at first, and there is more perspiration as the prostration advances.

Vomiting may occur at the onset, but there is nothing typical about it.

The bowels are often constipated.

The urine shows the modifications produced by any febrile state, and there may be slight albuminuria.

Physical Signs.—The disease is characterized by the peculiar absence of physical signs in the chest. Previously existing phthisis is occasionally present, and there is commonly enough bronchitis to occasion a few moist râles or rhonchi, and, if there be a tubercular pleurisy, there may be friction sounds; but neither percussion nor careful auscultation reveals any signs directly attrib-

utable to the miliary tubercles, even though they may be present in the lungs in great numbers. The reason for this is that the tubercles do not involve or compress the air-cells to any extent, nor are they close enough together to modify in any manner the percussion or auscultatory sounds of the chest.

Course.—The course of the disease will vary with the predominance of certain symptoms, in accordance with the degree of involvement of various organs. In some cases the typhoid condition is the prominent feature; in others, the subjective dyspnœa, cyanosis, and rapid breathing, or uncontrollable constant dry cough; in others, symptoms of peritoneal tuberculosis are distinctive; and finally tubercular invasion of the meninges of the brain may occasion symptoms so severe that they divert attention from the possibility of a general infection. In the latter variety the tubercles involve the base of the brain chiefly, and occur near the pons, optic chiasma, or in connection with the cranial nerves. There are violent headache, delirium or stupor, photophobia; and frequently symptoms of local pressure are evident, as unilateral convulsions or paralyses of the extremities or facial muscles, strabismus, and variations in size of the pupils. There is rigidity of the back of the neck. There is usually extreme dyspnœa in this variety of the disease.

Exceptionally, the disease exhibits a somewhat intermittent type, and the course is protracted for several weeks by irregular remissions, or even intermissions and exacerbations, of the fever and other symptoms.

The duration of the disease is commonly between three and four weeks. Exceptionally, bad cases are fatal in ten days, while mild cases sometimes are prolonged for as many weeks.

The patient commonly dies of exhaustion from the fever and the systemic effects of the virus, or death results from pulmonary œdema or from simple heart failure. The disease is uniformly fatal.

Diagnosis.—The diagnosis is largely based upon the exclusion of grave forms of pulmonary disease and the presence of the extreme dyspnœa and prostration with fever, and a rapid, feeble pulse.

Acute miliary tuberculosis is distinguished from meningitis by the greater frequency of pulse and respiration, the clearer mental condition, and the lack of the distinctly cerebral symptoms which belong to the latter disease.

There are some cases of acute miliary tuberculosis which so closely resemble typhoid fever that it is almost impossible to make a diagnosis with absolute certainty. These are the cases of the "typhoid" type in which the pyrexia, prostration, and general appearance are almost identical with the symptoms of enteric fever. The diagnosis must be based upon the following features: In typhoid fever there is frequently an eruption of rose-colored spots on the abdomen, back, and possibly the extremities. There is enlargement of the spleen and more or less tympanites, with some "pea-soup" stools. There is the history of a slow invasion, with marked frontal headache, epistaxis, and gradual daily rise of temperature during the first week. In miliary tuberculosis, on the other hand, the invasion and prostration are more sudden, the temperature is more irregular, there is marked rapidity of respiration with a very rapid

pulse, and there is no abdominal eruption or splenic enlargement. The disease advances much more rapidly than does typhoid fever.

When there are chills at the onset the disease has been sometimes mistaken for malarial fever; but the typical paroxysms, the enlarged spleen of the latter disease, and the influence of quinine will establish the diagnosis.

If there be much cough and expectoration, miliary tuberculosis may be mistaken for a severe acute general bronchitis, especially in advanced life; but the more rapid prostration, extremely rapid breathing, and rapid pulse of miliary tuberculosis soon resolve the doubt.

Prognosis.—The prognosis is always bad. It is generally believed that recovery is impossible, and that reported recoveries were instances of errors in diagnosis. Death will occur sooner in proportion to the number of different organs involved and the suddenness and severity of the invasion. Death occurs early in cases with meningeal tubercles.

Treatment.—The treatment is purely symptomatic, and remedies given with any other view are useless. The only indications which can be met are to stimulate the heart and to relieve the dyspnœa or other suffering by morphine.

If the temperature be very high, cold alcohol sponge-baths may be given; cardiac stimulants and alcohol must be administered to sustain the heart. The diet must consist of milk, broths, eggs, beef-juice, egg-nog, milk-punch, etc.

If there be meningeal complication, to relieve headache and delirium leeches may be applied behind the temples and an ice-bag placed upon the head. Severe cough should be controlled by sedatives and narcotics. Small hypodermic injections of morphine may be given to quiet restlessness and make the patient more comfortable. Codeine in half-grain doses acts well in relieving cough and restlessness.

SCROFULA.

By W. GILMAN THOMPSON.

Definition.—Scrofula is not a distinct disease, for it has no symptoms or lesions peculiarly its own; but it is a morbid condition of the system or a diathesis resulting from malnutrition and characterized by hyperplasia and inflammation, with caseation or suppuration of the lymphatic glands, and by a tendency to tedious, intractable inflammations of the skin, mucous membranes, bones, and joints. Persons are called scrofulous who exhibit this morbid tendency, although at the time they may not be actually suffering from any lesion. Many cases which formerly were described as scrofulous are now recognized as tuberculous or due to hereditary syphilis.

Synonyms.—Scrofulosis; Morbus scrophulosus; Scrophulose, Skrofeln (Ger.); Scrofule, Scrofulose (Fr.); King's Evil; the Evil; Quince; Cruels. Struma is used synonymously with scrofula by certain English writers. In Germany the name struma is applied to enlargement of the thyroid gland. When the cervical glands are greatly swollen the normal constriction between the head and shoulders is obliterated, and, as in the pig, there is no neck; hence the name scrofula, from *scrofa*, a pig.

Etiology.—Scrofula as a morbid tendency is either hereditary or acquired, and it is so closely related to tuberculosis that many believe the scrofulous condition to be merely one of the manifestations of chronic tubercular disease. There is some dispute as to the hereditary nature of scrofula. The term "hereditary" must not be regarded as implying that the scrofulous lesions are congenital, but merely that the constitution of the infant is so modified by its inheritance that, although it may be born in a fairly healthy condition, its feeble resisting power very soon succumbs to the scrofulous taint and the development of the various scrofulous lesions. Viewed in this light, the majority of cases are "hereditary." Hereditary scrofula manifests itself in the offspring when one or both parents are themselves scrofulous, the subjects of phthisis, carcinoma, other wasting disease, or chronic alcoholism; or when both parents are very young, too old, when there is too great a difference between their ages, or when they are closely related by blood. It is true that the marriage of such parents may be followed by the birth of healthy children, but they are far more apt to be scrofulous, especially when both parents are at fault.

Acquired scrofula may occur in children who were previously perfectly healthy. Poor hygienic surroundings influence the development of scrofula as much as any single factor. Foul air, damp, dark, ill-ventilated rooms, over-

crowding, indoor life, insufficient and improper diet,—all promote the development or acquirement of the diathesis.

Locality, climate, and *season* have no influence beyond the general hygienic conditions which they favor. Thus, scrofula is most frequently encountered in the temperate zone in a severe winter or a damp spring-time, and in a cold, moist locality which begets frequent catarrhs or compels an indoor life.

A diet of coarse, starchy food, without sufficient nitrogenous material, is a potent factor in the development of scrofula. Children nursed by a healthy mother are much less apt to become scrofulous than if they are fed upon artificial infant foods.

Sex has no special influence upon scrofula, but females are more apt to suffer from the glandular lesions, and males from caries (Lynch).

Age.—Scrofula is essentially a condition of childhood. It is rare in the first year, and is commonest between the ages of two and fourteen years, or from the establishment of the first dentition to puberty. It may exceptionally develop somewhat later in life or be acquired after puberty. It may thus occur among inmates of overcrowded workhouses or prisons, but is very rare after twenty-five or thirty years of age.

Social Conditions.—Scrofula is far more common among the children of the very poor, owing to unfavorable diet and lack of proper hygienic surroundings, but it is by no means unknown among the children of the wealthy, in whom heredity, overwork in school, and dietetic errors are predisposing causes.

Race.—In the United States the Irish, negroes, and the Hebrew children seem to be the most frequent subjects of scrofulosis. It has been suggested that this may be due to the quality of the food in previous generations.

Scrofula is less frequently observed than formerly, now that its causation and treatment are understood, and it is less common in the United States than in Europe.

The scrofulous diathesis may be evoked as a sequel to measles, scarlatina, frequent attacks of croup, and other debilitating diseases of childhood.

Many German pathologists, led by Koch, regard scrofula as a special clinical form of chronic tuberculosis of the lymph-glands, owing its existence to tubercle bacilli. Strümpell says that most scrofulous children do not *become* tubercular, but they already are so. Others believe that scrofula is not identical with tuberculosis, because the tubercle bacilli cannot invariably be discovered in scrofulous subjects, and in many such persons tubercular tissue is not produced. Tuberculosis is readily inoculated in non-scrofulous animals and men, and it is not necessarily accompanied by the enlargement of the lymphatic glands which is so common in scrofula. It is also true that fatal cases of phthisis seldom exhibit scrofulous symptoms or lesions. The scrofulous diathesis is often inherited, but tuberculosis is not; yet scrofulous subjects are very prone to acquire tuberculosis, because they are readily affected by any morbific influence, and their tissues afford excellent soil for the development of tubercle bacilli. Much argument has been expended upon both sides of

the question, but the tendency is fast becoming universal to give less prominence to scrofula as a morbid condition, and to include it in descriptions of chronic tuberculosis.

In scrofula the system is highly predisposed to local inflammations, with the added danger that the inflamed area, if not originally tubercular, may easily become infected with tubercle (Eichhorst). Any inflammation occurring in a scrofulous subject assumes a scrofulous type—*i. e.* it exhibits slow development or chronicity, lack of response to treatment, abundant cell-production, and tendency to caseation. The cellular infiltration inclines to remain permanent, instead of terminating, as is usual in non-scrofulous inflammation, in resolution, organization, or suppuration.

Morbid Anatomy.—There are no anatomical lesions peculiar to scrofula. They belong rather to the lesions of tuberculosis. The old theory that scrofula is caused solely by altered and impoverished blood is abandoned, although the blood and other alkaline fluids of the body may be less alkaline than usual. Lesions which scrofula exhibits are due primarily to cell-proliferation and inflammation. It is a diathesis of childhood, and young children have relatively large and numerous lymph-spaces and active lymph-cells, and their tissues are less compact than in adult life (Formad). In the case of scrofula the lymphoid tissues, either from nutritive disorder or some inherent peculiarity not yet understood, readily exhibit abnormal cell-growth. The capillaries supplying such tissues are inadequate, and the tissues consequently suffer from a deficient supply of nutritive food (Lynch). Hence it is that all inflammatory processes occurring in the scrofulous resolve very slowly. There is a preponderance of cellular elements in the scrofulous exudations, and the cells have low vitality (Virchow).

The common lesions of scrofula are those of—

(1) The lymphatic glands;

(2) The mucous membranes;

(3) The skin;

(4) The bones and joints.

(1) Enlargement of the Lymphatic Glands occurs so constantly at one time or another in the course of scrofulous cases that it is almost pathognomonic. The lymphatic glands most frequently involved are the cervical and submaxillary. Other glands affected may be the occipital, cubital, axillary, inguinal, bronchial, and mesenteric. There are two types of scrofulous glandular lesion:

(*a*) Simple hyperplasia; (*b*) Inflammation. They may occur independently, or either may precede the other. Both are usually excited by some irritation of the adjacent skin or mucous membrane, which results in the production of morbid materials, which are conveyed through the lymphatic vessels to the glands. Such irritation may proceed from disease of the tonsils, teeth, ear, etc. The Koch tubercle bacillus is found in many, but not all of the glands.

(*a*) Simple hyperplasia, or enlargement of the gland from multiplication of its cellular elements, is excited by exceedingly slight irritation, which in the

non-scrofulous would produce no appreciable effect. This hyperplasia may occur in a gland without previous inflammation, and the process is very gradual. The gland becomes greatly swollen, and feels hard, smooth, and tense. It is not painful, and is freely movable beneath the skin. Rarely one gland alone is involved; more often a series of glands becomes enlarged, forming a knotted chain. If the cellular proliferation continues, the glands will form a very large mass, which disfigures the neck and interferes with the proper movement of the head. The microscope reveals no foreign elements in such glands, and the tissue of the stroma is not increased. Since the lymphoid cells only are multiplied, it is easy to understand how the enlarged glands occasionally return to the normal size, for in health the number of cells varies considerably. It is difficult to trace the source of the new cells. They may come to the gland through the lymph-channels from some distant inflamed area, or they may spring from the normal gland-cells, or they are possibly wandering leucocytes.

Sometimes parts of a gland undergo anæmic necrosis and form caseous matter without active inflammation, although such a mass really acts as a foreign body, and is liable to originate inflammation.

(*b*) Inflammation, when it affects the glands, causes an increase in the number of lymphoid cells in the sinuses and follicles. The cells are swollen and have large nuclei. The white blood-cells in the vicinity are swollen by imbibition of albumin (Lynch), and they degenerate. In the glands there are at first hyperæmia and an exudation which is either diffuse or localized, forming nodular masses, which may resolve or suppurate and result in abscesses, or which more often are converted into a dry, friable, caseous material. In the mediastinum and mesentery the glands are apt to become cretaceous. The cut surface of the gland shows irregular yellowish or white spots of cheesy or calcareous material. The spots may be few and isolated, or all the normal gland-structure be replaced by cheesy matter enclosed by a thickened capsule. These processes may be acute, but they are usually very protracted, and the glands quite lose their vascularity and often become nodular. When the cellular elements undergo fatty degeneration and caseation, it is because the scanty blood-supply, still further reduced by the pressure of the swollen gland, does not furnish enough alkaline blood-plasma to liquefy the mass, and what is supplied is carried off too rapidly in the relatively large lymph-channels (Cornil and Ranvier). There is periglandular inflammation in the surrounding areolar tissue, and the capsule of the gland becomes thickened and is permeated with round cells. The overlying skin becomes adherent, bluish, and thin.

Giant cells and tubercles are very often found in the glands, and sometimes there are tubercle bacilli. The enlarged bronchial glands may produce pressure symptoms, or may suppurate and ulcerate through the bronchial mucous membrane and occasion broncho-pneumonia. The enlarged mesenteric glands occasion chronic enteritis and diarrhœa.

(2) The Lesions of the Mucous Membranes may arise by extension

from the skin, or they may spread from the mucous membranes to the skin. Hypersecretion prevails, and it is excited by the slightest irritation. There may be conjunctivitis, catarrhal ophthalmia, or suppurative inflammation of the middle ear, resulting in perforation of the membrana tympani and a mucopurulent, offensive discharge. Opacities of the cornea may be produced which last through life. There is a marked tendency to catarrhs of the nose and pharynx, which are very chronic, and the mucous membrane is covered with thick exudation which encrusts it, forming scabs. Coryza, laryngitis, and bronchitis occur, and are very persistent. Inflammations of other mucous membranes are less frequent. Scrofulous ulcers are indolent, with anæmic overhanging edges; they heal very slowly, and often a cicatrix closes in one part of an ulcer while it breaks down again and opens at another.

(3) The Lesions of the Skin are of considerable variety. When the diathesis exists, any skin disorder is affected by it and assumes the scrofulous type. Impetiginous eczema is the commonest of the scrofulodermata, and the face, hairy scalp, or extremities are affected by it. Lupus, prurigo, and lichen also occur.

(4) The Bones which are most frequently the seat of scrofulous changes are those about the ankle, the femur, and the vertebræ. The character of the inflammation is a "fungous" osteitis or periostitis, and the joints may be affected by synovitis, white swelling, or general arthritis. There may be caries, necrosis, and extensive suppuration, ending in total destruction of a joint. Tubercle bacilli may be found in most of the fungous bone-lesions. The bones and joints may be diseased without glandular enlargement, and conversely.

Symptomatology.—Different scrofulous patients rarely present identical pictures of disease, but most of them may be classed under two general types. These subdivisions are not very exact, and there are many scrofulous children who present the features of neither or of both in combination; still, from a clinical standpoint it is convenient to select distinctive types for description, and the following is the classification which has long been in use. The two types are—I. *The Erethitic*, and II. *The Phlegmatic.*

I. The Erethitic or Sanguine Variety.—In this form, which is usually hereditary, the child looks delicate and often pretty. The skin is fair and transparent, showing the blue veins distinctly, and blushing easily occurs. The eyelashes are long, the features are small, the weight is light, the muscles are soft, and the bones are slender. The hair is fine and the teeth appear early. The nervous system is apt to be irritable, with unnatural activity, but the mental development is good. This type is more frequent in females than males, and more apt to furnish severe and even fatal cases.

II. The Phlegmatic Type.—The phlegmatic or torpid variety is more apt to be acquired than inherited. The child is stout, clumsy, and thick-set. The expression is dull and heavy. The head is large. The upper lip and nose are full and the eyebrows thick. The chin is prominent. The skin is coarse and spongy. The nervous system is far less acutely irritable than in

the erethitic variety. The abdomen is large. The cervical glands are increased in size, and there is usually a naso-pharyngeal catarrh. Children with this type of scrofula have eczema and chilblains, and their wounds heal poorly. The muscles are soft, and there is over-production of subcutaneous fat. The pulse is feeble, and the temperature at times is subnormal. In bad cases the glandular enlargement becomes extreme, and a number of glands in the neck, thorax, or mesentery are involved. If a gland becomes acutely inflamed, there may be local pain and moderate fever, which disappear after extirpation of the gland or incision of the abscess. In addition, any of the anatomical lesions above described may give rise to special symptoms. If the mesenteric glands are involved, they can sometimes be felt through the boat-shaped abdomen, and there is exhausting diarrhœa. The child becomes pale, thin, and hollow-eyed, the hair falls out, and the skin is dry and thin. There is often retarded mental development. Females frequently have leucorrhœa.

In many cases the evidences of scrofula remain latent until evoked by some trivial excitation, as a slight bruise on a joint, which results in inflammation, swelling, hydrarthrosis, and final disintegration of the entire structure; or a spot of facial eczema may spread to the conjunctiva, and finally excite ophthalmia, with permanent impairment of vision; or a simple bronchitis ends in catarrhal pneumonia, with various accompanying symptoms of scrofula.

Course and Duration.—The duration of the affection depends largely on the ability of the parents to secure proper surroundings and treatment for the child. Advanced cases yield very slowly to treatment even under the best conditions. There is often periodic improvement, followed by exacerbation of the symptoms. The same symptoms may recur again and again, and new lesions will develop while old ones disappear. After puberty the scrofulous diathesis tends to disappear.

Terminations and Complications.—The majority of scrofulous cases recover completely, and the catarrhs and cutaneous eruptions are amenable to treatment. When the bones and joints are extensively involved, with necroses, abscesses, and fistulæ, the prolonged suppuration is apt to engender amyloid visceral disease, which eventually proves fatal. This occurs in the kidney, liver, or spleen. Enlarged bronchial glands may suppurate and ulcerate into the bronchi, and death may result from pneumonia. Inflammation of the mesenteric glands, accompanied by chronic intestinal catarrh and diarrhœa, causes death in young children more frequently than any other scrofulous condition. A less frequent but often fatal termination is catarrh of the middle ear, when it involves the mastoid cells and the inflammation extends to the meninges of the brain. The various scrofulous diseases of the bones and joints may result in permanent ankylosis or other disfigurement.

Scrofula so weakens the system that severe intercurrent diseases are much more fatal when they occur in connection with it than they are in a previously healthy person, and all accompanying morbid processes retrograde very slowly. Scrofulous children may die from croup, hydrocephalus, intestinal tuberculosis, or acute miliary tuberculosis.

Diagnosis.—In typical cases scrofula is easily identified. Separate lesions of the skin or bones, etc. may give rise to some doubt as to whether they are of scrofulous origin or are due to some other cause. In such instances the diagnosis of scrofula can usually be made from a careful study of the history of the patient and from the extremely chronic character of the inflammation and its slow development, the tendency to cell-proliferation and to caseation of lymphatic glands.

Congenital or acquired syphilis may be confounded with scrofula. In hereditary syphilis the lesions appear either at birth or much earlier than they do in scrofula, and, moreover, a history of syphilis is usually obtainable, and the disease yields promptly to mercury and potassium iodide.

Prognosis.—The prognosis is favorable when the patient is seen early, and when he can at once be placed under proper hygienic regimen and dietetic treatment. The prognosis is less favorable when the hereditary factors are strongly marked, and when the bones and joints or the bronchial and mesenteric glands are severely involved. In bad cases there is present a liability to miliary tuberculosis.

Treatment.—The treatment comprises (1) prophylaxis; (2) hygiene; (3) dietetic and (4) tonic measures; (5) the control of the local inflammations.

(1) *Prophylaxis.*—The ideal prophylaxis would include the prevention of marriage among all persons who are tubercular, scrofulous, or actively syphilitic, who are suffering from wasting cachexias or malignant disease, and among those whose age or consanguinity makes them unlikely to beget healthy offspring. Such extreme measures are obviously unattainable. It is possible, however, to do much for the children of these parents. An infant born with such heritage, whether appearing scrofulous or not, should not be nursed by its feeble, anæmic mother, but should have a vigorous wet-nurse. Failing this, it must be fed on the best of cow's milk, avoiding artificial foods. Unusual care must be taken at all times to secure an abundant supply of pure fresh air, with proper personal cleanliness and warm clothing. A child with enlarged lymphatic glands should be protected with great care from taking cold and from irritation which may establish chronic catarrhs. Enlarged tonsils should be excised. By giving careful attention to the details which follow many weak infants may be successfully carried through the period of greatest liability to scrofulous manifestations—namely, that which lies between the second and fourteenth years.

(2) *Hygienic Treatment.*—Scrofulous children should be kept in the open air as much as possible, and hence country life is best for them. They improve either among the mountains or at the seashore. Children having glandular enlargement are said to do better at the seaside than those who are eczematous (Bergeron). Sea-bathing is particularly beneficial in the former cases. It has been found in asylums and hospitals that children may become scrofulous even with proper diet and cleanliness, provided the air be damp, close, and impure. Special pains should be taken to keep the bowels in regular action, and to maintain the functions of the skin by clothing of proper

warmth and unirritating texture, and by cold baths for the stronger children or tepid sponge-baths for the feeble ones.

Older children should not be allowed to overtax their energy in study or confining work of any kind, and plenty of sleep is important. Well-regulated exercise in the open air should be taken daily; in very feeble cases massage is of service.

(3) *Dietetic Treatment.*—The diet should be simple and nutritious, and must contain a large proportion of nitrogenous food. It is common for the poor to feed their scrofulous children on bulky, starchy food—potatoes, etc.—which is not in itself injurious, but, by reason of its large volume in proportion to its nutritive value, overtaxes the enfeebled digestive powers. This error should be corrected, when possible, by substituting a portion of milk, eggs, or meat in the diet.

(4) *Tonic Treatment.*—The use of cod-liver oil for scrofulous affections has long proved so beneficial that it has been regarded by some as a specific. It is, however, simply a readily assimilable form of fatty food, and as such it proves of great value when properly administered. It is far more useful, as a rule, in the erethitic than in the torpid type. From 1 to 2 drachms should be given to children two or three times daily, an hour after meals; and because it must be continued for a long period, it is well to suspend its exhibition once a month for ten days, in order to prevent it from disgusting the patient. When it provokes decided distaste, the dose will often be taken with the promise of a peppermint lozenge to follow. The oil should be omitted in very hot weather. In simple cases, when possible, it is best to give the pure oil: otherwise, a carefully prepared emulsion with phosphates may be substituted. Iron, arsenic, and iodide of potassium are the other tonics which prove of most value, and the latter is particularly beneficial when glandular hyperplasia is prominent. The syrup of the iodide of iron is a very useful tonic for young scrofulous children, in doses of from 10 to 30 minims two or three times a day, well diluted. Pure beechwood creasote in doses of one-quarter or one-half of a drop three times daily, given in milk, is favorably recommended by Forchheimer, presumably upon the theory of the tubercular origin of the scrofulous condition, which is strongly advocated by him. Sulphide of calcium is believed by some to exert a beneficial influence upon the patient's general condition, and especially upon glandular enlargement or suppuration.

(5) *Treatment of the Inflammatory and Other Symptoms.*—When the bones or joints are implicated it is useful to add lactophosphates or hypophosphites of lime and sodium to the cod-liver oil. These hypophosphites, as well as bicarbonates of sodium or potassium, are to be given when glandular enlargement exists; and their utility has been referred to the asserted deficiency of alkalies in the blood.

The details of local treatment of the diseased bones and joints belong to surgery, and of the various eczematous disorders to dermatology.

The enlarged glands are not much benefited by local injection, inunction,

or applications of iodine, iodoform, or other remedies. If they become inflamed or if painful and suppurating, they should be poulticed and incised, for if left to suppurate and open of themselves, they leave very ugly stellate cicatrices and fistulæ. If general improvement in the patient's condition and removal of local sources of irritation, such as carious teeth, catarrhs, etc., are not followed by reduction in size of the glands, and if they are unsightly or increase in size, it is well to extirpate them. Sometimes they are removed by the scoop, but as they are apt to soften irregularly, and not uniformly, it is best to completely excise them by the knife. So long as they remain there is a risk, though a moderate one, of their breaking down and serving as foci for general distribution of pus or caseous material in the system.

Summary of Treatment.—Good hygiene; abundant and largely nitrogenous diet; tonics, such as cod-liver oil, lime salts, and iodides; surgical measures for the bone and joint complications.

SYPHILIS.

By W. GILMAN THOMPSON.

Definition.—Syphilis is a chronic infectious disease, communicated only by inoculation. It is characterized by an initial lesion of the mucous membrane or skin, accompanied by glandular enlargement and followed by a great variety of chronic lesions, the most typical of which is the gumma.

Synonyms.—Lues venerea; Lustseuche (Ger.); Mal vénérien (Fr.); the Pox.

Certain authors, as Hutchinson, class syphilis with the exanthemata, since it exhibits a period of incubation, of exacerbation, of remission, and of relapse in its final or tertiary stage. There are peculiar facts which are in accord with this view, but the types of the disease and its lesions are so numerous and varied that by a majority of writers it is classed by itself.

The type of syphilis may be benign or malignant or exhibit any intermediate grade of severity. It is almost always chronic, and only exceptionally acute if very malignant.

Etiology.—Syphilis is either acquired or inherited. The latter form is far less frequent than the former, and it will be separately considered. (*Vide infra.*)

Acquired Syphilis is transmitted by inoculation through the agency of the discharges proceeding from any of the earlier lesions of the disease. It is thus communicated to a previously healthy person through an abraded surface of skin or mucous membrane. It is ordinarily acquired during sexual intercourse through very slight abrasions of the mucous surface of the genitalia, and possibly by direct absorption where the surface is delicate, as it is over the prepuce and glans penis.

Since the propagation of syphilis is not necessarily confined to venereal acts, it is convenient to speak of genital syphilis in distinction from extragenital syphilis, referring solely to the mode of acquisition of the primary lesion. Thus, instruments carelessly used about the throat or mouth of a syphilitic subject, infected razors, pipes, drinking vessels or eating utensils, the operation of tattooing, direct vaccination (especially if the pustule contain blood), or kissing with chapped or cracked lips,—are all more or less common agencies in the transmission of the virus from the infected to the healthy.

Syphilis which occurs in young children who have lived among adults affected with the disease is quite as apt to be acquired as hereditary.

A healthy mother may be inoculated by her own child, who has acquired the disease from an infected nurse.

It is not possible to reinoculate a person with syphilitic virus if he is already under the constitutional influence of the disease, and it is very rare to have more than a single point of original inoculation.

The period of viability of the syphilitic virus is not known, but it is supposed to retain its virulence for months, if not longer, when removed from the body. Syphilis can be inoculated directly from the blood or from the secretions of sores, mucous patches, etc. of patients who are in the early stages of the disease. Epidemics have several times been caused by syphilitic midwives inoculating a number of mothers, who in turn have inoculated their own children and husbands.

The Bacillus.—The most recent view of the cause of syphilis, and the theory which is gaining many converts, is that it is due to a micro-organism. Klebs, Birch-Hirschfeld, Lustgarten, and others have described a bacillus found in various syphilitic lesions. In 1884, Lustgarten described the bacillus of syphilis as being smaller than the bacillus tuberculosis, and occurring in curved or straight forms, singly, in pairs, or in little groups or chains, but not in rods. The length is from two to seven thousandths of a millimetre, and the width is about three ten-thousandths. It is believed to possess spores. It is not found free, but is contained in round cells. Klemperer and other observers have verified the existence of this bacillus, and it is believed by many that it constitutes the active agent in the propagation of syphilis. This germ is found in the excretions of syphilitic sores and in some gummata and condylomata.

It may be possible that the constitutional symptoms are caused by products developed by the action of the germ in the tissues with which it is in contact, as in the case of diphtheria, typhoid fever, and other diseases; but further research is required before this theory can be regarded as established. Inoculation and cultivation tests made with the bacillus of syphilis have not yet proved as convincing as they have with other bacilli, and therefore the belief in the causative relation of the germ to the disease is at present based chiefly upon analogy, but there is very reasonable ground for its acceptance.

It is doubtful whether true syphilis exists among the lower animals. It is said to have been inoculated successfully from man into rabbits and apes.

Distribution.—Syphilis occurs in all parts of the civilized world and among many savage nations. In past centuries it is said to have been epidemic and very malignant. Among civilized people the disease is so much better understood, and so much better treated than formerly, that upon the whole it is becoming milder. It is common among soldiers and sailors, and is carried by them especially to garrison and seaport towns and cities. Its spread is effected almost exclusively by existing social conditions; hence it is comparatively infrequent in rural districts, but common in cities or wherever overcrowding and poverty combine to favor lax morality among the sexes.

Park declares that he has encountered syphilis among the natives in parts of Africa never before visited by any white man or Arab; and that the Monbuttu and Bari tribes in the Equatorial Province inoculate with syphilitic virus,

with the result that a rash and other familiar symptoms soon appear, but the course of the disease seems to be favorably modified.

Apparently every race is susceptible to inoculation, and individual immunity is certainly very rare. When syphilis is introduced among new races, as it was conveyed by Europeans to the native Sandwich Islanders, it works great havoc. Among the Chinese the disease is apparently more virulent than it is with other races, at least when it is contracted from them by sailors.

GENERAL DESCRIPTION.—The lesions of syphilis, as well as its clinical history, are divided into three stages or periods, called respectively the primary, secondary, and tertiary. While this division is of value in the description of the disease, it must be stated that in actual practice it is found that wide variations occur, the symptoms of one period occurring with those of another with great frequency.

The primary period is that of local manifestation, characterized by the appearance and development of the "initial lesion" or "chancre," a sore produced by inoculation, with accompanying enlargement of neighboring lymphatic glands. This period lasts for about six weeks on the average. It is also sometimes designated as the "incubation stage" of the secondary period.

All acquired syphilis is believed to originate with a chancre. When none is found, it has either eluded observation or it has been slight and has healed before examination was made for it. Occasionally it escapes detection by occurring within the urethra.

The secondary period is characterized by the development of general or constitutional symptoms, particularly a diffuse roseola and the "mucous patch." It lasts for a varying length of time, from two months up to two or three years. As a general rule, the secondary symptoms and lesions have disappeared at the end of the second year. This period embraces the mild and earlier lesions of the mucous membranes and skin, and some of those of the viscera and nerves.

It is often very difficult to draw a strict line of demarcation between the lesions of the second and third stages, and, moreover, the secondary eruption frequently appears before the primary sore is cicatrized. There may be a latent period of several months or many years between the disappearance of the secondary and the beginning of the tertiary lesions, or the two periods may overlap.

The *tertiary period* commonly commences between the third and sixth years, although there are wide departures on both sides of these limits. It is characterized by the development of inflammatory growths called "gummata," and by a great variety of visceral, cutaneous, mucous, and nerve lesions. Its duration is influenced by treatment, but it may last from one or two to twenty years or more, and the lesions, once formed, may outlast the activity of the disease.

The lesions of the second stage are often symmetrically disposed on both sides of the body, but the tertiary lesions are noted for their asymmetry.

While syphilis is inoculable during the first and second periods, the tertiary

form is non-infectious, as a rule, although Fournier and other experienced observers have seen cases of transmission of the tertiary disease.

Morbid Anatomy.—In general the morbid processes of syphilis are characterized far more by cell-proliferation than by pus-formation.

The morbid changes resulting in any form of syphilis may be peculiar to the disease or they may be common to other diseases, and yet when they occur in syphilis they elect a peculiar site and distribution, as is the case with many cutaneous eruptions. The more that congestive or inflammatory processes characterize a lesion—that is, the more active they are—the less special or peculiar will that lesion become, because new tissue requires time for its formation (Gowers).

The chief varieties of syphilitic lesions may be grouped under the general terms of inflammation and tissue-formation. The former develops early, as, for example, roseola and certain other skin affections. The latter appears much later, in the growths which are termed gummata, etc. A variety of non-typical inflammations may arise in the course of syphilitic disease, but there is only one growth, the gumma, which is in any degree characteristic.

The lesions present, as stated, wide variation in the time of their appearance, but they are here considered in the order in which they are most frequently observed.

Lesions of the Three Periods.—The typical *chancre*, called also the "initial lesion," "hard" or "Hunterian chancre," is developed at the site of inoculation on the abraded mucous membrane or skin, and characterized by hyperæmia and cellular infiltration, which are followed by an ulcerating papule or a shallow, indolent ulcer. It is usually quite small, being about the size of a split pea, but it may become considerably larger. It feels hard to the touch, and presents a circumscribed induration from infiltration of cells around its base. It is of a dark-reddish hue, and is coated by a glairy, viscid, thin secretion, presenting a glazed aspect. It is further characterized by the absence of irritation and of the formation of pus.

The appearance of the chancre will vary with its site. Chancres of the lips, prepuce, labia majora, or scrotum exhibit extended induration, and one can readily isolate them by manipulation; but on the glans penis, cervix, or where the surface is firmly bound there is much less hardness. The explanation of this fact is that in loose connective tissue the cellular infiltration readily extends without compressing the blood-vessels, but where the connective tissue is very scanty the chancre, deprived of nutrition, readily ulcerates without the occurrence of much induration.

Sometimes the lesion becomes papular or it is large and flat, or a large sore upon the lip may resemble epithelioma. Quite exceptionally it develops into a warty growth.

The most frequent site for chancre in the male subject is on the prepuce or at the base or surface of the glans penis. It may occur at the external urethral meatus or, exceptionally, within the urethra. In females it is more frequently found upon the labia or at the posterior commissure than deep within the vagina.

Next to the genitalia, the most frequent sites for the primary lesion are the parts about the head, mouth, and throat, and the hands. Cephalic chancres have about the same average duration as those of the genitalia.

The initial lesion, subjected to irritation from clothing, coition, etc., may become greatly inflamed. Pus forms and an extensive slough follows, which may be phagedænic or even gangrenous. This is more liable to occur in persons possessing a predisposition to suppuration from any cause, and in those addicted to alcoholic and venereal excesses.

In case of a very moderate degree of primary erosion with no induration the glands are but slightly swollen, but sometimes the erosion may be overlooked, and the intumescence of the glands is then the only symptom of the primary stage. In many cases the initial sclerosis results in a ridge or plaque or nodule, which persists into the secondary period.

A certain amount of lymphatic adenitis follows every syphilitic inoculation, for the virus finds its way to the general circulation through the lymph-glands. It commences from the eighth to the fourteenth day after the chancre appears, and lingers for at least six or seven weeks, or through the period of secondary incubation. Extra-genital initial lesions are often accompanied by considerable glandular enlargement.

The degree of enlargement frequently, but not always, is commensurate with the extent of the chancre, and it appears synchronously with the induration about the primary sore. It occurs usually in those glands situated nearest to the site of the primary lesion, and upon the same side with it. Thus with genital chancre the glands first affected are the inguinal, and in oral chancre they are the glands of the neck and angle of the jaw. There are exceptions to this rule, however, and sometimes glands on the opposite side of the body become first hypertrophied. Moreover, a chancre on the median line is not always followed by a bilateral glandular enlargement. At first one gland becomes much more prominent than the rest, and the size varies from a bean to a small hen's egg. After some time the glands on the opposite side, corresponding to those first affected, begin to enlarge. The extent of the adenopathy will depend on the number of glands with which the lymphatic vessels in the vicinity of the primary sore happen to communicate. The glands when first involved are humid and engorged, but seldom extensively indurated.

If the periglandular tissue becomes inflamed, as it occasionally does, especially in the cervical region from the motion of the jaws and neck, it is difficult to identify the separate glands, which are obscured in a general tumefaction.

When suppuration takes place in the glands, the process is apt to be slower than it is in non-specific inflammations. Occasionally all the peripheral lymph-glands in the body are swollen simultaneously. This universal enlargement of the accessible lymphatic glands is of much service in confirming an otherwise doubtful diagnosis of past syphilitic infection. Those chiefly investigated are the epitrochlear gland and the members of the cervical chains.

The mucous patch is a lesion of the secondary period of syphilis. It is

found on the mucous membranes or the skin about the mouth or anus, especially where the skin becomes continuous with the mucous membrane. Such lesions may occur singly or several may appear at once. The patches have a fairly uniform oval or rounded contour; they are slightly raised above the surrounding healthy tissue; they are easily excoriated, but are not invariably ulcerated, although they usually are so. They then become moist, and they are covered by a grayish film. They are not commonly painful to the touch, and often escape observation by the patient; but in the mouth they are irritated by salt or pepper. When they involve the skin, there is cellular infiltration of the cutis and the papillæ are enlarged. In size they vary from that of a small pin's head to an inch or more in diameter, and several of them may coalesce into one large sore. There are more or less induration and thickening about their periphery. In females mucous patches occur more regularly than exanthems.

Ulceration may take place in the mucous membrane of the œsophagus, rectum, trachea, or bronchi. Sometimes the soft palate becomes so eroded as to entirely disappear, or a perforating ulcer may eat out its base and leave it suspended by two lateral bands. There may be similar destruction of the epiglottis and vocal cords.

The mucous patch heals over completely in from six to ten weeks, leaving a glistening cicatrix if it was deeply ulcerated; otherwise there is no trace left. Men who smoke are particularly apt to have these patches develop in the mouth, on account of the local irritation produced by hot tobacco-fumes. When situated between two opposing surfaces like the buttocks, where there is much friction, the patches may vegetate into condylomata.

Other lesions of the secondary period, including various cutaneous eruptions, inflammations of the eye, etc., will be briefly referred to in connection with the symptomatology of the period. For a more detailed account of them the reader must be referred to special works upon syphilis, dermatology, or ophthalmology.

The lesions which arise during the tertiary period may eventually affect any organ of the body. Their extent is often quite independent of the intensity of the primary sore or of the secondary symptoms and lesions. They embrace the formation of gummata, syphilides, and various degenerative and necrotic processes. They are not symmetrically situated, like the secondary lesions. They are amenable to treatment, but recur if the treatment be withheld. They are not accompanied by pyrexia or by cachexia, as a rule, although marked anæmia and a lack of mental and bodily energy may be observed.

When tertiary lesions develop early, they are explained by the excessive cellular proliferation, which chokes the lymphatic vessels, invades and thickens their walls, and prevents the proper removal of waste material (White).

Gummata are sometimes described as "gummy tumors," but they are essentially inflammatory products originating in the connective-tissue structures of membranes, viscera, skin, periosteum, etc.

Gummata are either isolated and distinctly circumscribed, or else they may be diffused throughout the affected tissue, infiltrating rather than supplanting it. To the unaided eye they appear as nodules, varying in size from a pin's head to a hen's egg, which are either soft or firm, of a gray color, and translucent or opaque. The inflammation may be quite acute, with considerable swelling and congestion of the affected region, but it is more often chronic.

The inflammatory products, consisting of small, irregular or round cells, with a soft gelatinous basement substance, may be diffusely spread throughout a viscus, but frequently they are collected in a limited area, forming a distinct mass or "gummy tumor," which either remains for some time without change or is absorbed, or more often it advances to the stage of cheesy degeneration, or it is converted into fibrous tissue or into a suppurating mass.

The gumma has no true capsule, but it may be enveloped in granulation and fibrous tissue, which contracts.

Much of the tissue-formation as observed in syphilis is very similar to that accompanying ordinary inflammatory action, but there is a common tendency for caseation to supervene and for contracting fibrous tissue to form cicatrices. As a rule, the lesions of the skin, mucous membranes, bones, and cartilages produce ulceration and extensive sloughing, but the deeper visceral lesions result in the formation of gummy, caseous, or calcareous masses. The gummata, when they do occur in the skin or mucous membranes, may be very destructive through resulting ulceration and extensive cicatrization. In this manner the tonsils, soft palate, and nose may be destroyed. Cicatrices of the mucous membranes may by contracting cause ultimate stricture. The traces of former gummata in the various viscera of the body are often visible in irregular, depressed, frequently star-shaped cicatrices.

The gummata may be slowly absorbed without appearing at the surface. In this case the skin is left thinned and sunken over the site of the gumma.

Syphilides.—The tertiary syphilides are polymorphic; that is, several varieties may be found simultaneously, as in the secondary period. They are apt to form in circular spots or crescents. They are especially prone to develop where there is local irritation or friction of the surface. They are not characterized by accompanying pyrexia. Many are of the papular type and seldom cicatrize under the use of mercury; others affect the tissues much more deeply than the skin lesions of the secondary stage, and ulcerate, leaving ugly depressed scars. They are usually fewer in number than the secondary syphilides, but are more apt to coalesce. When scaly the scales are seldom lustrous or very abundant. (For a detailed account of the great variety of tertiary syphilodermata the reader is referred to special works upon syphilis.) Partial loss of hair and teeth is of common occurrence.

Visceral Lesions.—The subjects of visceral or internal syphilis of the various organs, such as the brain, cranial nerves, and spinal cord, the liver, kidneys, spleen, and aneurisms of syphilitic origin, will be treated in proper detail in this work under the respective titles of the diseases of the various organs. These lesions are comparatively rare. In a series of 21,757 cases

of syphilis they were observed in 88. They consist principally of visceral gummata and cirrhoses, which result in more or less profound disturbance of function, give rise to pressure symptoms, and beget various secondary changes. Of the deep-seated organs most frequently affected by severe tertiary lesions, the brain and its membranes rank first. Of the abdominal viscera, the liver is oftenest involved, while the kidneys are included with rarity. The syphilitic process may go on to lardaceous or amyloid degeneration of these viscera.

Other tertiary lesions are destruction of the nasal cartilages and bones, and sinking in of the bridge of the nose, associated with intractable and very offensive ozæna.

Serpiginous ulcers of the skin and mucous surfaces, arterial sclerosis, inflammations of the cornea, iris, and retina, periostitis, and caries or necrosis of the bones of the skull or of the extremities,—all occur from time to time. Periosteal thickening in the form of nodes along the tibial crests is quite frequently noted. The tongue occasionally becomes infiltrated, hypertrophied, and deeply fissured upon the dorsum, or it is the site of gummy growth and ulceration.

Locomotor ataxia is very frequently though not always associated with a syphilitic history. Individual muscles may exhibit infiltration of the interstitial connective tissue and fatty degeneration of fibres.

Symptomatology.—*Primary Period.*—Three weeks is the ordinary period of incubation of a chancre. At the end of this time a more or less typical local lesion appears, which is almost immediately followed by swelling of the nearest lymphatic glands.

Secondary Period.—At the end of six weeks the active secondary period is announced by the development of moderate fever—102° or 104° F.—headache, malaise, lassitude, and possibly pains in the back and legs. A chill may precede the fever. At times these symptoms are so severe as to lead one to suspect the commencement of a zymotic disease. The temperature may rise to 105° F., and run an irregular course for a week or two, but the fever is usually of little moment, the temperature remaining at 101° or 103° F. There is angina with diffuse redness of the fauces and hard palate. Sometimes isolated small white spots are to be seen in connection with the hyperæmia. The tonsils may be swollen. A modified rash or roseola develops profusely upon the buttocks, trunks, and thighs, and there are frequently one or two papules upon the dorsum of the tongue. Similar papules may appear upon the scalp. They are small, hard, do not tend to ulcerate, and they are pathognomonic. Other forms of eruption may appear, but in general early cutaneous syphilides are characterized by symmetrical distribution, lack of pain or itching, rounded outline, and of a muscle-red or "coppery" hue.

The early eruptions are usually erythematous and papular, extensively distributed. The later-appearing varieties include vesicles, pustules, tubercles, condylomata, and squamous eruptions, psoriasis, lichen, etc., which are less diffuse, and which tend to become grouped together in localized areas. A roseola sometimes has been observed in the mouth. Several varieties of

syphilides may be simultaneously present. In women and, especially, in children the roseola may be very evanescent. It is not found possible to prevent the appearance of the secondary rash by any abortive treatment.

At the same time with the eruption a typical mucous patch appears somewhere within the mouth or at its angles, on the gums, tongue, or buccal mucous membrane, or on the skin. A common site for it is opposite the second molar tooth. There may be one or several of these patches, and they develop in succession at irregular and increasing intervals until the tertiary period.

Other common symptoms of this stage are laryngitis, with a red, dry, hyperæmic mucous membrane, iritis, and possibly retinitis. There is also alopecia, and the finger-nails sometimes become brittle. The hairs of the eyelids and eyebrows may fall off.

The ulcers, inflammations, and cutaneous syphilides are characterized by lack of pain and discomfort.

The symptoms and lesions of the secondary period may last for two or three or more months, and be followed by an interval of good health, which continues for several months or for many years before any tertiary lesions arise. In other cases the secondary symptoms or lesions continue for two years, or until those of the tertiary period succeed them. In many cases they last about a year.

It should be observed that not all cases present the symptoms above described with equal distinctness. In many instances the entire secondary stage is mild, or, while certain of the symptoms are prominent and severe, others are unnoticeable.

In a certain proportion of cases a syphilitic cachexia develops during the secondary period. It is characterized by the following features: The skin is muddy or sallow, the bowels are costive, the tongue is coated with a white fur, the breath is offensive, and there is more or less anæmia, with headache, palpitation, and lassitude.

The occurrence of tertiary symptoms and lesions is favored by lack of proper antisyphilitic treatment in the secondary period; by diatheses and cachexiæ, such as scrofula, tuberculosis, scurvy, etc.; by chronic alcoholism; and by conditions of want and misery which lower the general vitality and resisting power of the system.

Tertiary symptoms show a great predisposition to relapse. When the lesions occasion pain, as frequently is the case with periostitis, the pain is markedly worse at night. If there are cerebral lesions, the pain is frequently localized and confined to a circumscribed area on one side of the skull, which is sore and painful to the touch.

The encephalopathies may result in pressure symptoms, such as paralyses or convulsions, or in mental apathy or disturbances of the special senses.

Various symptoms will arise in connection with visceral lesions, but they will be dealt with under the several headings of the diseases of separate viscera, and do not require amplification here.

Course.—No other disease exhibits greater variations in course and variety of feature. In some cases the course is brief and the symptoms pass almost unnoticed, while in others one outbreak of symptoms succeeds another, yielding to no treatment, and involving in turn almost all the organs and tissues of the body. In cases of ordinary severity there is the greatest variety in the clinical picture presented. In some cases, even after a typical primary sore has occurred, the secondary symptoms are so mild that they are overlooked. In other instances the tertiary symptoms may first disclose the presence of the disease, and, very exceptionally, some of the tertiary symptoms have been observed in connection with, or before, the appearance of decided secondary manifestations.

When syphilis occurs in old age the healing processes are retarded, the chancre is apt to ulcerate extensively, the enlargement of the lymphatic glands is troublesome, the secondary syphilides are especially confluent, relapses are frequent, and the tertiary lesions are very prone to give rise to grave nervous symptoms.

Terminations.—Much argument has been expended on the questions of the self-limitation and the curability of syphilis. Gowers, who has especially studied the later manifestations of syphilis in the nervous system, is, contrary to the general belief, inclined to doubt the proofs of absolute cure. Others argue that the possibility of reinfection long after the development of active symptoms is in favor of the positive cure of the original disease, and, further, that syphilis is a self-limited disease, running its course untreated in about four years. Some syphilographers even claim that the lesions of the tertiary stage outlast the disease itself; that is, the activity of the disease is entirely expended, while the new growths which it occasioned are more permanent. On the other hand, treatment may remove all traces of local lesions, and yet the disease breaks out anew. For example, a syphilitic woman has been known to give birth, in turn, first to a stillborn syphilitic infant, secondly, while under treatment, to a healthy viable child, and finally to herself relapse when the treatment was withheld (Gowers).

The great majority of cases terminate favorably under proper treatment, and death is comparatively rare from syphilitic lesions. The most fatal forms are the advanced cases of gummata or other lesions of the nervous system, abscess of the liver in connection with bone lesions, and alterations in the arteries, resulting in the production of aneurism or occlusion.

Diagnosis.—In many cases the diagnosis of syphilis is sufficiently obvious from the distinctness of the lesions. In other instances, especially in obscure cases of the tertiary period, a very careful cross-examination of the patient fails to elicit any history of early infection, and a correct diagnosis is largely aided by the response to treatment. Caution should be observed in questioning some patients, especially married women, as great care should be exercised in avoiding interrogations which might occasion suspicion or extensive family discord. In such cases a diagnosis can often be obtained through indirect queries in regard to the existence of eruptions, alopecia, angina, swollen glands, etc.

The diagnosis of an extra-genital chancre is formed upon the general aspect of the sore, the induration at its base, with a tendency to form a scab, especially where a hairy surface retains the viscid secretion, and the enlargement of the nearest lymphatic glands, although the indolent course of the latter is not as pronounced as it is in genital syphilis. In extra-genital chancres, while the induration is commonly distinct, it is frequently absent when the site of the chancre is on the finger or tongue. Sometimes the induration is obscured by caustics or irritation.

The early diagnosis is often rendered difficult by the symptoms passing unnoticed by the patient, who stoutly denies their occurrence.

The true syphilitic chancre must be distinguished from the soft non-infectious chancre or "chancroid." The latter develops as a pustule which ulcerates in two or three days, forming a depressed irregular sore with undermined periphery. The base is not indurated, and there is a secretion of pus from the surface. The chancroid appears earlier than the true chancre: it bleeds more easily, and is somewhat painful on pressure. The ulcer can be inoculated upon healthy persons, and reinoculated on the same person, so that several such sores may appear together and coalesce. Finally, it is followed by no secondary symptoms. In these several respects it differs from the true syphilitic chancre, yet the two may occur simultaneously by a double infection and at the same point of inoculation. When there is grave doubt as to the real nature of the sore, the appearance of symmetrical, painless enlargement of the lymphatic glands in the groin confirms the diagnosis of syphilis.

A syphilitic roseola, accompanied by rise of temperature and prostration, is sometimes mistaken for measles. The presence of mucous patches, the history of inoculation, and a careful examination of the appearance and distribution of the rash, together with the absence of the catarrhal symptoms belonging to measles, will establish the diagnosis of syphilis.

When potassium iodide is prescribed in doses of a drachm in twenty-four hours without producing iodism, the chances are very strongly in favor of the syphilitic character of a lesion, yet personal idiosyncracy in regard to toleration of iodine occasionally prevents this test from being absolute.

When repeated abortions occur without other assignable cause, and especially if the fœtuses are macerated, syphilis may be strongly suspected in the mother if not in the father as well.

Sequelæ.—The former existence of syphilitic disease can often be recognized by symmetrical cicatrices on the extremities or face or in the mouth, periosteal nodes on the tibial crests, a sunken nose, absent teeth and hair, corneal cicatrices from old ulcers, irregularities of the pupil from iritis and adhesions, perforations of the hard or soft palate, deafness, etc.

Prognosis.—The prognosis is excellent for almost all cases coming early under treatment. It is made worse by chronic alcoholism, and is worse if the disease develop very rapidly and severely at the onset, which is rarely the case; and it is worse the further the disease has advanced without medical control.

Relation to Other Diseases.—Syphilis bears no definite relation to other diseases, but it is sometimes noted that syphilitic lesions are kept somewhat in abeyance while other diseases are in progress. When erysipelas occurs in connection with a syphilide, the latter may be actually improved when the acute inflammation has subsided.

Prophylaxis.—Syphilis is one of the diseases which theoretically might be completely eradicated, but long experience has taught that legislation can but partially control it. This question is too broad for discussion here, as it involves considerations such as the compulsory examination of prostitutes, soldiers, sailors, and wet-nurses; government license of houses of ill-fame or their abolition; and other allied problems.

Syphilis is always more powerful for transmission in its earlier development, and is generally believed to lose its infectious quality completely in the third stage, but not until then.

Treatment.—The treatment of syphilis is conveniently divided under the following headings:

I. Local; II. Specific; III. Tonic; IV. Hygienic.

Syphilis is a particularly satisfactory disease to treat, because a large majority of cases, even of those already presenting extensive lesions, respond promptly to the measures employed.

A certain proportion of cases never develop symptoms of sufficient severity to lead the patient to seek counsel, and time alone is the healing agent. Not infrequently the general health of syphilitic subjects is very good, and it may be so good as to lead them to neglect obtaining treatment for really grave lesions. Other patients are always suffering from one ailment or another.

I. *The Local Treatment.*—The local treatment of the initial lesion is of little avail if it has gained decided headway. Aseptic or antiseptic applications are mainly valuable for purposes of cleanliness, and not for any abortive action. If the chancre is seen very early and if it is very small, it may be thoroughly cauterized with nitric acid or completely excised, but this eradication offers only an indefinite hope of preventing the development of secondary symptoms, and of late years it has been very generally abandoned. If imperfectly done it makes the original sore worse. It is important to keep the chancre clean and free from irritation.

Some authorities claim good results from the local application of a mercurial ointment to the chancre.

The local treatment of the mucous patches comprises cauterization by nitrate of mercury or other caustic, and cleanliness, secured by application of 1 : 1000 corrosive-sublimate solution. Condylomata may be washed with salt-solution and then dusted with calomel.

For the local treatment of the ozæna, and affections of skin, bone, eye, and ear, the reader must be referred to the special articles upon the diseases of the organs involved and to works on surgery.

II. *Specific Treatment.*—The two drugs, potassium iodide and mercury in its various preparations, are specific agents against syphilis, and are prac-

tically the only remedies which control it. Their use in syphilis is therefore referred to as "specific treatment," and their employment in combination is often described as "mixed treatment." Remedial measures are capable of diminishing the intensity and of shortening the duration of many lesions of syphilis, and of greatly reducing the chances of the infection of healthy people by the syphilitic.

Mercury has been employed for this purpose for nearly four hundred years. Its mode of action is unknown, but it is held by those who endorse the germ-theory of syphilis that it kills the bacillus. It may, however, only render it inert, or it may act by destroying or antagonizing the leucomaïnes formed through the agency of the germ, or by altering the tissues or "soil" in which the germ naturally thrives. At present these questions must remain purely speculative until further experimentation and observation throw new light upon this important topic.

There are different views in regard to the method of administering the two remedies in the several stages of the disease. Some give potassium iodide alone; others give it with mercury to favor the activity of the latter. It is the generally accepted belief that the greatest value of mercury is in the earlier course of the disease, while potassium iodide is more useful in the later; but there is a growing tendency at present to continue the mercurial treatment into the final stage of the disease. We have ceased to fear the continued use of mercury since its action and modes of administration have become better understood.

Treatment, while it does not altogether prevent the appearance of secondary or even tertiary lesions, exercises a strong control over their extension. There are some syphilographers of large experience, like Hutchinson, who maintain that syphilis can be aborted by treatment to such a degree as to altogether suppress the secondary stage and hasten the disappearance of the primary lesion.

Mercury is of no avail while the disease is still localized, but it antagonizes such constitutional symptoms as may arise in the early stages. The exact mode of its action is not known.

Mercury may be employed in a great variety of preparations, and due regard must be paid to the circumstances and condition of the patient, as well as to the urgency of the symptoms. Greasy and malodorous external applications, besides being disagreeable, expose the patient to detection. If, however, the symptoms are severe or if eruptions appear upon the face or head, it is necessary to obtain prompt action of the drug, and this is best done by inunctions of one-half to one drachm of the mercurial ointment or of an oleate or albuminate of mercury or of a 1 per cent. corrosive-sublimate solution, or else by hypodermic injection of one of the more soluble preparations of mercury, such as the salicylate, which have recently become popular with many practitioners.

Syphilides are sometimes, but not always, more benefited by the local than by the internal use of mercury.

Fumigations are also employed. For more continued use the protiodide of

mercury in doses of one-fifth of a grain three times daily, or the bichloride or biniodide in doses of one-fortieth to one-twentieth of a grain may be given, preferably in solution, either alone or in combination with ten grains of potassium iodide. Some clinicians have a decided preference for the gray powder, hydrargyrum cum creta, in doses of one grain, continued for six months at a time. It is often advisable to combine it temporarily with a little opium if any symptoms of intestinal irritation appear.

As a prophylactic measure against stomatitis while taking a prolonged course of mercurial treatment, it is necessary to pay special attention to cleansing the teeth, and to gargle the throat and rinse the mouth after each meal with a 5 per cent. solution of chlorate of potassium (Eichhorst). If the mouth becomes sore, a soothing gargle of mucilage or flaxseed tea, with a drachm of potassium chlorate to the pint or with listerine properly diluted, must be used. Irritant or hot food should be withheld, and smoking must be forbidden. Laxatives are also indicated.

Should any of the characteristic symptoms arise—such as tenderness or swelling of the gums, fœtor of the breath, slight salivation, or diarrhœa and abdominal pains—it is an indication that the physiological limit of the mercury has been passed, and the drug must be promptly withheld until the unfavorable symptoms subside. Any diet which is liable to over-stimulate the intestines and cause diarrhœa should be avoided.

It is wrong to give mercury when the diagnosis is doubtful, and useless to give it in the first few weeks after inoculation; but it should certainly be prescribed as soon as the prodromata of the secondary period appear. It should be continued for at least a year, observing the above precautions, and with occasional brief intermissions. It is desirable to follow this medication with a year or two longer of "mixed treatment," the duration of the treatment being regulated somewhat by the intensity and variety of the earlier symptoms. It is well to combine some preparation of iron with the mercurial treatment for its tonic effect and to counteract anæmia. If the symptoms become suddenly urgent at any time, a speedy effect may be obtained by the internal use of calomel in doses of one-tenth of a grain every hour for a few doses.

The iodides of potassium and sodium are pre-eminently valuable in the third period of syphilis, and the iodide treatment must be continued for at least two years in most cases, or, better, for three years, to render the patient tolerably secure from the outbreak of fresh manifestations of the disease. An ordinary dose for continued use as a preventative of new symptoms is ten grains thrice daily, given in milk or in the compound syrup of sarsaparilla, which conceals the peculiar taste entirely. The drug should always be well diluted. When decided lesions and symptoms demand it, the daily quantity of the iodide is gradually increased by adding five grains of the iodide or five drops of a saturated aqueous solution to each dose, until a half-drachm or a drachm is given thrice daily. In exceptional cases, especially where pressure-symptoms are produced by gummata of the important viscera or of the

central nervous system, or when any serious complications supervene, such as convulsions or paralyses, it is of vital importance to saturate the system with the remedy as rapidly as possible; and it may be necessary to administer half an ounce, or exceptionally an ounce, of the iodide in the course of twenty-four hours. As a rule, syphilitic patients show great tolerance of the drug, and in an urgent case one is justified in giving very large doses immediately, in order to save life. When the drug is pushed to its full extent care should be exercised to keep the bowels open with Rochelle salts or other saline.

Indications of iodism are usually first observed in a papular or pustular eruption which appears upon the face, shoulders, or other portions of the body, and which often resembles acne. There may be also gastro-intestinal irritation, coryza, and œdema of the eyelids and lips. When these symptoms appear the drug must be reduced in quantity, but it need not usually be entirely suspended.

The urine must be frequently examined in order to be certain that excessive elimination of iodine is not exciting albuminuria. When the stomach rebels against large doses a good deal may be absorbed by the rectum.

When iodism first appears, changing the preparation from potassium iodide to the sodium salt will cause less irritation to be produced.

Occasionally decided mental depression attends the use of even moderate doses of potassium iodide, but this effect is not serious, and it passes away with the temporary withholding of the drug.

In the tertiary period iodides are either given alone or in combination with mercury, the latter method being quite in vogue at present. A convenient formula for this mixed treatment is—

℞. Hydrarg. bichlor., gr. $\frac{1}{30}$;
Potass. iodi., gr. v;
Tinct. cardamom. co.,
Tinct. gentian. co., āā. ʒss.
M. Quantity for one dose.

Or,

℞. Hydrarg. biniodi., gr. $\frac{1}{30}$;
Potass. iodi., gr. viij;
Syrupi sarsapar. co.,
Aquæ, āā. ʒss.
M. Quantity for one dose.

The medicine should be well diluted in water, or half a tumblerful of water should be taken after each dose.

In a certain proportion of cases repeated courses of treatment, interrupted once in six or eight weeks for an interval of a week or ten days, secure better results than continued doses, to which the system becomes inured.

The specific treatment of syphilis should be continued in the above manner for at least a year after all symptoms have gone.

III. and IV. *Tonic and Hygienic Treatment.*—The specific treatment of syphilis is of little avail without the use of tonics and proper hygienic regulations. The tonic treatment consists in the administration of phosphates, cinchona, arsenic, strychnine, or cod-liver oil, and iron should be given in connection with one or other of these remedies.

The *Hygienic Treatment* includes the careful supervision of the patient's daily habits of life. Abundant fresh air, frequent bathing, warm clothing, duly-regulated exercise and rest, and a nourishing diet are of great importance. It is almost useless to attempt the specific treatment of syphilis without accompanying it with an appropriate hygienic regimen. Under the latter conditions a patient who has been upon a protracted course of the iodide without benefit will often show very rapid improvement.

Of course indulgence in sexual intercourse should be forbidden, at least until the third stage of the disease is reached, not only on the patient's own account, but to prevent inoculation of others, and the use of alcohol should be greatly restricted, and, if possible, interdicted entirely. The patient should be reassured as to the probable favorable termination of his symptoms, and made to appreciate the importance of a strict compliance with the rules laid down for him.

Special directions in regard to diet should be given in order to keep digestion at its best. The dietary need not be greatly restricted, but it should be plain and wholesome. Fruits and the coarser cereals are useful on account of their laxative effect, for patients are much less apt to suffer from iodism if the digestion be normal and the bowels freely opened.

Summary of Treatment.—As soon as the secondary symptoms appear the patient should be put upon a daily mercurial treatment, to be continued for a year and a half. The dose should be moderate, to avoid salivation. It must be at once discontinued should salivation appear. On the other hand, if any new symptoms develop it may be temporarily increased. The iodide of potassium may be added at the end of the first year, and it should be continued for eighteen months longer. The tonic and hygienic treatment must be begun early, and persisted in while any symptoms remain.

After two and a half, or better, three, years of such treatment, and if no symptoms return within six months or a year after its discontinuance, the patient may be regarded as cured, and he may marry without endangering either mother or offspring.

HEREDITARY SYPHILIS.

Definition.—Hereditary or congenital syphilis is that variety of the disease which is transmitted from one or both parents to their offspring. It exhibits great variety in its manifestations, but the only essential difference in symptoms or course from acquired syphilis is the absence of the stage of chancre. It is usually "conceptional"—*i. e.* it is transmitted from either one

or both parents at the time of conception, the virus having affected the ovum or the spermatozoon. It may, however, be transmitted to the embryo from the blood of the mother at any period of placental circulation, and in this case the disease is apt to be less severe.

Synonyms.—Erbsyphilis (Ger.); Syphilis hereditaria.

Etiology.—The following statements in regard to hereditary syphilis have been attested by numerous reported cases, and are worthy of note.

When the Father Only is Syphilitic.—If actively syphilitic—*i. e.* with primary or secondary lesions—the father usually transmits the disease to the child, but no definite relation exists between the severity of the disease in father and child.

This form of infection is the least injurious to the child. The father may infect the mother through the circulation of the child *in utero*. This latter statement has been denied by some syphilographers; but whether the mother shows active syphilitic symptoms or not, it is believed by many observers that a syphilitic infant cannot infect its own mother after birth, even though it virulently inoculates a healthy wet-nurse. This is known as "Colles's law;" and even if the mother does not develop extensive lesions herself, it indicates that she has been rendered immune to any external inoculation from her offspring. Eichhorst strenuously denies the validity of this law, and says that he has seen mothers infected by their syphilitic infants after birth through abrasions of the nipple, etc. Such a mother becomes anæmic, and is apt to have more or less glandular enlargement and periosteal inflammation.

Many more children are infected by fathers than by mothers, because more males have syphilis, and not because the disease is more readily transmitted by males. If the mother should have the disease, she is quite as apt to transmit it as is the father.

When the Mother Only is Syphilitic.—If the mother be syphilitic prior to conception, she infects the ovule, or she may infect the embryo at a later period. In such cases the disease is often more virulent in the infant than when inherited from the father alone.

The infant has the best chance of escape when both parents were healthy at the date of conception, but the mother subsequently acquires syphilis a few months before giving birth to the child. Under these circumstances it is rare for the child to be affected, and the later the mother contracts the disease the better chance the child has of escaping.

It is possible for syphilis in the mother to be so modified by treatment that the child escapes entirely. A child is not infected during labor by passing over a vaginal chancre or mucous patch, either because it is already protected by intra-uterine inoculation or by its own vernix caseosa.

Abortion occurs in approximately one-third of all syphilitic women (Parrot), and it is especially prone to occur while secondary symptoms are actively developing in the mother. It commonly takes place at the third or fourth month, and the fœtus is frequently found macerated. If miscarriage occurs after the period of viability, the child is apt to be born dead. This may also

occur if the mother were originally healthy, the child having been infected through the father. After the birth of a syphilitic child the placenta is found diseased by proliferation of cells in the villi, extravasations of blood, and compression of the vessels by new cells and connective tissue (Fraenkel). The whole organ is enlarged, and it may contain gummy or calcareous nodules. Vascular obliteration is chiefly responsible for the death of the fœtus by cutting off its supply of nourishment. The umbilical vessels are found to have thickened walls, and there may be thrombosis of the umbilical vein.

When Both Parents are Syphilitic.—It is asserted that both parents may have active syphilis and yet beget a healthy child. This is certainly a rare occurrence, but it may transpire, even with secondary lesions, provided both parents have been for some time under treatment.

On the other hand, both parents may have a very mild type of syphilis, and yet transmit a very severe form of the disease to the child. If the father is syphilitic, and the mother first becomes so in the later months of pregnancy, the hereditary syphilis in the child will probably be intensified (Neumann). Both parents are less liable to beget children who are syphilitic after the disease is a year old, but Fournier collected notes of 60 cases of hereditary syphilis where the disease had existed in one or both parents for more than six years. He recently published statistics of 500 syphilitic families in which there were 1127 pregnancies. Of these, 600 resulted in the birth of healthy children, but 46 per cent. ended in abortion, early fœtal death, or syphilitic infection. The infant mortality when inherited from the father alone was 28 per cent.; from the mother alone, 60 per cent.; from both parents, 68 per cent. Le Pileur places the mortality even higher, and declares that not over 7 per cent. of the children of syphilitic mothers outlive the disease.

Parents still capable of begetting syphilitic children are said to be in a condition of "syphilization."

Parents having tertiary syphilis do not beget children who are actively syphilitic, but they are weak and atrophic. Among such children are sometimes found cases of chorea, epilepsy, hydrocephalus, and idiocy.

Curious anomalies are sometimes observed among the children of actively syphilitic parents. Thus Hutchinson has reported a case of birth of twins, one of whom was syphilitic and the other not.

Morbid Processes in General.—Hereditary syphilis may develop during embryonic life, or it may remain latent for a varying period after birth, even up to twenty years. In the great majority of cases it appears before the child is three months old. In the embryo and infant it occasions cachexia, dystrophies, malformations, and predisposition to various morbid changes (Fournier).

Considerable doubt exists as to whether these malconditions can be transmitted to the third generation without fresh infection.

Among the dystrophies are slow general growth and retarded puberty and virility, so that the subject still appears like a child when eighteen or twenty years old, with a pasty complexion, scanty hair, irregular thickenings of the

skull, periosteal nodes on the tibiæ and elsewhere, keratitis, and serrated incisor teeth.

Among the malformations which are found to accompany congenital syphilis are hare-lip, spina bifida, hydrocephalus, club-foot, and various herniæ.

Among the morbid predispositions there is a tendency to a variety of neurotic affections and to readily acquired convulsions.

Hereditary syphilis, although not at all identical with them, is often associated with scrofula, rachitis, croup, or tuberculosis.

Morbid Anatomy.—The morbid anatomy of hereditary syphilis does not differ essentially from that of the acquired form. The pathology of the inflammations, mucous lesions, and gummata is identical.

The mucous lesions which are most frequently observed are hyperæmia and papillary infiltration, labial fissures, mucous plaques or patches on the inner buccal surface, and on the gums, tongue, nose, and genitalia. Large phagedænic or gangrenous ulcers may invade the pharynx or larynx. These patches may extend and leave extensive cicatrices. Inflammation may spread along the Eustachian tube, and involve the middle ear in a purulent otitis, resulting oftentimes in neuroses and permanent deafness. In severe coryza, beginning with hyperæmia and excessive secretion from the Schneiderian membrane, thick crusts form, with necrosis of tissue beneath and ulceration. The inflammation and erosion finally involve the bones of the nasal septum, producing a sinking in of the nose, which becomes deformed for life.

The cutaneous lesions are polymorphous syphilides, but chiefly papular. They are either bullous (pemphigus neonatorum) or erosive papules and pustules, or psoriasis, or simple roseola of a coppery hue. There are also ulcers of various sizes, moist or encrusted, and condylomata with flattened encrusted surfaces and a fœtid discharge. The only characteristic syphiloderm of infantile or hereditary syphilis is pemphigus, which in its specific form has a papular base, such as occurs in other eruptions of the secondary stage. The blebs are either transparent or oftener are distended by brownish or even bloody fluid. They cover an area of inflamed red skin, and are surrounded by a faint areola. A favorite site for them is on the palms and soles.

The various syphilodermata are to be sought especially upon the genitalia and the periphery of the anus and mouth, and on the scalp. The papules and condylomata are apt to occur in the deeper flexures of the skin of the neck and joints, where there is more or less irritation from opposing surfaces. Within a few weeks after birth a simple roseola, with irregular round or oval macules, may appear, commencing on the abdomen, and later spreading over the body and limbs. It is dry at first, and fades on pressure; later it acquires a permanent darker, coppery hue. It may become moist, or scaly where the skin is thick, and it forms papules with cellular infiltration.

The liver is permeated with fibro-plastic material and embryonic cells. The capillaries are occluded or compressed, and the whole organ is large, hard, yellow, and anæmic. There is interstitial sclerosis, with collections of round lymph-cells resembling microscopic gummata (Cornil).

The pancreas may undergo fatty degeneration. There may be purulent degeneration of the thymus gland, but this is not typical. The lymphatic glands may become more or less enlarged.

The spleen is often enlarged by simple hyperæmia or there is hyperplasia.

In the nervous system lesions of the meninges and blood-vessels predominate and are very fatal. They may produce hemiplegia, paralyses, convulsions, etc. There may be cerebral gummata. These lesions occur at any age after the first few months.

There is frequently corneal imflammation, with opacity or interstitial keratitis and photophobia.

The teeth exhibit characteristic changes. There are vertical single notches at the edges of the upper middle incisors. These teeth are short and narrow. (See Fig. 23). The notches belong to the second dentition, and are said by

FIG. 23.

Notched Central Incisor Teeth of Hereditary Syphilis (Hutchinson).

Hutchinson to be pathognomonic, though various other writers attach much less importance to this condition. The notches are usually attributed to the influence of stomatitis upon the young growing teeth.

The teeth of both the first and second dentition are very irregular in size, development, and position, and they decay easily, for the enamel is very soft.

Tertiary Lesions.—Among the gravest of the tertiary lesions are inflammations of the bones and joints and gummata. The lesions may be congenital or develop during early childhood. There may be gummy infiltration of the phalangeal and other joints. There are enlargement and discoloration about the joints and effusion into their cavities.

The bones which are very frequently involved are those of the forearm and leg, the humerus and femur, clavicle and sternum. Other favorite sites are the bones of the nose and skull, and the cartilages of the larynx and trachea are sometimes affected. Extensive cellular proliferation commences where the bones are actively growing, especially in the long bones above mentioned, between the shaft and epiphiseal end. The normal process of ossification is altered and retarded. By pressure the new cells occasion degeneration and necrosis. Osteo-chondritis and osteo-myelitis ensue. Pus may burrow outward beneath the periosteum. There may be an excessive deposit of lime salts, which encrusts the cartilage and projects into its substance. Granulation tissue intervenes between this deposit and the shaft of the bone, and the shaft itself may be thickened by a growth of new bone on the outer surface. The epiphyses and diaphyses of the long bones may become separated, giving crepitus, and spontaneous fracture of the shaft has been known to ensue.

Periostitis occurs over many bones, especially where they have the thinnest

covering of soft parts, and are consequently more liable to injury, like the sternum and tibia.

The more extensive lesions of the bones and joints cause deformities and loss of ability to move the limbs. Severe bone disease will cause the death of the infant through septic infection or exhaustion.

The gummata of infantile syphilis are not often developed at birth, and they may not appear before the child is eight or ten years of age. They are frequently found in the liver in connection with an interstitial hepatitis, which causes enlargement of that organ, and sometimes ascites. A few gummy nodules may invade the walls of the pulmonary vessels or bronchioles even in the fœtus.

The disease shows but two periods, the chancre of the first stage of the acquired affection being, of course absent in the hereditary form. In consequence of this we do not find the local enlargement of the lymphatic glands, although a universal adenopathy is often to be discovered later.

The secondary period lasts for about a year or a year and a half, either commencing with birth or, more frequently, within four or six weeks after birth.

The tertiary period may, in bad cases, overlap the secondary, and develop very early in the history of the disease as compared with acquired syphilis. The gummata in such instances appear with the secondary eruptions. In other cases there is an intermediate period of freedom from all symptoms, lasting from the termination of the secondary stage until the second dentition or puberty.

Symptomatology.—A large number of infants with hereditary syphilis appear healthy when born, and do not develop any symptoms for several weeks. Even when symptoms or lesions appear, they are not always recognized as belonging to syphilitic disease. The chancre being absent, the disease commences its manifestations with the symptoms which correspond with the secondary stage of the acquired form. There is an erythemato-papular eruption or a simple roseola on the buttocks and about the genitalia, which may spread to other regions of the body. In a typical case there is obstinate coryza with a watery nasal discharge and snuffles, and roseola; the voice is hoarse, and the cry is of a peculiarly harsh and irritating *timbre*. This is due to the presence of laryngeal hyperæmia or to mucous patches. When the coryza is severe the nasal secretion becomes very thick, and dries in scabs. The child will then be unable to suckle, because, the nasal passages being obstructed, the mouth must be constantly employed in breathing, and it cannot be closed long enough for the child to draw any milk from the breast. Such infants may actually starve to death unless fed by a spoon.

The areas of splenic and hepatic dulness may be somewhat enlarged.

The skin is poorly nourished, muddy, dry, inelastic, and flabby; the hair is thin and patches of alopecia appear; the finger-nails may ulcerate; and the facial expression is curiously old and wrinkled, as in marasmus. The child is dull and listless. The various syphilides of the secondary period make their appearance. The alimentary canal is irritable, and there is impaired

secretion and absorption. There are occasional vomiting and diarrhœa. More or less bronchial catarrh is commonly present.

On inspection of the cavities of the mouth, pharynx, and nose mucous patches may be found.

Groups of lymphatic glands, especially those about the elbow joint, may be found enlarged.

Later in the tertiary period the lesions of the bones and joints appear. There are inflammation and opacity of the cornea, interstitial keratitis, and photophobia, and, somewhat rarely, iritis. There may be optic neuritis or retinitis. Middle-ear catarrh occurs in some cases, which may lead to perforation of the tympanum or purulent inflammation of the mastoid cells.

Sequelæ.—Hereditary syphilis often leaves permanent marks upon the adult in the form of notched teeth, scanty hair, coarse skin, and radiating cicatrices at the corners of the mouth and elsewhere. There may be permanent deformities produced by bone and joint lesions, impairment of vision, deafness, and neuroses or paralyses.

Diagnosis.—The diagnosis will depend upon an accurate history of parental syphilis and upon the appearance of the child. When the infant is born with well-developed syphilitic lesions of the skin, joints, bones, etc., the diagnosis is readily made; but many children present obscure or slight symptoms and lesions which may be confounded with other affections. Others, again, show no symptoms of any kind until two or three months after birth, and such children appear healthy and normal in every respect.

The diagnosis must often be based on the conjunction of several symptoms rather than upon any one. Such symptoms are the coryza, hoarse voice and cry, a prematurely old expression, flabby skin and muscles, and the presence of papules, pustules, or bullæ about the mouth, scalp, anus and genitalia, or on the body.

The mucous patches may be confounded with stomatitis. In simple stomatitis the mucous patches cover a larger surface and occur more often in the sulcus between gum and buccal mucous membrane. The exudate is serous, and vesicles form which are not present in the syphilitic patch. In parasitic stomatitis there are far more inflammatory action and swelling than in syphilis.

The syphilitic affections of the bones are to be distinguished from rhachitis by the fact that in the latter there are symmetrical enlargement of the epiphyses, slow closure of the fontanelles, bending of the shafts of long bones, and a lack of involvement of other structures. Rhachitic changes in the bones are rare in the first half year, and in syphilis there are usually cutaneous and other symptoms. Besides, there is the history of the case, which will throw much light upon it if thoroughly investigated.

Prognosis.—About one-third of all syphilitic infants are stillborn. For the remainder the prognosis depends largely upon the time of appearance of the early lesions.

Children born with syphilitic eruptions seldom survive the first year.

Those in whom the disease develops shortly after birth may live for a few years with enfeebled constitutions and then die, or they may live to become adults. About one-fourth of the children born syphilitic die within the first half-year. The majority of infants in whom the secondary lesions are well developed do not outlive the secondary period.

In general, the later the symptoms develop the better the chance of ultimate recovery. If no symptoms develop within eight or nine months after birth, the child may escape the second stage and pass on to the third, or it may escape serious lesions altogether.

If, on the other hand, gummata, periosteal nodes, or other tertiary symptoms appear within the first year, the prognosis for ultimate recovery is extremely bad.

Treatment.—The treatment of hereditary syphilis should be commenced at once through the mother by giving her mercury and iodide of potassium, and continued with the child after birth. The transmission of syphilis is wonderfully controlled by the prolonged treatment of the parents, so that the mortality may be reduced from over 60 per cent. to 2 or 3 per cent. No syphilitic person should marry or attempt to beget children within from three to four years after primary inoculation, and not even within this time unless he has been under continuous observation and treatment, although the great majority of syphilitic parents do not transmit the disease after the secondary stage is over. After the lapse of three years, and if no lesions have appeared within the previous six months, it is regarded as safe for a man to marry without endangering either mother or offspring.

A syphilitic child must on no account be allowed to nurse from a healthy woman, as the latter is certain to become infected through a fissured nipple or in one of the numerous methods of extra-genital contamination.

The treatment of the infant is governed by the same principles as those of the treatment of the adult. Mercury may be given internally in the form of hydrargyrum cum creta or as the biniodide or bichloride, the former being preferred by many. A method early recommended by Brodie consists in spreading a little mercurial ointment, in the strength of a drachm to the ounce of emollient, upon a flannel bandage which the child wears over the abdomen; or an ointment of one part of red precipitate of mercury to 100 parts of lanolin may be used. In this manner enough of the drug is readily absorbed through the delicate skin of the infant.

Mercury given by the mouth to young infants sometimes produces intestinal colic, irritation, and purging. In such cases inunctions are preferable. On the other hand, if there are cutaneous eruptions or if the skin be too tender, the inunctions are not desirable, and one of the milder preparations of mercury must be selected for internal use. Should any indication of stomatitis appear, the child's mouth should be cleansed after each nursing by a rag dipped in a 2 per cent. solution of chlorate of potassium.

When the syphilodermata are extensive and refractory, baths of corrosive sublimate are sometimes useful, provided there be no abraded surface to admit

of too rapid absorption. For such baths Wiederhofer advises the use of two or three grains of corrosive sublimate, with fifteen grains of ammonium chloride to the half pint of distilled water, to be added to the child's bath-tub full of warm water. Only a wooden or earthen tub should be employed. Of course care must be taken to protect the mouth and eyes while the child is being bathed.

As soon as any evidence of tertiary disease presents, the iodide of potassium must be added to the mercury in doses of one-half to two grains three times a day; and this mixed treatment should be continued even longer than in the adult before the child is secure from further outbreaks of the disease. It may seem advisable to discontinue it from time to time for a few weeks, but its administration should be kept up at intervals until puberty. The syrup of Gibert is a serviceable combination for children. It is modified in various ways, but in the following formula it is well borne:

℞. Hydrarg. biniodi., gr. ss;
Potassii iodi., ʒij;
Syrupi zingiberis vel glycyrrhizæ,
M. Aquæ destil., āā. fʒij.
Sig. Dose gtt. v–x in water for an infant of six months.

It is to be remembered that a certain amount of potassium iodide can pass into the nursing infant from its mother's milk when she is taking the drug in considerable quantity.

Syphilitic infants should be bathed often and have their diapers frequently changed to avoid irritation of the skin. If the cutaneous eruptions are moist, they should be treated with mild astringents or with antiseptic dusting powders, and be protected from the air and from abrasion by absorbent cotton. Mucous patches will sometimes require cauterization, and great care should be taken to keep their surfaces clean and to prevent their virulent secretions from infecting others.

Syphilitic children are feeble and do not thrive upon the bottle, so that, if the mother cannot nurse her own infant, a syphilitic wet-nurse will be better for it.

Infected infants should be very carefully isolated from healthy children, who might readily become infected by caressing them or by using the same nursing-bottle, spoons, or toys which have been in the mouth.

The tonic treatment should be carried out as in the case of adults. The saccharated carbonate of iron in half-grain doses is well borne by infants, and cod-liver oil may be added.

LEPROSY.

By WILLIAM PEPPER.

Definition.—Leprosy is a chronic, infectious, and contagious disease, characterized anatomically by tubercular nodules of the skin and mucous membranes and changes in the nerves, and clinically by various tropho-neurotic manifestations with mutilations.

Nomenclature.—The disease is also called lepra, and was formerly known as elephantiasis Græcorum.

Historical and Geographical.—The frequent references to leprosy in the Bible give a peculiar historical interest to this disease, though doubtless a variety of other affections were confounded with it. It was known from the earliest times in India, Egypt, Palestine, and Arabia, and also, though the records are less satisfactory, in China. The first appearance in Europe was about a century before the Christian era, when Greek writers first mention its occurrence. It invaded Italy a few centuries later, and Galen and Aretæus gave excellent accounts of its characters. During the Middle Ages it advanced throughout Europe along the lines of invasion of the Roman armies, and assumed the characters of an universal scourge. As a result of rigid enforcement of isolation the disease declined during the thirteenth and fourteenth centuries, and at the end of the latter had disappeared from England and the greater part of continental Europe.

At the present time the principal centres of leprosy are India, China, and the Sandwich Islands, the last being of particular interest from the fact that the disease took root in that locality within the last fifty or sixty years. In Europe, Norway, and the Baltic provinces of Russia are its important foci. In our own hemisphere leprosy prevails extensively in Mexico, parts of Central and South America, certain of the West Indian islands, and to a less extent in some of the Gulf States of this country. The last is true especially of Louisiana, and in 1891 Blanc estimated that there were in New Orleans alone fully 75 cases. Parts of the province of New Brunswick are also affected. On the Pacific coast the disease is frequently seen among the Chinese.

Etiology.—Leprosy is peculiarly a disease of young persons, the great majority of cases occurring from the fifteenth to the thirtieth year. It is slightly more common among men. Social condition plays a part in the etiology in so far as squalor and overcrowding expose to contagion, but all classes of society are susceptible. Heredity certainly has some influence, as several well-authenticated congenital cases definitely prove. The theory of

Galen, lately advocated by Hutchinson, that a diet of decayed fish leads to the disease, is not substantiated by recent experience.

The *specific cause* is the *bacillus leprœ*, discovered by Hansen in 1874. This organism resembles very closely the tubercle bacillus, but may be distinguished by its staining properties, its shape, growth, and distribution. It occurs abundantly in the leprous tubercles, and has been found in the blood. It has been successfully cultivated, but inoculation of animals has failed to produce the disease. It has, however, been established that inoculation of man with parts of the growths will cause the disease.

The contagiousness of leprosy cannot be doubted, though this always requires direct inoculation, as in the case of syphilis. Sexual congress has been indicated as the method of transmission in the majority of cases (Morrow). Instances of inoculation through vaccination are also recorded.

Morbid Anatomy.—The bacillus finds two favorite seats—the skin and the nerves. In the former it gives rise to the characteristic leprous tubercles, in every respect similar to the other granulomata. Microscopically, these tubercles contain lymphoid, epithelioid, and giant cells, and numerous bacilli between or within the cells. Eventually these tubercles soften and discharge thick puriform material, or in rare cases more or less complete organization occurs and limits the further progress of the disease. In the nerves extensive neuritis marks the invasion of the bacilli. In the late stages of the disease leprous new growths may be found in the internal organs, especially the spleen and liver.

Clinical History.—Two distinct types are recognized: the tubercular and the anæsthetic or nerve leprosy, and in some cases a combination of the two occurs.

Tubercular Leprosy.—The period of incubation is indefinite, some cases having followed infection by a few months, others by as much as twenty years. A prodromal stage of excessive sweating, mild, irregular fever, and lassitude has been described, but is rarely prominent. The onset of the disease is marked by the appearance of erythematous patches, slightly elevated and hyperæsthetic. These, after a time, disappear, but return with greater distinctness, and may then persist for a long time. The color of the patches may be a livid red, or there may be only a diffuse mottling. In all cases, when persistent, they become darker from deposition of pigment.

The tubercles occur in the skin and mucous membranes. They are particularly common about the face, to which they give the heavy features designated by the name *leontiasis*. Other localities are the ears, extremities, mammary glands, and scrotum, but all parts, excepting the scalp, may be involved. The palms and soles, however, are rarely involved. Of mucous surfaces the mouth, throat, larynx, and conjunctivæ are most frequently affected. The nodules vary in size from a pea to a large nut, or they may run together, producing extensive new growths. The skin over the tubercle is tense and glistening, especially at certain times when the redness, heat, and tenderness indicate inflammatory reaction. The hairs of the affected areas drop out, and in

leprous countries disappearance of the eyebrows is regarded a significant symptom. Subsequently the tubercles soften and ulcerate, discharging thick yellowish or brownish puriform matter, which forms thick crusts and contributes to the repulsive appearance of the terminal stages. The ulceration may extend deeply, even involving the bone. This is often seen in the nose, where the septum becomes destroyed, with a falling in of the bridge of the nose. In the eye the tuberculous and ulcerating processes cause extensive destruction, until the globe becomes a shapeless mass. In certain cases, on the other hand, ulceration does not occur, but by organization of connective tissue destruction is checked, and possibly the whole progress of the disease is arrested.

Anæsthetic or Nerve Leprosy.—In this form the characteristic symptoms are the spots, the anæsthesia, bullæ, trophic alterations, and mutilation. No definite order of occurrence can be assigned to the different symptoms.

The spots appear insidiously or sometimes acutely with fever. They affect particularly the back, buttocks, knees, arms, and face, and vary in size from that of a small coin to extensive areas. At first they are often erythematous, slightly elevated, and hyperæsthetic; later they become pigmented or pigmentless. Usually the centre is light-colored, the periphery dark, but the whole area may be white.

The characteristic feature of the spots is the altered sensibility. At first hyperæsthetic, they soon become anæsthetic, and retain this as a pathognomonic feature. In addition to anæsthesia, absolute suppression of sweat from the surface of the spots has been pointed out by Manson as a symptom of great significance.

The bullæ of nerve leprosy occur at any stage of the case, and may continue to appear for a long period. They come out suddenly, last a few hours, and then break, leaving a red spot, which may persist as a chronic ulcer or heal kindly. In size they vary from that of a nut to a hen's egg, and are filled with a yellowish-green liquid. The bullæ occur in any part of the body excepting the scalp; they are frequent on the palms and soles.

As the process of neuritis advances there is widespread hyperæsthesia, then anæsthesia and pains radiating along the nerves. When large superficial trunks are involved, these may be felt as thickened cords under the surface. Later on, atrophy of muscles, partial palsies, and various trophic disturbances occur. In the hands may be seen the claw hand, which also occurs in other forms of neuritis; and the phalanges, as also those of the foot, may inflame, swell, and be removed by ulceration. In the foot a perforating ulcer of the sole is quite common.

Diagnosis.—Tubercular leprosy must be distinguished from tubercular syphilis. The distinction is easily made by the altered sensation, by the history, and by the distribution of the lesions. Kalindera advocates the application of a blister and the examination of the serum for bacilli. Nerve leprosy is distinguished from vitiligo and similar affections by the altered sensation, and from syringomyelia, or Morvan's disease, by careful examination of sensation, by the partial palsies, and by the distinct spots.

Prognosis.—The prognosis is always bad, but, as there are at least 155 cures recorded in the literature of this disease, the outlook is not hopeless. The tubercular cases usually live from eight to ten years, the anæsthetic fifteen to twenty years. Acute cases with rapid death have been described by Leloir. The course of the disease may sometimes be arrested or partial restoration may occur, but this is rare.

The causes of death are mainly exhaustion, colliquative diarrhœa, obstruction to the air-passages, and inspiration pneumonia.

Treatment.—Prophylaxis is of the greatest importance. The good effects of isolation and hygiene were seen conclusively in the subsidence of the disease in Europe during the Middle Ages.

Of the many remedies successively praised and condemned, iodide of potassium, arsenic, chaulmoogra, and gurjun oil seem most worthy of trial. Danielssen after forty years' experience regarded the iodide as of distinct service. It should be pushed as freely as is possible without producing iodism. Chaulmoogra oil in doses of 2 drachms and gurjun oil in 10-minim doses may be of use. The latter has received special attention of late. It may be used by inunction when the stomach is sensitive. Palliative and supporting measures will be needed at the end.

DIPHTHERIA.

By W. GILMAN THOMPSON.

Definition.—Diphtheria is an acute, infectious, and inoculable disease, occurring sporadically and epidemically. It is characterized anatomically by a croupous inflammation of the mucous membrane and abraded surfaces. This affects chiefly the pharynx and upper air-passages, and has a marked tendency to spread to adjacent parts. It is attended with enlargement of the associated lymphatic glands. Clinically the disease is marked by irregular fever, great debility, and frequent albuminuria; by a tendency to death from toxæmia, from membranous croup, or from heart-failure; by slow, uncertain convalescence and by peculiar paralytic sequelæ.

Synonyms.—Diphtheritis; Cynanche contagiosa; Angina maligna; Diphthérite (Fr.); Diphtherie (Ger.).

Etiology.—*The Bacillus.*—Diphtheria is undoubtedly a germ disease, caused by the activity of the bacillus. There has been much dispute as to the identity of the bacillus, but the clinical history of the disease and all that is known in regard to its propagation afford conclusive proof of its bacillary origin. A variety of microbes are found in the mouth and throat in connection with diphtheria, such as staphylococci and streptococci, which render difficult the isolation of a specific diphtheritic germ. Harmless germs may even accumulate in the lymph-vessels leading from the inflamed surface. It is an important question, especially in regard to any theory of treatment, whether diphtheria is a general disease with a local lesion or whether it is primarily a local disease. In other words, is there a general systemic infection which may give rise to constitutional symptoms prior to the appearance of any local lesion produced by action of bacteria, or are the constitutional symptoms, fever, albuminuria, paralyses, etc., caused by absorption of ptomaïnes generated by the agency of the bacteria?

The majority of authors are still agreed in calling diphtheria a general or constitutional disease which develops a local lesion at some period of its course, though many are inclined to advance the opinion that in exceptional cases the local lesion may first appear. Further researches in regard to the bacterial origin and mode of infection by the disease may alter their views.

The *bacillus diphtheriæ* was discovered by Klebs in 1883 and by Löffler of Greifswald in 1884. It is found in the exudate or false membrane on the surface of the mucous membrane, and is often coughed out with shreds of pseudo-membrane. The causative relation of this germ to diphtheria has been disputed by some bacteriologists, but there is accumulating evidence in favor of its being the originator of the disease. From the fact that both

observers deserve credit for the discovery of the germ it is often called the "Klebs-Löffler bacillus." Among those who have confirmed the observations and experiments of Klebs and Löffler are Babes, Ortmann, V. Hoffmann, Welch, Abbott, and other experienced observers.

The Klebs-Löffler bacillus has been passed through a series of twenty-five rabbits by inoculation of the false membrane, and still retained its virulence. The bacilli are more numerous and appear better nourished in the pseudo-membrane than in prepared culture media. The diphtheria bacillus is a little shorter than the tubercle bacillus, but is much broader and has thickened or clubbed extremities. It is sometimes curved, sometimes spindle-shaped. It is 2.5–3μ long and non-motile. It is capable of deep staining, and then presents a segmented granular appearance. The bacilli often occur in groups. On the outer surface of the false membrane several varieties of bacilli, including the Klebs-Löffler germ, are found. Immediately below is a layer containing many cells and but little fibrin, and here, again, the bacilli in groups are apparent. Finally, in the deepest fibrin layer which rests upon the mucous membrane no Klebs-Löffler bacilli are present (Welch, Abbott). The bacillus diphtheriæ grows readily on a variety of culture media. It is killed at 58° C. in ten minutes (Welch, Abbott).

This bacillus, inoculated in the lower animals, produces symptoms resembling diphtheria, with pseudo-membrane, underlying necrosis, paralyses, and albuminuria. In man it comes in contact with the faucial or other mucous surface or the abraded skin, and propagates there, but it does not penetrate deeply into the mucous membrane, nor is it taken up by the blood-vessels or lymphatics. The bacilli therefore do not invade the entire body, but remain at the site of the local lesion, imbedded in the pseudo-membrane.

As a result of experimental inoculation of the Klebs-Löffler bacilli in animals typical microscopic changes occur in various organs, notably the liver and kidneys. In the liver Babes observed swelling, degeneration, and also proliferation of the hepatic cells. Leucocytes accumulate with fragmented nuclei. The capillary endothelial cells are swollen, and the vessels themselves contain hyaline and yellowish granular matter. Similar changes occur in the renal epithelium and blood-vessels. These results have recently been substantially confirmed by Welch and others. At the site of inoculation a gray pseudo-membrane appears over necrotic tissue with extreme fragmentation of nuclei. This is surrounded by an area of hyperæmia and congestion with ecchymoses. The neighboring, and even the distant lymph-glands are swollen and hæmorrhagic. The various serous cavities contain increased exudation.

Oertel has shown that these visceral and local changes accompany human diphtheria; and it is of the greatest interest and value to know that they may be experimentally produced by pure cultures of the Klebs-Löffler bacillus.

The Klebs-Löffler bacilli produce one or more ptomaïnes or toxines, which are absorbed by the lymphatic and blood-vessels and give rise to the constitutional and toxic symptoms. The toxines can be generated by cultivation of

the germs in artificial media, and they have been recently isolated and freed from bacilli by Roux, Brieger, Fraenkel, and others.

The Ptomaïne.—The principal ptomaïne when swallowed, like many other poisons, produces but little effect, but when inoculated it causes unknown chemical alterations in the tissues of the body; and it is suggested that a certain degree of immunity which follows an attack of diphtheria is due to this cause (Fraenkel, Behring). It is destroyed at a temperature of about 140° F. It is soluble in water, but is precipitated by alcohol. It resembles proteids in composition. There is reason to believe that the bacilli themselves are really innocuous apart from their production of the specific ptomaïnes. One ptomaïne has been isolated which, if injected into animals, causes increased secretion from the mucous membrane of the eyes, nose, and mouth, together with chill and fever. This is followed by convulsions, involuntary evacuations, paralysis, dyspnœa, and death.

Immunity.—By recent experiments with inoculation of attenuated cultures of diphtheritic virus Behring, Kitasato, and Fraenkel have succeeded in rendering certain animals immune to diphtheria. With guinea-pigs and rabbits Behring has been able to regulate the dosage with almost mathematical accuracy, so as to produce the disease with varying grades of intensity—with paralyses or without, with constitutional symptoms or with only slight local infiltration. What is still more important, he has been able to cure already infected guinea-pigs by inoculation with the blood of animals rendered immune by previous inoculation. This occurs even with the inoculation made at some distance from the local lesion.

Behring and Kitasato have shown that in rabbits the action of the attenuated diphtheritic virus destroys the toxines formed by the bacilli, rather than the bacilli themselves.

According to recent experiments of D'Espine, the bacillus diphtheriæ is killed by local applications of aqueous solutions of the strength of 1 : 8000 for corrosive sublimate, 1 : 2000 for salicylic acid, and 1 : 50 for carbolic acid.

The bacilli still live at temperatures below that of the body, even at 68° F. Boiling destroys them. They thrive and multiply in milk. They preserve their vitality for four or five months or more in dried membrane. Cultures of bacilli have been kept for sixteen months without losing their virulence. A brush used to swab the throat of a diphtheritic child was put aside in a drawer uncleaned: after four years it was taken out and infected a man who used it. In a Normandy village, twenty-three years after an epidemic of diphtheria, some of the bodies of those who died of the disease were exhumed, and an epidemic at once broke out, first among those who opened the graves, and extended to others (Sevestre).

Diphtheria in Animals.—Diphtheria is common among certain domestic animals, such as fowls, pigeons, calves, cows, pigs, and especially cats, and it may be inoculated in sheep. They acquire a false membrane in the throat, with constitutional symptoms, and various bacilli are found at the site of the

inflammation. These bacilli are not in every case identical with the Klebs-Löffler bacillus. In birds, at least, the bacilli are different, but it is highly probable that true diphtheria may be transmitted from cats to man, and conversely. In cats the disease principally affects the lungs and bronchi. Filthy stables, dirty poultry-yards, and dove-cotes favor the spread of diphtheria among domestic animals.

It is claimed by Power and others that bovine diphtheria can be transmitted to man through infection of the milk by an eruptive disease of the udders, and cows inoculated under the shoulder with diphtheritic microbes exhibit the germs in their milk after developing local lesions of the udders. Transmission of diphtheria to man through milk is certainly rare, if it be possible. Roux and Yersin have isolated a microbe which excites diphtheria in fowls, rabbits, etc., and have also separated from it a toxine which when injected causes asphyxia and paralysis in those animals.

Pseudo-Diphtheria.—Some authors are disposed to describe two varieties of diphtheria—one accompanied and occasioned by the Klebs-Löffler bacillus, and the other due to some different virus or germ (possibly the streptococcus pyogenes). The latter variety they call "pseudo-diphtheria." Prudden has recently made exhaustive researches in cases of diphtheritic inflammation occurring in connection with measles, scarlatina, etc., in order to determine the character of the bacilli present. In such cases he failed to find the Klebs-Löffler bacillus, although he obtained a streptococcus. He concludes, therefore, that the latter germ is the cause of these secondary cases of diphtheria.

The statements regarding the etiology of pseudo-diphtheria and the germ or germs associated with it are very conflicting, and further research will doubtless throw much light upon the subject. Abbott[1] concludes a report of a recent investigation of this question by saying: "From these observations we feel justified in agreeing with the opinion that has been advanced by other observers, particularly Hoffmann and Roux and Yersin, that under varying conditions the virulence of the true diphtheria bacillus may be observed to fluctuate in the degree of its intensity—at one time possessing the property in a high degree, at another presenting a decided attenuation, and not unfrequently a complete absence of pathogenic power."

The pseudo-diphtheria germ has been found in the pharynx in healthy children.

In pseudo-membranous angina, occurring in connection with scarlatina, a streptococcus is found, but the true bacillus diphtheriæ is absent. Germs are also found in inflamed conditions of the mucous membrane accompanying measles and erysipelas, but the Klebs-Löffler bacillus occurs only in diphtheria.

False membrane closely resembling diphtheritic membrane, excepting the absence of the diphtheria bacillus, is formed on mucous surfaces after the application of various irritants, such as hot steam, cantharis, strong corrosive

[1] *Johns Hopkins Hospital Bulletin*, vol. ii. No. 17, p. 146.

sublimate, arsenic, ammonia, etc. Huebner has shown that a false membrane may even form as a result of mechanical obstruction to the local vascular supply. Hence the presence of the germ of diphtheria is not essential for the production of a false membrane.

Much of this bacteriological work is so recent that it is both difficult and unwise to adopt positive conclusions from it until further evidence, which is now being diligently sought, shall be brought to light.

The habitat of the microbe when outside of the body is not known, but it is believed to live in surface soil which is contaminated with organic matter. It is known to grow on excretory refuse.

Mode of Infection.—Infection is not believed to occur through the alimentary canal. Some observers claim that it is possible for the diphtheritic virus to enter the lungs by inhalation, and, being absorbed by the pulmonary vessels, develop constitutional symptoms before the local lesion in the throat is manifest; but the weight of evidence is decidedly in favor of regarding the mucous membrane of the upper air-passages as the common site of infection.

It is not proved definitely that in man an abraded surface is necessary for the virus to find lodgment, but many believe that a diseased, denuded, or catarrhal mucous surface is essential in man, as it is for inoculation in the lower animals. It is certain, however, that diseased mucous surfaces are far more susceptible to the infection than healthy surfaces. Moreover, it is very easy to overlook slight abrasions after the local symptoms have become manifest.

The mode of infection is either by direct contact of shreds of diphtheritic membrane, or more frequently through infected air, or through the agency of various fomites, such as contaminated clothing, handkerchiefs, toys, books, etc. Shreds of fibrin are frequently expelled with a violent cough, and if they happen to lodge in the eye or mouth of the physician or nurse, they seldom fail to infect. Many valuable lives have been sacrificed in this way. Physicians have lost their lives from endeavoring to clean out a tracheotomy-tube by sucking a plug of mucus or membrane from it which threatened to suffocate the patient. The disease has been transmitted by kissing.

Diphtheria is highly contagious within the immediate neighborhood of the patient, but the radius of direct contagion is limited to a few feet. For this reason a diphtheritic patient may be perfectly well treated at home without endangering other members of the household, provided the necessary local quarantine can be established and rigidly enforced.

The bacilli cling to different objects with great tenacity, and in this way are conveyed for considerable distances while preserving their vitality. Moisture is more favorable to their growth than dryness.

The spread of diphtheria is greatly favored by bad hygienic conditions, such as are found in damp, dark houses, unwholesome cellars, the presence of sewer-gas, etc. There is some doubt as to how far the latter is directly concerned in the propagation of diphtheritic virus, but it is certainly true that persons who live under unfavorable hygienic conditions and inhale

sewer-gas acquire anæmia and a general lowering of vitality that makes them susceptible to any contagion.

Fifty different epidemics of diphtheria occurring in various localities in England were recently investigated with great care, and in only four could the outbreak be traced to direct contagion. The rest were all connected with foul cesspools, deficient drainage and sewerage, or the proximity of dirty animals and decomposing organic matter, such as manure.

Diphtheria prevails in isolated rural districts as well as in towns. In this country it has often appeared unaccountably in a new and sparsely-settled frontier village or in an isolated family on a ranch. There is a possibility of its having been communicated by domestic animals, but as yet, excepting perhaps in the case of cats, this is by no means proved.

The ignorance of many persons regarding the contagion of diphtheria is a factor in extending an epidemic. It is often propagated through clothing washed or manufactured in a room where a diphtheritic patient lies. Successive cases may occur among the members of the same family at intervals of a few weeks.

Age.—Diphtheria is principally a disease of childhood, for, although it may occur at any period up to fifty years of age, the susceptibility to it diminishes rapidly after youth, and it is commonest under ten years of age, the majority of cases occurring between the second and seventh years. It is very rare in early infancy, although it has been acquired by the newborn (Eichhorst).

Sex.—Diphtheria is practically uninfluenced by sex. Some authors say that it is slightly more common among females.

Climate.—Diphtheria is found in all climates, but less frequently in the tropics than in cold and temperate regions. It is a common disease throughout the United States and Europe. In certain localities it is endemic, and at intervals becomes epidemic. It is favored by cold and damp weather.

Immunity.—One attack of diphtheria does not confer immunity; in fact, a patient may have several attacks, and may even be reinoculated during convalescence by germs which have accumulated in the vicinity from lack of cleanliness.

Season.—In the United States diphtheria is more common in the winter and spring than at other seasons.

Associated Diseases.—Other acute diseases affecting the throat are very commonly associated with diphtheria. Foul effluvia from excrement and sewer-gas doubtless beget in some cases sore throat which is not diphtheritic, but laryngitis, tonsillitis, pharyngitis, and quinsy are all apt to prevail during an epidemic of diphtheria, and to originate in like conditions of environment, although there is no anatomical relation between them.

Epidemics of diphtheria sometimes are related to the prevalence of other diseases; thus, they are apt to follow epidemics of measles or to accompany scarlatina or variola. Diphtheria and typhoid fever are often prevalent simultaneously in the same localities. Taylor[1] relates a case in which scarlatina,

[1] *Lancet*, Aug. 2. 1890, p. 232.

measles, and diphtheria occurred simultaneously. Erysipelas sometimes occurs with diphtheria in the same individual; and such cases become rapidly malignant.

Morbid Anatomy.—Since diphtheria usually attacks the upper air-passages, the mucous membranes which are most frequently affected are those of the tonsils, soft palate, pillars of the fauces, posterior pharyngeal wall, larynx, trachea, and nose. In addition, the diphtheritic inflammation may involve the gums, root of the tongue, buccal wall, œsophagus, fundus of the stomach, rectum, vagina, the puerperal uterus, and open wounds on the surface of the body or an abraded surface like a blister or leech-bite.

Diphtheritic inflammation is characterized by the production of fibrinous exudation. The stroma of the mucous membrane is filled with more or less disintegrated leucocytes, pus, and fibrin, which also appear on its free surface. There is coagulation-necrosis of the epithelium, which is shrivelled and distorted in form, with indistinct nuclei or none at all. The false or pseudo-membrane is composed of flaky, necrosed epithelium, disintegrated leucocytes, pus, fibrin, and bacilli in varying relative amount, and sometimes red blood-cells. The mucous membrane beneath is more or less necrotic, and the submucous layer is also necrotic in bad cases. There is hyperæmia, and often tumefaction of the surrounding mucous surface, which secretes a muco-purulent exudation. The redness of the surrounding area is often distinctly localized, and it may be confined to one side of the throat.

The fibrin is supposed to be derived mainly from inflammatory exudation which transudes from the capillary walls, and which is coagulated by ferment derived from disintegrated leucocytes (Weigert). The cell-bodies may assist in forming the fibrin, but the epithelial cells probably do not play any very active rôle in the inflammation, although some pathologists have claimed that they are altered in shape and produce branching fibrillæ. As the inflammation subsides the necrosed portion of mucous membrane sloughs off, and the epithelial surface is restored by outgrowth from neighboring cells. If there has been deep involvement of the mucous and submucous layers, cicatrices may be formed, such as sometimes occur in the tonsils. Otherwise, after the false membrane has sloughed off, the mucous surface is left clean and smooth. Several successive membranes may form at the same site, and this is especially the case when they are forcibly stripped off. The process of exfoliation occupies several days.

The Klebs-Löffler bacilli are found scattered through the meshes of the fibrillated fibrin or in granular fibrin. With them are often seen other varieties of microbes, especially streptococci and staphylococci, occurring in colonies or isolated. These other forms of microbes may be found situated more deeply than the bacillus diphtheriæ.

The inflammation affects the mucous membrane with an intensity varying from congestion, with a thin, grayish or yellowish film spread over the surface in isolated irregular patches or in a single layer, up to the formation of a thick, firmly attached pseudo-membrane covering a tumefied mucous surface with more or less necrosis of the deeper layers.

The Membrane.—The character of the pseudo-membrane varies somewhat with the structure of the particular mucous membrane affected. It is apt to be more fibrinous and more firmly adherent to a surface covered originally by squamous cells instead of by ciliated or other forms of epithelium.

The membrane is of a grayish-white color, and if superficial can be stripped off, and is found to be elastic and firm. Shaken in water, it does not disintegrate. It swells in acetic acid. If the deeper tissue be involved, the membrane is more adherent, and attempts at its removal may lacerate the surface and produce bleeding. This is especially the case over the irregular tonsillar surfaces. The color deepens as the membrane becomes older. It grows yellowish, and may be streaked with red from admixture of blood, or it becomes dark brown. It may soften and break down into an offensive, ichorous, brownish discharge. After death it is apt to decompose and soften rapidly. Very rarely the membrane may be absent, in which case the inflamed surface is swollen and of a grayish-white color from infiltration.

When the pseudo-membrane is advancing the edges are thin, and they shade into the surrounding area of inflammation, but if repair is about to take place, the patches may thicken or wrinkle at their edges, which become distinctly separated from the mucous membrane. The pseudo-membrane is loosened by effusion of serum and immigration of leucocytes beneath, and by ulcerative process, so that it sloughs off in fragments or, less often, as one piece.

Lesions of the mucous membrane undoubtedly aid the spread of the virus, and hence the injurious effect of forcibly stripping off the false membrane and exposing raw bleeding surfaces. It is said that the reason the tonsils are more frequently the starting-point of diphtheritic inflammation is partly because of their prominence, but also on account of the fact that their epithelial covering is not always everywhere continuous, and hence the virus has easier access to their mucous membrane. The virus may, however, undoubtedly attack a mucous membrane in which no abnormality is discoverable, possibly because a slight abrasion is so readily overlooked after the local inflammation has begun.

The *heart* shows more or less fatty infiltration between the muscular fibres and around the blood-vessels. The muscle-fibres themselves may have swollen nuclei. Both ventricles are often dilated.

The *epiglottis* may be congested, but it is exceptional for it to become sufficiently œdematous to impede respiration.

The *bronchi* may appear normal or present a catarrhal or croupous inflammation. The diphtheritic membrane may extend over their mucous surfaces down to the bronchioles. There may be broncho-pneumonia or lobar pneumonia from inhalation of shreds of fibrin and putrid material from sloughs in the mouth. If death has occurred from suffocation, the lungs may be slightly emphysematous (Flint).

The *lymphatic glands* near the site of the local inflammation are the seat of hyperplasia. The lymphatic glands at the angle of the jaw and in the neck

are most apt to be affected, especially if the nares are involved. The various salivary glands may also be enlarged. As a rule, the glandular swelling subsides without suppuration. Sometimes the periglandular tissue becomes infiltrated and greatly swollen.

In malignant cases there is deep sloughing, or even gangrene, at the site of local inflammation, and there may be hæmorrhages from the various mucous membranes, or general purpura and parenchymatous degeneration of viscera. The spleen may be somewhat enlarged, and both spleen and liver may be hyperæmic. The blood will coagulate poorly, and it is very dark. Antemortem heart-clots and venous thrombi may occasionally form. Small hæmorrhages have been found in the meninges of the brain and spinal cord.

Symptomatology.—The period of incubation of diphtheria may be very brief, lasting only twenty-four or thirty-six hours, or it may occupy a week. It will depend somewhat upon the severity of the epidemic, the incubation period being shorter where the poison is concentrated or propagated by experimental inoculation.

The symptoms are both local and constitutional, and they may vary considerably, for mild and malignant types may develop side by side. There is no definitely fixed relation between the general symptoms and the intensity of the localized inflammation.

Prodromata.—Diphtheria usually commences with certain prodromal symptoms, such as malaise, anorexia, headache, and sometimes nausea and vomiting. There may be chilly sensations, but a distinct chill is not common.

Invasion and Course.—In a few hours the patient complains of slight dryness and soreness of the throat on swallowing, but the pain is seldom as acute as in tonsillitis. There may be pain also on speaking, or aphonia. Sometimes the throat is slightly anæsthetic. There is a feeling of increasing weakness and more or less general muscular soreness. There is a slight rise of temperature. Upon examining the throat the tonsils are found slightly swollen and reddened, and there may be hyperæmia of the pillars of the fauces and uvula. One or two small grayish or yellowish spots will be observed upon the inner surface of one or both tonsils. They are covered by a thin, firmly-adherent membrane. The spots remain unchanged for a day or two, and in the mildest cases only the tonsils are affected. In other cases the spots promptly coalesce, and a continuous membrane is formed which covers the whole of the tonsils, stretching up over the fauces and soft palate. There is more or less redness and swelling of the adjacent mucous membrane, and there may be small ecchymoses in it. Sometimes there is no pain at all in the throat, even in bad cases, or there is moderate pain, dysphagia, and thirst. The constitutional symptoms continue. The pulse becomes more rapid, 120 or 140, and very feeble. The first sound of the heart is indistinct. The patient feels ill and looks pallid. The temperature may rise in a day or two to 104.5° F., or even higher, but in many cases it remains below 102.5° throughout the disease. The whole course of the fever is irregular. The respiration is not particularly affected, but it may

be quickened. The breath is fœtid. The tongue is coated and sometimes swollen. There is complete anorexia, and nausea may be present. The bowels are costive. There is more or less swelling of the cervical lymphatic glands, with pain or soreness on opening the jaw, which is sometimes felt also in the ears. The glandular enlargement is usually symmetrical.

By the second or third day albumin may be found in the urine, which may become scanty and high colored, or, in other cases, may still appear normal to the eye. After a week or ten days the throat begins to improve, the constitutional symptoms abate, and the patient, somewhat enfeebled, slowly convalesces; or the disease proceeds to a fatal issue, in which case, instead of the membrane ceasing to extend, it advances, passing either upward or downward.

If the membrane reaches into the vault of the pharynx, it passes the posterior nares and comes forward to fill the nasal cavities. There may be deafness from swelling of the orifices of the Eustachian tubes or extension of the inflammation into them.

When the nares are involved there will be a thin, muco-purulent discharge from the nose, which may excoriate the septum, alæ, and upper lip. It later becomes brown, and is frequently hæmorrhagic and extremely offensive in odor. There are snuffles and sneezing, and young infants cannot suckle, and must be fed with a spoon. Mouth-breathing becomes necessary. The nose itself and the upper lip become somewhat red and swollen. The glands at the angle of the jaw and the submaxillary glands become enlarged and tender, owing to their connection with the Schneiderian lymphatic vessels. This glandular swelling is often the first sign of nasal diphtheria. Sometimes the connective tissue between the glands is infiltrated, and the entire neck may be greatly swollen, so as to interfere with the motion of the head. In bad cases the inflammation may extend along the lachrymal ducts and reach the conjunctivæ, which become red, swollen, watery, and finally are covered by pseudo-membrane. There may be uncontrollable epistaxis. In other cases the inflammation extends along the Eustachian tube, and finally excites an otitis media, with perforation of the tympanum and other grave lesions.

Should the membrane spread downward, as is very frequently the case, the larynx is lined with pseudo-membrane which obstructs respiration. The first symptom noticed is hoarseness, which occurs between the third and sixth days. Then the breathing becomes quick and shallow or noisy and stertorous. There may be aphonia. There is a peculiar characteristic harsh-ringing, croupy cough. The patient becomes cyanotic, cold, anxious, and sits up or tosses about in bed, gasping for breath, with the head extended, the mouth open, the alæ nasi working vigorously, and all the accessory respiratory muscles called into action. Owing to the impeded entrance of air, the supraclavicular spaces and the lower intercostal spaces are sunken by atmospheric pressure during inspiration. Breathing is superficial, rapid, and irregular. The patient may cough up pieces of membrane and secure temporary relief, but the dyspnœa returns promptly, as new membrane rapidly forms. A new membrane sometimes appears in half an hour.

The cough may be paroxysmal from a functional spasm of the vocal cords. Salivation may be present, and there is repugnance to food of any kind. In bad cases the vomiting may continue and be accompanied by diarrhœa.

While the local inflammation is augmenting the constitutional symptoms become very unfavorable. The pulse grows more and more rapid, feeble, and irregular. There is increasing pallor, and sometimes a cold perspiration covers the body. The fever continues or diminishes. The albuminuria increases; granular and epithelial casts, with sometimes a few red blood-cells, appear in the urine. The prostration is extreme. Neurotic symptoms are not prominent, and the mind is usually clear, but a typhoid condition with delirium may ensue. Young children sometimes have mild convulsions. Paralysis may appear at this time, involving the uvula, muscles of deglutition, or one or more of the extremities. Finally death results from suffocation unless tracheotomy or intubation be performed (*vide infra*), or from asthenia and cardiac paralysis.

Special Symptoms.—The *temperature* may remain low throughout the disease, or, beginning high with an initial angina, it may fall, later on, to 101° F., with increasing blood-poisoning. A continued high temperature is not at all characteristic of diphtheria.

The dyspnœa may have several causes. It may be due to obstruction in the larynx from swelling and accumulation of thick pseudo-membrane or to a piece of membrane, partly detached, which acts like a valve, flapping to and fro with the respiratory movements and closing the glottis in inspiration. Again, it may be due to alteration or disintegration of the red blood-disks, and their inability to convey oxygen, produced by a toxic condition of the system. It may arise from heart failure and impeded pulmonary circulation, or it may occur from sudden spasm or paralysis of one or both vocal cords.

When death ensues from extreme dyspnœa and suffocation, the mind becomes dull or there is coma, followed by convulsions and opisthotonos. The heart becomes very feeble and the cardiac sounds are inaudible; the radial pulse cannot be felt. The surface of the body is cold, moist, and blue.

The urine presents the features commonly found in acute febrile disease. The urates are increased, the color is deepened or normal, the specific gravity is raised, and the quantity is lessened. There is more or less albumin in over 50 per cent. of the cases. There are epithelial and granular casts, and sometimes a few blood-cells. Albuminuria is rare in the first day or two. It usually occurs between the third or fifth and the tenth day. It may be due to acute nephritis or to toxines which modify the albuminous ingredients of the tissues and blood, or it may occur late in the disease from renal congestion in connection with dyspnœa and faulty aëration of the blood. In the latter case it sometimes disappears after intubation or tracheotomy. The intensity of the albuminuria is apt to conform to the severity of the disease. The albuminuria usually subsides as the symptoms abate, and the parenchymatous nephritis seldom becomes chronic. It is exceptional for it

to occasion uræmia or œdema. When acute interstitial nephritis occurs there is great infarction of the stellate veins (Sympson).

The pulse is uniformly rapid. In infants it often reaches 180 or 200; in older children, 140 or 150. It may drop suddenly below the normal, which is always a serious indication of increasing cardiac weakness. There is often sudden cardiac paralysis during convalescence, which may appear after the patient has been walking about. The right side of the heart may become suddenly dilated, with feeble sounds, irregular action, and absent impulse.

Malignant Cases.—In any severe epidemic of diphtheria, and sometimes sporadically, a certain number of cases assume a distinctly malignant type. They may be of two classes, in both of which the invasion is acute, with rigors, headache, vomiting, and sudden prostration.

In the first variety the system is overwhelmed with diphtheritic poison, so that death occurs in two or three days, from disintegration of the blood and heart-failure, before the membrane has had time to extend. In these cases there is somnolence, stupor, or delirium, the heart-action rapidly fails, the skin is cold and clammy, and there may be ecchymoses in it.

In the second variety the membrane spreads very rapidly, simultaneously covering a large surface, and laryngeal and bronchial inflammations quickly supervene. In the worst cases the entire tonsils, fauces, uvula, buccal cavity, posterior pharynx, larynx, and nasal cavities are covered with thick, brown, foul-smelling membrane. Extensive sloughing, and even gangrene, may result. Such cases are almost hopeless from the commencement, and every effort must be made to quarantine and disinfect them.

As a rule, adults are more apt to suffer from severe constitutional symptoms, and children from laryngeal extension of the inflammation. Adults often die while there is comparatively little membrane present.

Duration.—Diphtheria is a disease without definite duration. The average case lasts about ten days or a fortnight. Very mild cases may recover in a week, while others, more severe, last three or four weeks. There are cases which continue for two months without the throat becoming entirely well, although the constitutional symptoms may abate. Bacilli have been found by Klebs lingering in the throat after the false membrane had disappeared.

Terminations.—Diphtheria may end in recovery, or death ensues from one or other of the following causes: extension to the larynx and suffocation by occlusion; laryngeal spasm or paralysis; asthenia; cardiac paralysis, and syncope.

Complications.—The two most frequent complications, which are also the most severe, are albuminuria and paralysis. They are so frequent, in fact—especially the albuminuria—as to be regarded by some authors as symptoms. On the other hand, they may occur later as sequelæ.

Endocarditis is a rare and fatal complication of diphtheria.

A rare complication is the invasion of the conjunctiva by the diphtheritic inflammation. This is always an exceedingly grave accident, and it may

result in the destruction of the cornea inside of two days, with a total loss of vision.

Fatal epistaxis or severe hæmorrhages from the diseased mucous surfaces are unusual complications. Extreme alterations in the pulse-rate on either side of the normal may occur, but a very slow pulse is exceptional.

Sequelæ.—The sequelæ of diphtheria are both mild and severe, and they may last throughout several months. Congestion of the fauces and tonsillar hypertrophy may continue for some time. The uvula is often hypertrophied, and chronic nasal catarrh may be instituted.

The paralyses which follow diphtheria, or which in some cases appear in connection with the height of the disease, are various. They are commonly trophic motor paralyses, but may be both motor and sensory, and commonly occur within two or three weeks after disappearance of the throat symptoms.

They are peculiar, for one set of muscles often regain their function while another is losing it, and they are as likely to occur in mild as in severe cases. As a rule, recovery takes place in from six to eight weeks, but some cases are protracted for a year or two.

The tendon-reflexes are very frequently abolished, and the normal knee-jerk may be absent for some time. Absence of pain or tenderness along the nerves, such as occurs in multiple neuritis, is noted.

The soft palate is oftenest affected. It hangs loose, is insensitive, and the voice becomes nasal. Occasionally one-half only is paralyzed, and it is drawn toward the opposite side. This paralysis may occur irrespective of any inflammation of the uvula. The reflex action is abolished. The tongue and pharyngeal muscles may be involved.

The various ocular muscles are often paralyzed, giving rise to loss of accommodation, double vision, or strabismus. One or both vocal cords may be paralyzed and seriously interfere with respiration. The diaphragm and intercostal or cervical muscles may be paralyzed. Exceptionally there is paralysis of the sphincters of the bladder and rectum, with retention of urine and fæcal incontinence. There may be loss of tendon-reflex at the knee, with inco-ordination in gait and without loss of muscular power in the legs. This ataxia is accompanied by loss of sensation in the feet and legs, by swaying of the erect body, and loss of balance when the eyes are closed (the Romberg symptom).

The legs are more apt to be paralyzed than the arms. Paralysis of the heart is quite frequent, and is supposed to follow degeneration of the cardiac nerves or ganglia. Various forms of anæsthesia and dysæsthesia are sometimes present.

The paralyses are, in part at least, due to degenerative changes in the peripheral nerves, though in some cases central lesions may occur. As a rule, the muscles supplied by the affected nerves do not atrophy, and they retain their response to electric stimulation, both galvanic and faradic.

Some one of these forms of paralysis occurs in 40 per cent. of cases in a

bad epidemic. The prognosis is generally good for final recovery from the paralysis unless the muscles of respiration or deglutition or the heart are affected. Chorea and epileptiform attacks have been observed exceptionally, and there may be peripheral hyperæsthesia. There are often persistent anorexia and obstinate anæmia.

Diagnosis.—It is more common to mistake various lesions of the throat in adults for diphtheria than to overlook diphtheria when once fairly established. Young children, however, seldom complain of the throat symptoms in any manner, and their throats should be examined as a matter of routine in any doubtful illness. Otherwise, cases of diphtheria may advance beyond control before they are discovered.

In simple inflammation of the pharynx and in follicular tonsillitis, as compared with diphtheritic inflammation, the invasion is more sudden, the temperature higher, and the redness of the throat more diffuse, and not confined to one side, as is often the case in commencing diphtheria. In tonsillitis the uvula is not involved, but in diphtheria it usually is, and the nasal cavities may be also affected. Moreover, in follicular tonsillitis the yellowish-white spots can often be removed from the crypts which the secretion occupies, and the inflammation frequently abates in thirty-six hours, whereas in diphtheria it continues for a week or two. A doubtful-looking layer of mucus is often removed by having the patient gargle the throat with salt water.

Cases of difficult diagnosis occur occasionally from the fact that the membrane is concealed by originating in the nares or, as reported by Jacobi, in the trachea before other surfaces are affected. The tracheal origin is denied by many observers.

There are cases of inflammation of mucous surfaces which are traumatic, and which closely resemble diphtheria from an anatomical standpoint, except from the absence of bacilli—such as are caused by irritating substances swallowed or inhaled by mistake, as, for example, chlorine, ammonia, live steam, etc. When an epidemic originates many cases occur which pass undetected as an ordinary "sore throat," and their real nature is not apparent without very careful investigation.

The separate identity of diphtheria and membranous croup has occasioned much discussion. It has been held, on the one hand, that the two diseases are etiologically and anatomically identical, however much their clinical aspects may differ; and, on the other hand, that throughout they are two very distinct diseases.

If the Klebs-Löffler bacillus be accepted as a *sine qua non* in the etiology of diphtheria, then croup must necessarily be separated from it. As a matter of fact, the anatomical appearance of the membrane in both diseases is identical, excepting only the absence of the Klebs-Löffler bacillus, the two lesions differing only in degree, while the clinical histories are very different.

In croup the inflammation begins in the larynx, whereas in diphtheria it rarely commences there, but passes by extension from the pharynx. In croup the necrotic change is more superficial and may be limited to the epithelium,

whereas in diphtheria the deeper layers of the mucous membrane are involved, and even the submucosa may sometimes slough. Although the membranes may appear alike, excepting in regard to the presence of the Klebs-Löffler bacillus, clinically the two diseases are very unlike.

Membranous croup is a local disease; diphtheria is a general disease with a local inflammation. Croup is not epidemic or contagious, and very rarely affects adults, as diphtheria may. Albuminuria does not occur, no paralytic sequelæ follow, and the lymphatic glands are less apt to be enlarged than in diphtheria. In croup the inflammation begins suddenly in the larynx, and does not readily tend to spread to the trachea or œsophagus. The invasion of croup is apt to be more sudden and severe than it is in diphtheria.

The sore throat occurring with scarlatina is sometimes mistaken for diphtheria, and, in fact, the latter disease may sometimes occur in conjunction with scarlatina. In the scarlatinal throat the redness is much more diffuse than in diphtheria, and there is the characteristic "strawberry tongue." In doubtful cases a careful bacteriological examination should be made, when streptococci may be found, but the Klebs-Löffler bacillus will be absent.

In erysipelas of the throat the cervical glands are less apt to be enlarged, and the tongue is brown and dry, and the mode of extension of the two diseases is very different.

Occasionally, syphilitic mucous patches in the pharynx, with inflammation of the fauces, may resemble diphtheritic inflammation, but the history of the case and the absence of acute constitutional symptoms, with the course of the disease, will soon establish the diagnosis.

In adults in any doubtful case of throat lesion the urine must be examined. The sudden appearance of albumin favors the diagnosis of diphtheria, for it does not occur in simple tonsillitis or pharyngitis. Moreover, the peculiarities of the pseudo-membrane, its extension, and the progress of the constitutional symptoms will seldom fail to distinguish diphtheria from the former affections.

Prognosis.—The prognosis should always be guarded. It varies in different epidemics. It is favorable in the absence of extension of the membrane to the throat or nose, in the absence of albuminuria, and with fair digestion and a strong heart-action. Patients seen early and properly treated stand a very fair chance of recovery.

Cases of nasal diphtheria are apt to end fatally unless vigorously treated. It is probable that this is so because of the great vascularity and abundant lymphatic vessels of the Schneiderian membrane, which readily absorb septic material and distribute it in the system. A brownish, watery, nasal discharge, streaked with blood and having an offensive, sweetish odor, is a worse omen than a thick membrane. Involvement of the hard palate and mouth to an extreme degree is worse than extension to the nose.

The quantity of the membrane formed does not always indicate the severity of the disease. Patients may exhibit a mere trace of it in the fauces while they are in collapse from systemic blood-poisoning; or the membrane may

be an eighth of an inch in thickness and cover a wide area, and yet recovery may take place. As a general rule, however, a rapidly-extending, uncontrollable inflammation makes the prognosis very grave. The temperature affords very little guidance in prognosis. The disease is the more fatal the younger the child. The worst cases occur while the epidemic is advancing, not when it declines.

The prognosis is worse when scrofula exists, and when the diphtheria follows an attack of measles or scarlatina which has already exhausted the child.

Mortality.—The death-rate of diphtheria varies with different epidemics. It sometimes exceeds 40 per cent. and has even reached 76 per cent. With 900 cases recently treated in Strasbourg the mortality was 46.7 per cent. In New York it averages above 47 per cent., and may reach 55 per cent. When cases are isolated and favorably placed and treated it is much less. Over 50 per cent. of the deaths from diphtheria occur in children under five years of age, and about 75 per cent. occur among those under ten years of age. Despite every effort for the control of diphtheria, the death-rate has remained undiminished for many years, and it often proves fatal to very robust children.

Treatment.—*Prophylaxis.*—The greatest danger in the spread of diphtheria is through the agency of "ambulatory" cases—*i. e.* cases in which the symptoms are so slight as not to prevent the patient from going about, and which nevertheless communicate the disease readily to other persons. A mild case in one individual may by contagion beget a severe or malignant one in another.

When diphtheria is epidemic precautionary measures should be taken wherever children are crowded together in school-rooms, asylums, or hospital wards; strict personal and general cleanliness should be enforced; and any child having the least indication of a sore throat should be kept apart from the rest. Cleanliness of streets, yards, privies, etc. is very important, for the disease thrives in filth. An abundant supply of pure water is highly essential, and good ventilation of dwellings and school-rooms should be insisted upon. It is often desirable to close the public schools temporarily to prevent contagion. Isolation and disinfection are cardinal principles in regard to the treatment of diphtheria, no matter how mild it may appear. Any infected animals should at once be killed. In the event of a death from diphtheria the body should be wrapped in a sheet wet in 1 : 3000 corrosive-sublimate solution, and placed immediately in a sealed casket, and the funeral should be strictly private, for the disregarding of this precaution has been a fertile source of epidemics.

The Hygiene of the Sick-room.—The patient should be placed in a bare room, free from superfluous hangings, rugs, or furniture, and kept very quiet in bed. The temperature of the room should not rise above 68° F. The air should be kept pure by free ventilation. If an adjoining room can be secured in which windows can be opened, it will greatly facilitate ventilation without exposure to draughts. In cold weather an open fire is desirable for ventilation

as well as warmth. In warmer weather a lamp should be kept constantly burning inside the fireplace in order to make a current up the chimney. The bed should be so placed that both sides can be readily accessible. All discharges from the patient should be carefully disinfected. Cheese-cloth rags should be used instead of handkerchiefs, and immediately burned. Brushes used in the throat or nose should be kept in corrosive-sublimate solution, and swabs should be burned after a single application. No one should be admitted to the room excepting the physician, nurse, or such members of the family as may be in constant attendance. Those in charge of the patient should be held in strict quarantine from the other occupants of the house. It is advisable for the physician on entering the sick-chamber to don a long linen apron or a sheet, for in examining the patient shreds of membrane are sometimes coughed up and light on the clothing, and the disease has been carried to others in this manner. In making applications to the throat which excite coughing one should be particularly careful lest the membrane be expelled in the face, and plain glasses may be worn to save the eyes from such danger. On leaving the room the clothing should be brushed, and the hands and beard should be washed in corrosive sublimate, and it is well to pass promptly into the open air. Those in constant attendance upon diphtheritic cases do well to spray their own nostrils and throats several times a day with weak corrosive-sublimate solution (1 : 10,000) or a similar disinfectant.

When diphtheria is epidemic the patients should not only be isolated from the healthy, but from each other, for crowding them together in wards greatly increases the virulence of the disease.

Many of the foregoing details may seem trivial, and they are often overlooked, but experience with infection from truly malignant cases of diphtheria has demonstrated that they are of the utmost importance.

The patient should be disturbed no more than is necessary for the accomplishment of treatment.

Local applications in diphtheria should always be vigorously employed, unless the patient is a very young or nervous child who is almost frightened into convulsions by their use. As a rule, a little tact and perseverance on the part of the physician and nurse will in time overcome any resistance. If the patient be very feeble or unconscious, care must be taken that no poisonous applications are swallowed. The pharynx can often be reached by pouring disinfectants in the nose—an operation which is much less alarming to a young infant than forcibly opening the mouth.

Local applications are useful—(1) as germicides; (2) for cleansing purposes; (3) to dissolve the false membrane; (4) to allay irritation. As a rule, the applications should be warm and mild.

The most successful local treatment, however, is that which is employed with the object of disinfecting and cleansing unaffected surfaces, and of thereby making them less liable to inflammation and to beget noxious products for absorption. Local treatment is of very little avail as a curative measure when the lesion is once established. It is well to constantly disinfect the nasal pas-

sages in every case, to prevent possible extension of the inflammation in a direction in which it is often overlooked.

The common methods of making the local applications are by an atomizer or spray, a nasal syringe or douche, gargle, insufflation, or by the use of a camel's-hair brush or a piece of absorbent cotton on a swab. The choice of method will depend upon the conditions to be met in a given case. Gargles are not very effectual. As a rule, the syringe is best for nasal diphtheria, and the spray is most useful for applying antiseptics or solvents to the false membrane, while the swab is of service when single stronger applications are indicated or where a piece of loosened membrane is to be detached. It is sometimes useful to combine several methods. A small syringe, with a small piece of rubber tubing slipped over the nozzle, serves well for cleansing the nose. The nurse should be instructed to apply the stream horizontally and very gently. A nasal syringe has been devised with a soft-rubber top which fits the nostril conveniently. In syringing the nose the head should be held well forward and the child told to breathe through the mouth. Should pain in the ears be complained of after the syringing, it must be abandoned, and the fluid may be poured in with a spoon or medicine-dropper.

If the nares are entirely occluded by thick membrane and secretion, they may be cleansed by a probe carrying cotton dipped in a little 50 per cent. solution of carbolic acid. This strong solution should only be used upon the false membrane. Afterward the nasal cavities may be kept clean by a saturated solution of boric acid, or one of ten minims of carbolic acid to the ounce of lime-water.

It is obviously unwise and unnecessary to attempt the use of a laryngoscope to facilitate examination. All applications to the nasal mucous membrane should be very mild and warm. Frequently warm local applications to the throat give more relief than cold, but many patients crave the cold and enjoy cold water, cracked ice, simple water-ices, ice-cream, etc.

In order to keep down a rapidly-spreading inflammation and an accumulation of false membrane, which would be inevitably fatal, it is absolutely necessary to adopt measures which at the time may appear severe. Thus in very bad cases local applications must be made continuously every half hour by day and every hour by night, and exceptionally even oftener, although they interfere with sleep. Sometimes patients become so fatigued that they will drop asleep while the application is being made, or they will bitterly complain of being so frequently disturbed. Experience teaches, however, that vigorous local treatment is the only means of preventing the extension of inflammation to the larynx and nares, and many lives may be saved by firmness in this respect. When children once pass into a septic coma it is wellnigh impossible to arouse them. It taxes the physician's best judgment to avoid the danger of exhausting the patient's strength by too energetic local treatment, and the even greater dangers of sepsis and occlusion of the air-passages by rapidly-spreading false membrane.

If there be an accumulation of false membrane already formed in the larynx,

an emetic of turpeth mineral will sometimes enable the child to discharge a considerable portion of it, with immediate relief to the accompanying dyspnœa.

Much tact is required in the management of young infants, who are apt to be greatly alarmed by attempts at local treatment. In such instances firmness and gentleness will often overcome objection. It must be remembered that hard crying with deep inspiration may loosen a bit of false membrane, which is carried into the larynx or trachea to set up a fresh infection with fatal result. Often, however, the infants are too ill to cry violently.

Any application should be abandoned which tends to derange the stomach. Very young children, as a rule, will not tolerate the spray, and they are unable to gargle. In such cases the tincture of the chloride of iron may be given in doses of five, ten, or fifteen minims in glycerin and peppermint-water every half hour. A child a year old will take a drachm or more in the course of twenty-four hours with benefit. Swallowing the frequently repeated doses serves the purpose of local application fairly well, for a certain amount adheres to the diseased mucous surface. A useful formula for a young child is—

℞. Tinct. ferri chloridi,	f℥jss;
Glycerini pur.,	f℥ss;
Aquæ menth. piper.,	f℥j;
Aquæ,	q. s. ad f℥iv.—M.

Sig. Give f℥j every half hour.

The iron acts beneficially by constricting the blood-vessels, and possibly also the lymphatic vessels, and by diminishing absorption of septic products. It is also antiseptic, reduces local hyperæmia and swelling, and toughens the membrane already formed.

The chlorate of potassium is sometimes given with iron, but it is apt to impair digestion, and, in large doses, may excite hæmaturia. Since the kidneys are often more or less inflamed, this remedy should be used with great caution.

The pseudo-membrane should never be torn off or rudely removed, so as to expose a fresh bleeding surface to reinoculation and extension of the inflammation, but a good deal of loosened membrane may carefully be dislodged by a bit of dry flannel fastened to a strip of wood and used as a probe. After the pseudo-membrane has been coagulated and hardened by topical applications it is often freer, and can be more readily removed, but unless it comes off very easily it should be let alone. Loosened membrane, unless it be removed or coughed out, is liable to be swallowed or inhaled.

If it becomes necessary to remove loosened false membrane or make a local application, a kicking child may be rolled in a blanket and held up by the nurse while a swab is used. A teaspoon or a medicine-dropper may be employed to pour fluid into the nostrils, instead of the rubber tubing and syringe.

A great number of local applications are made with a view of hardening and freeing the pseudo-membrane and checking its extension. Such are the following solutions: tincture of the chloride of iron, 10 per cent., in glycerin and water (applied with a brush); common salt, ⅔ per cent.; salicylic acid, ¼ per cent.; creolin, 1 per cent.; creasote; resorcin, 10 per cent., in glycerin; menthol; carbolic acid, 1 per cent.; boric acid, saturated aqueous solution; corrosive sublimate, 1 : 2000 or 3000; potassium permanganate; sodium hypophosphite.

Applications of strong solutions of silver nitrate, hydrochloric acid, or carbolic acid to the diseased mucous surface have been extensively tried, but such harsh measures are now universally condemned; for, if local applications are made too strong, they weaken the neighboring healthy mucous membrane and render it liable to infection. Nothing caustic or irritating should ever be applied, for, although such substances may temporarily destroy the membrane, it soon re-forms over a larger area, with increased inflammation and tumefaction, and deep sloughs may follow.

Vapors of turpentine, eucalyptus, carbolic acid, etc. are sometimes employed about the room and for the patient to inhale, but they are of doubtful efficacy. Considerable relief is often obtained by the use of steam generated in a "croup kettle" or in an ordinary kettle, the steam being directed under a sheet arranged as a hood over the patient's head. Lime-water or eucalyptol is sometimes added to the vapor. The steam favors suppuration, and the false membrane is loosened thereby. Such inhalations are especially valuable in the laryngeal form of the disease.

The continued use of a corrosive-sublimate spray may over-stimulate the membrane and produce too much mucous secretion. Should this be the case, it must be employed less frequently and in alternation with some less irritating application.

Single applications of corrosive sublimate on a swab may be used in the strength of 1 : 1000, but the spray should be diluted to 1 : 2000 or 1 : 3000 and used with care. For young children it should be still weaker—1 : 5000 or 1 : 10,000. A little common salt is often added. Jacobi recommends the use of a spray containing a grain of corrosive sublimate to the pint, with a drachm of table salt added.

In young infants there is some risk in the free local use of corrosive sublimate, because it is difficult to estimate how much is being swallowed and absorbed. In such cases salicylic acid, 1 : 1500, makes a less injurious wash for the nasal cavities. A saturated solution of boric acid in water is also serviceable as a douche; as is common salt, one drachm to a pint of warm water.

For solvents of the mucous membrane preparations of pancreatin or papayotin, 1 : 20 in water, are used with benefit as sprays or topical applications. It is somewhat doubtful whether these substances really dissolve the false membrane to any great extent, but they do seem to check its extension

and to cleanse the surface. A good spray for use when corrosive sublimate cannot be safely employed consists of—

℞. Acidi carbolici, ♏xij;
Liquor calcis, f℥iv.—M.
Sig. Apply with an atomizer every half hour or hour.

Recently hydrogen peroxide has been extensively tried, with excellent results, as a spray in a 5 per cent. or 10 per cent. aqueous or glycerin solution of the 15-volume-strength solution. This does not dissolve the membrane, but it acts as a cleansing and disinfecting agent. It does not corrode, nor does it injure, sound tissue when properly diluted.

Local applications to the outside of the throat are useless to relieve the inflammation of the mucous surfaces. If the cervical glands are swollen and painful, they may be relieved by application of belladonna liniment or iodoform ointment, and an ice-bag. Should they suppurate, which is unusual, they must be actively poulticed and then incised.

When the eye becomes inflamed the opposite one should be protected from infection by a pad and adhesive plaster. The constant application of cold to the lid and the use of a saturated solution of boric acid are the best remedial agents.

Intubation and Tracheotomy.—Intubation of the larynx is a method devised by O'Dwyer of New York. It consists in the operation of inserting a small gold-plated tube between the vocal cords and leaving it there, so that air can freely pass in and out, the channel previously blocked by false membrane being kept open by the tube. The tube is carefully adapted to be held between the vocal cords, and various sizes are made to fit any larynx. The tube is passed into the larynx by an ingenious holder, which releases it when in position, and an instrument is also employed for its removal for the purpose of cleansing it, extracting plugs of membrane, etc. The holder is grasped in one hand, while the index finger of the other hand serves as a guide for the tube, a gag being usually necessary to protect the finger. As a precaution against the tube being coughed up and swallowed a thread is tied to it, which may be brought out of the mouth and fastened.

The practice of intubation has many and decided advantages over tracheotomy. It does away with the necessity for tracheotomy, a surgical operation which the patient's family usually abhor, if they do not actually forbid it. It is speedily performed by one skilled in the use of the instrument, and it can be done at a moment's notice. The tube can be readily cleansed and reinserted. The relief afforded is as instant as that of tracheotomy, and the operation of inserting the tube is more quickly performed and it may be done earlier. The tube irritates the throat no more than a tracheotomy-tube. In case of recovery the tube is more promptly removed than a tracheotomy-tube —in five or six instead of ten days—and there is no wound left to close.

The disadvantages which it may have are—first, that it is said to push the

false membrane from the larynx down into the trachea, whereas the tracheotomy-tube passes in below the larynx; second, it is sometimes coughed out by the child, who suffocates before it can be replaced; third, it may be difficult for the child to swallow without drawing food into the trachea. This trouble is chiefly confined to fluids and not to semifluid or solid food. With proper precautions, however, these shortcomings can be reduced to a minimum.

If necessary, rectal alimentation may be temporarily resorted to, or a small œsophageal tube may be passed, though the latter method is apt to be too irritating. Many children can learn to swallow well with a little practice after the tube is inserted.

The operation of tracheotomy cannot be here detailed, as it belongs to surgery.

After either intubation or tracheotomy the tube should be watched constantly with great care, lest it become occluded with mucus or pseudo-membrane and cause suffocation. If a tracheotomy-tube be employed, it should be covered by a compress of cheese-cloth or other light material soaked in a warm antiseptic solution. The tube should be fitted with a proper attachment, so that a piece of rubber tubing can be fastened over it, and with the aid of a hard-rubber syringe mucus may be sucked out of the tube by the nurse. A soft-rubber catheter should also be in readiness to pass through an obstructed tube when necessary to clean the passage. Jacobi recommends a long feather for the same purpose, as bits of membrane and mucus are apt to adhere to it.

Symptoms which make either tracheotomy or intubation urgent are quickened, stertorous respiration, unremitting dyspnœa, cyanosis, increased restlessness and anxiety of the patient. Aphonia with difficulty in both inspiration and expiration indicates the presence of false membrane in the larynx (Jacobi). There is also marked falling in of the suprasternal and epigastric regions accompanying the inspiratory efforts.

If the lungs are already involved or if the pseudo-membrane has passed down beyond the larynx, opening the trachea is of no avail. Either operation may be performed when there is no real hope of recovery, simply to save the patient from a very distressing death from suffocation.

Unless immediate relief follows tracheotomy or intubation when done early, there is strong probability that the trachea and bronchi are already invaded by the inflammation.

A statistical comparison between the mortality of tracheotomized and intubation cases is of little value, for tracheotomy is usually left as a last resort, and both methods are only employed in laryngeal diphtheria, which is exceedingly fatal in young children. Both methods serve to prevent the child's dying of suffocation, and a small percentage are actually saved. In 1890 statistics from various sources were collected of 2368 cases in which intubation had been practised, with a recovery of 27.3 per cent. The mortality from tracheotomy, however, is quite as high. Fully 95 per cent. of the cases of laryngeal diphtheria in children and infants die unless relieved by intubation or tracheotomy.

Treatment of Special Symptoms.—Paralyses of the pharyngeal muscles, soft palate, or tongue may interfere with deglutition and articulation. Attempts to swallow fluid result in its regurgitation through the nose. Such patients must be fed by an œsophageal tube. For the paralyses strychnine and other tonics, such as quinine and iron, are of service. Massage or a mild electric current, galvanic or faradic, may be used, but the latter is of doubtful efficacy, for most cases recover of themselves. If there be cardiac paralysis, absolute quiet in a recumbent position must be enjoined, and strychnine and brandy must be given hypodermically, with ammonia or camphor internally.

If the temperature be high, sponge-bathing of the surface with cold water and alcohol in equal parts is useful. Dyspnœa is sometimes relieved by placing the child in a warm bath for a few minutes.

Internal Remedies.—The internal use of corrosive sublimate in diphtheria has greatly gained in favor during the past few years, and it is now accepted as the best remedy. It seems to be particularly well tolerated by children having diphtheria, and they are soon able to take large doses, gradually increased. As much as one-fortieth of a grain may be given every two hours to a child three or five years old. The rule is to administer small, frequent doses, carefully watching the effect, and the drug must be diluted in water or milk to 1 : 5000. Jacobi states that a child a year old may take as much as half a grain in the twenty-four hours, divided into small doses. Should stomatitis or any indication of intestinal derangement, such as irritable diarrhœa, occur, the drug must of course be stopped at once; and in every case its effects should be most carefully observed, and the doses should be very small at first until it is ascertained that they are well tolerated.

The method of using the tincture of the chloride of iron has been detailed above, for the benefit derived from it seems quite as great locally as in any other way. It is commonly combined with glycerin, diluted in water, and is given in large, frequent doses.

Stimulation and *cardiac tonics* should be employed early in the disease, and constantly, for there is much more hope of preventing heart-failure than of counteracting it when present. Digitalis, strophanthus, caffeine, ammonium carbonate, strychnine, camphor, and alcohol are the remedies most favored. The citrate or sodo-benzoate of caffeine, when the urine is scanty, is useful for its diuretic effect.

Alcohol is demanded early in nearly all cases. In those which commence with severity it should be given at once to prevent cardiac failure, in the form of diluted whiskey or brandy. Infants tolerate brandy well. An infant may be given twenty drops of brandy, or a child of four or five years from one to two drachms, every hour or two, or oftener if very feeble. When swallowing is difficult or if the stomach be enfeebled, stimulants must be given by the rectum. Half an ounce of brandy in a little milk may be administered by this means every two or three hours to a child of four or five years.

The *diet* should be light and nutritious. During the active stage of the disease, while there is fever and if any difficulty in deglutition exist, the food

must be fluid, or semi-solid if more easily swallowed, and should consist of thick broths, beef tea, egg-albumin, egg-nogg, milk, milk-punch, etc. Ice cream and wine-jelly may be given. Rice and gruels are also serviceable. During convalescence patients are anæmic and require iron and the bitter tonics. Great care should be exercised to prevent sudden exertion until the heart is restored to its normal strength.

After recovery the patient must be sponged with a disinfectant, and must have the hair thoroughly shampooed. All clothing worn in the sick-room should be disinfected by steam or otherwise. The bed-linen and under-clothing must be boiled and soaked for five hours in a 1 : 2000 solution of corrosive sublimate or for twenty-four hours in a 2 per cent. solution of carbolic acid. It is customary for health-boards to burn sulphur in the bed-room, using three pounds to every thousand cubic feet of air-space, then airing the room thoroughly for a day or two. The sulphur has been shown to be of somewhat doubtful efficacy (Prudden), and it has little effect unless abundant moisture or steam be present; and this is seldom practicable in a private dwelling.

The room should be thoroughly washed and scrubbed with corrosive sublimate, 1 : 1000, or carbolic-acid solutions, and the walls should be rubbed down with bread-crumbs. If the case has been of malignant type, it is better to repaper the walls and repaint the ceiling and woodwork before any one, especially a child, occupies the room. Furniture must be rubbed with cloths wet in a 5 per cent. solution of carbolic acid.

The infectivity of a patient may outlast the local symptoms, and it is a safe rule to keep children away from school for at least a month after the disappearance of all local symptoms. Diphtheritic paralysis is known to occur in animals and man some time after all local symptoms have gone, showing that the disappearance of the membrane must not be invariably taken as a criterion of complete recovery.

ERYSIPELAS.

By W. GILMAN THOMPSON.

Definition.—Erysipelas is an infective disease, caused by a specific micrococcus and characterized by high fever, an intense local inflammation of the skin and adjacent mucous membranes, with a tendency to rapid extension and to become contagious, especially in the presence of wounds.

Synonyms.—St. Anthony's fire; Érysipèle (Fr.); Erysipel, Rose, Rothlauf (Ger.).

Etiology.—Erysipelas is divided clinically into two forms: 1. Idiopathic or "medical" erysipelas, involving chiefly the face and the head; 2. Traumatic, originating at the site of a wound anywhere upon the surface of the body. For the peculiar features of the latter the reader is referred to works upon surgery.

These two varieties are now generally admitted to be due to a common cause—namely, the presence of a specific virus, a micrococcus, which enters the body, develops an intense local inflammation in the integument around the point of entry, and forms poisonous products, called toxines or ptomaïnes, which pass into the systemic circulation and excite febrile and other constitutional symptoms. The micrococcus, *Streptococcus erysipelosus*, has been described by Fehleisen and Ziegler. It is always obtainable from the inflamed tissues. It presents a chain-like form, gives characteristic gelatin cultures, and when inoculated in rabbits and in man develops true erysipelas. It has been argued that in idiopathic erysipelas the virus may gain access to the body otherwise than by inoculation, because (*a*) the constitutional symptoms occasionally precede the local manifestations, and (*b*) a cutaneous abrasion is not always discovered. On the other hand, it is true that careful search usually shows that the inflammation has commenced at the site of a slight abrasion, either at the angle of the mouth or eye, or at the septum or ala of the nose, or at a fissure behind the ear, or at the auditory meatus, or sometimes on the mucous membrane adjoining a carious tooth or where an eczematous papule has been scratched away. In other cases, where such abrasion first admitted the virus, the local swelling may obscure the point of entry, or the abrasion has healed before the symptoms are observed. Moreover, it has been suggested that the local inflammation may really be coexistent with the earliest constitutional symptoms, but the products of the inflammation are removed at first by the blood and lymphatic capillaries as fast as they are formed, only giving rise to local signs when they become excessive. Hebra and those of his school have held that erysipelas is a local disease, and that

all the symptoms are referable to the intense dermatitis. The majority of observers, however, class erysipelas as a general or constitutional disease having some points of resemblance to specific fevers. In infants the disease often commences at the umbilicus, genitalia, or the site of vaccination. In puerperal women it invades the genitalia, spreads down the legs, and involves the uterus in septic inflammation, which attacks the extensively eroded surface. Certain persons possess a constitutional predisposition to the disease and have repeated attacks.

Age, *season* of the year, and *climate* do not particularly influence the susceptibility to the disease, nor does *sex*, apart from the puerperal state of women, in which condition it is especially virulent and infectious. Though not necessarily originating through such conditions, the extension of erysipelas is greatly favored by bad hygienic surroundings, filth, and overcrowding. It has often existed for months in a crowded district or a hospital ward, and it does not always spread far even under concentration. The traumatic variety is more apt to be transmitted to puerperal patients than the idiopathic.

Morbid Anatomy.—There is at first hyperæmia and intense redness of the skin, followed by an infiltration of lymph and corpuscles into the cutis and subcutaneous connective tissue. The cells are round, granular, and occur in scattered form or in aggregations. The inflammation may be slight, amounting only to simple erythema, or much more severe, involving the subcutaneous fat. When the skin is loose there may be extensive œdema. The cellular elements of the rete mucosum and the derma are softened and swollen with serous exudate. Vesication frequently occurs. Chains of micrococci are found in the rete Malpighii and in the lymph-spaces. The capillaries and lymph-vessels contain an excess of corpuscles. The inflammatory process may go on to the formation of pus, which is commonly diffuse, but sometimes results in abscesses or gangrene. The pus-cells exhibit more or less fatty degeneration. Congeries of micrococci are found in the lymph-spaces and vessels, around which there is more or less necrosis. When recovery begins, the serum is quickly absorbed, the skin becomes flaccid or shrivelled and pale, the leucocytes disappear rapidly, and the granular débris in the superficial layers of the cutis is soon absorbed. There are no typical visceral lesions. If the fever has been high, there may be local congestion hyperæmia, or parenchymatous degeneration of viscera—such lesions as are incident to the fever or blood-poisoning. The blood becomes thin, dark, and coagulates poorly. There may be acute œdema of the brain. In mild cases the inflammation of the skin disappears so completely after death that it may be impossible to detect it.

Symptomatology.—The symptoms are both local and constitutional.

The local symptoms are typical. The skin surrounding an abrasion or fissure becomes rapidly hyperæmic, red, swollen, tense, smooth, and shiny. It feels hot to the touch, and the patient complains of burning pain or itching. The margins of the inflamed patch are irregular, but distinct, and appear elevated both to touch and sight, and form an abrupt contrast with the normal

skin beyond. Such an inflamed patch extends rapidly, until within a day or two it involves nearly all of the face and in some cases the scalp, even reaching down to the neck and shoulders. The maximum inflammation is usually attained by the third day.

In very mild cases the inflammation does not progress beyond a simple erythema, and only a small part of the face may be affected. The crimson color at first fades momentarily under pressure. In more severe cases the inflammation extends by advancing the elevated periphery or by radiating œdematous lines which pass into the normal skin. Rarely, isolated patches, commencing as round elevated spots near the primary inflammation, coalesce with it. The raised spot becomes more and more œdematous, especially where the integument is loose. The eyelids are so swollen as to obscure vision, and the face becomes unrecognizable. Small vesicles and blebs form on the surface and coalesce to form large bullæ, filled with serum, which is at first clear, but may become purulent. This serum is capable of reproducing the disease when inoculated in man or animals. At this stage recovery may occur. The color of the skin grows more natural, inflammation subsides, the œdema slowly disappears, the blebs are absorbed, rupture, or dry to crusts, and after five or six days of inflammation the skin undergoes thorough desquamation and becomes quite normal.

In worse cases the swollen skin becomes soft and boggy, pits on pressure, and the surface is more or less livid, bullæ may become sanguinolent, and diffuse subcutaneous sloughs and superficial gangrene appear or circumscribed abscesses form. The spread of the inflammation is somewhat affected by the character of the skin: it often stops short at a deep fold like the naso-labial or at the line of the hairy scalp, and it often is arrested at the chin. If lymphatic glands are near, lines of hyperæmia may be seen extending toward them from the inflamed area.

When the disease affects the face and head the adjacent mucous membranes are involved; the lips and gums are swollen and red; the nares and conjunctivæ are covered with mucous secretions, forming dry crusts; the tongue is dry and fissured or swollen; and the pharynx congested. Occasionally the inflammation spreads rapidly over the entire body (erysipelas migrans, "erratic" erysipelas), producing enormous swelling of the extremities. In such cases the skin presents simultaneously the various stages of the inflammation. The cervical glands often become painful and swollen.

Constitutional Symptoms.—The constitutional symptoms may precede the dermatitis by a few days or hours, or they are coincident with it. They are the symptoms common to the febrile state. There is usually a sudden onset with rigors or a chill, followed by a sharp rise of temperature to 104° or 105° F., accompanied by prostration, nausea, and perhaps vomiting, constipation, and headache. In mild cases there may be so little malaise that the first thing noticed is the local inflammation. Sometimes the patient complains of a sore throat. The fever is usually remittent, but is irregular, and the temperature may be lower in the evening than in the morning. It tends to remain high

for several days. Occasionally the temperature drops to normal, and soon rises again to 104° or 105° F. Remissions in the fever may be accompanied by sweating. The fever and other constitutional symptoms advance or abate with the local inflammation. If the latter extends or increases in intensity, the pulse becomes rapid and feeble, possibly intermittent; the tongue is dry and brown; the temperature continues high, and may reach 106.5° or 107° F; the urine is scanty and moderate, albuminuria ensues, and the patient rapidly passes into a typhoid condition with delirium, which may become maniacal; subsultus, involuntary evacuations, often with diarrhœa and extreme prostration, with finally coma and death. Delirium often occurs without the typical typhoid condition.

Course.—Facial erysipelas and the idiopathic cases in general run a favorable course, as a rule, and subside gradually. Occasionally the subsidence is by crisis. Convalescence will naturally depend upon the duration of the malady, severity of the complications, and the age of the patient. In ordinary cases it is fairly rapid.

Reinfection apparently occurs at times in the same individual. In its migrating form erysipelas will gradually attack new areas, while those first invaded are completely healed. In this way recovery may be greatly retarded, with intervals of normal or even subnormal temperature.

Mild cases subside in two or three days. Cases of ordinary severity with complications last about six or seven days. Less frequently the disease is prolonged for a fortnight, and cases have been reported, especially of the migrating variety, which have lasted for many weeks.

As a whole, the disease is less fatal and of somewhat less frequent occurrence now than formerly. The majority of the idiopathic cases recover completely. Death results from the primary toxic effects of the disease, from exhaustion and cardiac failure, or from gangrene and other complications, inducing a general septic condition.

Complications.—Pneumonia is the most frequent complication. Purulent meningitis, peritonitis, and inflammations of other serous membranes, like the pleura and pericardium, have been observed as complications, but they are comparatively rare, and are due to blood-poisoning from the local inflammation rather than to its extension. After violent maniacal delirium the autopsy does not always reveal the extension of inflammation through the orbit to the meninges or brain. The inflammation may invade the larynx, extending from the mouth, and it may pass down toward the lungs and be complicated with fatal pneumonia. Œdema of the larynx or epiglottis may necessitate tracheotomy. As a rule, the eyes escape, but they may become involved in the inflammatory process and more or less permanently injured. Gangrene occurs in the loose tissue of the eyelids, as well as in the scrotum, vulva, and occasionally in the mouth or pharynx and elsewhere on the body. The writer saw it appear on both feet in a non-fatal case. It is usually superficial. The joints are sometimes involved by extension of the inflammation to their serous surfaces or by metastatic abscesses, but this is more common in surgical

cases. Adenitis and lymphangitis are less infrequent accompaniments of erysipelas.

Sequelæ.—The sequelæ of erysipelas are few and comparatively unimportant. The hair falls if the disease has invaded the scalp, but returns again. Scars may be left by extensive sloughs. Repeated attacks cause permanent induration of the skin of the eyelids, nose, or ears, the deformity amounting almost to elephantiasis (Virchow). Individuals who have had one attack are apt to have another after an interval. Some persons have an attack almost every year. They are apt to be the subjects of fistulæ, varicose or other ulcers, ozæna, and the like—conditions which maintain a favorable seat of invasion by inoculation. Where erysipelas has involved previously existing patches of chronic eczema or lupus, the latter sometimes show decided improvement when the invading disease has gone. After erysipelas has passed over any portion of the skin, it leaves it softer and finer than before invasion. Exceptionally, there may be anæsthesia or hyperæsthesia of the inflamed area, due to alterations in the structure of the cutaneous nerves. Neuralgia sometimes ensues. Abscesses may form in the eyelids, or there is rarely keratitis or optic neuritis.

Diagnosis.—A typical case of erysipelas is not likely to be mistaken for anything else. In its early stages it has been confounded with urticaria, eczema, and other skin affections. In pemphigus fever is absent and the bullæ do not have an inflamed base. The important diagnostic features of the erysipelatous eruption are its elevated, clean-cut margins, its peculiar method of spreading, the swelling of the affected skin and subcutaneous tissue, with tendency to form blebs and bullæ, the bright-red, circumscribed area of inflammation, which contrasts with the normal skin adjoining, and the tendency to exhibit a definite relation between the severity of the eruption and the height of the fever.

Prognosis.—Erysipelas occurring in previously healthy persons runs a favorable course in a large majority of cases. The prognosis is bad in infants and very old persons, in alcoholic subjects, and in those who are debilitated from long-continued wasting diseases of tubercular or malignant character. Occurring in the puerperal state, the disease constitutes a very grave complication, and frequently ends fatally. As a general rule, the prognosis is much less favorable in the traumatic variety than in facial erysipelas. As a complication of Bright's disease, erysipelas is greatly to be dreaded. If the disease be epidemic in a crowded habitation, it becomes very virulent. The gangrenous form is very fatal from sudden collapse.

Treatment.—Mild cases of facial erysipelas recover spontaneously without any treatment. There is no specific remedy for the disease, and attempts to abort it by either local or general treatment, while apparently successful at times, fail completely in the majority of cases. The indications for treatment are—

I. To prevent the spread of the contagion to others;

II. To keep the patient comfortable and to prevent suffering from the local inflammation;

III. To support the strength by stimulants and nutritious diet;

IV. To deal promptly with complications.

I. *To Prevent the Spread of the Contagion to Others.*—A patient having either variety of erysipelas should be isolated, and those in attendance should keep away from puerperal patients and such as have open wounds, and they should carefully protect any abrasions on their own persons, and maintain absolute cleanliness of the hands. All dressings used about the patient should be destroyed by fire; his bed-linen and clothing should be disinfected as in the case of any contagious exanthem, and any instruments used about him should be most carefully disinfected afterward. If the disease develop in a hospital ward among surgical cases, the patient must be isolated and the ward emptied, thoroughly fumigated, and disinfected by washing with corrosive-sublimate solution. Facial erysipelas has been treated in a medical ward without its spreading to other medical patients, but it is always best to isolate such cases.

II. *Local Treatment*—The local burning and pricking may be relieved by mild astringent and soothing applications. Vaseline oil containing 5 per cent. of carbolic acid, linseed oil with lime-water (carron oil), are useful for this purpose. When such applications are made, the skin should first be gently washed with soap and warm water, and the preparation then slowly rubbed in around the inflamed area, the friction being made toward the affected surface, not away from it.

Among the various topical applications the writer has obtained the most relief by the use of a mask of soft lint cut to fit the face (with holes for the eyes and nose), which is frequently wrung out in a cold lead-and-opium wash, such as

℞.	Liq. plumbi subacetatis,	f℥jss;
	Tinct. opii,	f℥ss;
	Aquæ,	q. s. ad f℥viij.—M.
Sig.	For external use.	

If the skin be very tense and painful, it is sometimes more benefited by a hot poultice than by cold. If the tension be extreme and gangrene seem imminent, small linear incisions may be made in the skin, but this is to be avoided if possible. Dusting powders relieve the burning, but have the disadvantage of obscuring the outlines of the eruption. In order to limit the spread of the cutaneous inflammation caustics have been vigorously used, and even incision of the healthy skin has been tried with the hope that the advancing lesion would stop at such barriers. Strong solutions of carbolic acid, caustic alkalies, chloride of iron, iodine, turpentine, nitric acid, ointments of mercury and zinc, powders of iodoform and resorcin, subcutaneous injections of phenic acid, and the actual cautery, have all been tried repeatedly and in turn abandoned. At times they seem to temporarily hold the march of the disease in check, but more often it defies their limits, and is even aggravated

by the irritation of the applications. Koch recommends painting the inflamed surface with a thin layer of the following mixture:

℞. Creolin, 1;
Iodoform, 4;
Lanolin, 10.—M.
Sig. For external use.

Elastic compression of the integument surrounding the inflammation has been employed in some instances to limit the inflamed area; and contractile collodion painted over the surface is another application which has been used, but both of these means, while they are quite harmless, are of uncertain advantage.

If the cavity of the mouth be invaded, gargles of alum or boric acid should be used.

III. *Stimulants and Diet.*—The diet should be nutritious and adapted for easy digestion. If the fever be high and there be a tendency to vomit, nourishment should be given in small quantities and at very frequent intervals, as every hour. In ordinary cases peptonized milk, beef juice, egg-nog, milk punch, and light starchy foods are given. Stimulants should be prescribed freely in cases which begin with severity or in milder cases where the symptoms are prolonged. Alcohol is well borne, and 18 or 20 ounces of whiskey or brandy given in twenty-four hours may be perfectly assimilated without toxic effect. If the pulse be feeble or irregular, ammonium carbonate, camphor, digitalis, or strophanthus should be added to the brandy. Patients in whom the disease spreads rapidly over the body require particularly energetic stimulation. The tincture of the chloride of iron has long been employed in England and the United States, and many believe that it is a specific in erysipelas. This is not the fact, although it seems to benefit some cases. It is usually well tolerated, even if given every two hours, in half-drachm doses in glycerin and water.

IV. *Treatment of Complications and Very Severe Symptoms.*—The delirium is best controlled by hypodermic injections of morphine, or, if it become violent and maniacal, by the one-hundredth of a grain of hyoscine hydrobromate, provided that the heart-action be not too feeble. An ice-cap should be kept on the head. The temperature, when high, is to be controlled by cold alcoholic sponge-baths or wet packs and compresses. Antipyretic drugs should be avoided on account of the depression which they cause. Abscesses should be poulticed and evacuated early. Gangrenous areas must be carefully dressed with disinfectants.

After recovery from the inflammation the patient should be given a warm bath containing sodium bicarbonate, and then the surface previously inflamed should be sponged with a 1 : 30 solution of carbolic acid. During convalescence great care should be taken to avoid excesses in food or drink or over-exertion. A course of tonics, such as iron and cinchona or nux vomica, may be continued

until the patient's strength is fully restored. If convalescence be greatly protracted, a change of air and scene is indicated.

Recent experiments have shown that a curious antagonism apparently exists between erysipelas and certain forms of new growths. Inoculations of pure cultures of Fehleisen's erysipelas coccus have been made upon cases of lympho-sarcomata, with the effect of reducing their size when the erysipelatous inflammation subsides. According to Kleeblatt, the cocci multiply within the tumor and break down and destroy its cells. The growth is considerably diminished in size, and a temporary improvement results, although the tumor may return again.

MALARIAL FEVERS.

By W. GILMAN THOMPSON.

Definition.—Malarial fevers constitute a group of miasmatic, non-contagious, paroxysmal fevers in which the principal lesions are found in the blood and spleen.

Synonyms.—Intermittent, Periodic, Marsh, Swamp, Miasmatic, Malarial, or Paludal Fever; Ague; Fever and ague; Chills and fever; Fièvre paludiene (Fr.); Wechselfieber (Ger.). The term "malaria" is applied to a miasm or poison which begets the fever.

The chief characteristic of a malarial fever is its periodic recurrence or exacerbation at definite intervals. Each exacerbation is termed a "paroxysm," and the typical paroxysm includes—(1) a cold stage; (2) a hot stage; and (3) a sweating stage.

On Malarial Fevers in General.

Types of the Malarial Fevers.—There are two primary types of malarial fevers: I. the intermittent; II. the remittent. The first is characterized by the entire absence of fever between the paroxysms—the second by the presence of more or less fever of a continued type which does not cease between the paroxysms.

The paroxysms may recur at different intervals, and the principal malarial fevers are named in accordance with these intervals, as follows: I. Quotidian; II. Tertian; III. Quartan.

The "interval" of malarial fevers is the time intervening between the beginning of one paroxysm and that of the next. It is to be distinguished from the "remission" or the "intermission," which is the period between the end of one paroxysm and the beginning of the next.

In quotidian fever the interval is twenty-four hours, and the paroxysms return daily at the same hour.

In tertian fever the interval is forty-eight hours, and the paroxysm returns every other day at the same hour, or on every third day, according to the Latin method of counting the day of commencement of the fever as the first day; hence the name of "tertian" fever.

In quartan fever the interval is seventy-two hours. Other malarial fevers are described as quintan, sextan, heptan, and octan, the names denoting the interval between the paroxysms, but these varieties are so rare as to be regarded rather as curiosities than as definite types of ague.

In ordinary cases of simple malarial fever of whatever type the parox-

ysms return at a uniform hour, and the intervals are consequently uniform. When, however, the disease is becoming more severe, the interval may be shortened by half an hour or an hour or two, and this is called an "anticipating" fever. If, on the other hand, the interval is similarly prolonged, it is called a "postponing" or "retarding" fever, and the disease is becoming more mild. This occurs not infrequently under the influence of treatment.

The average length of the paroxysm in each type of intermittent fever is as follows: In quotidian intermittent fever it is ten to twelve hours; in tertian, six to eight hours; in quartan, four to six hours. It is uncommon for one of these types to alter to another, but the remittent fevers may gradually terminate by becoming intermittent.

Either of these types may be duplicated in the same individual, as, for example, giving double quotidian, tertian, or quartan. In the double quotidian two distinct paroxysms of unequal intensity occur every twenty-four hours.

In the double tertian paroxysms occur at the usual tertian interval, and milder paroxysms occur besides on the alternate days, but at a different hour from the others. There is thus a severe paroxysm on one day, a mild one on the following day, then a severe one again, and so on. There may be two tertian paroxysms occurring on the same day, followed by a free day, and then by two more paroxysms on the third day.

In the double quartan type there are alternating severe and mild paroxysms, with one free day between—*i. e.* there is a severe paroxysm on the first day, a mild one on the second day, then a free day, followed by a severe paroxysm, etc. There is even a triple quartan type, in which there is a paroxysm every day, the paroxysms corresponding at four-day intervals.

The tertian and quotidian types may be combined, giving two paroxysms on one day, one on the next, then two on the succeeding day, etc. Most of these varieties, excepting the first form of double tertian described, are so rare as to be mere curiosities.

The relative frequency of quotidian and tertian agues in the United States is approximately the same, the quotidian being very slightly more common than the tertian variety. The prevailing type in the tropics is usually quotidian, whereas in temperate climates it is frequently tertian. Quartan fever is rare in the tropics, and is infrequent at all times, seldom constituting over two per cent. of all malarial cases. In the same locality all these forms may coexist, or at one season quotidian ague may predominate, while tertian becomes most frequent in the next. With infants the quotidian type is the common form of ague.

The malarial paroxysm may begin at any time of day. In the larger number of cases quotidian and tertian agues begin in the forenoon. Quartan ague usually occurs either before or after noon. The milder types of ague are more regular in the hour of recurrence than are the more severe.

The foregoing classification of malarial intervals may apply either to the

PLATE I.

FIG. 1.

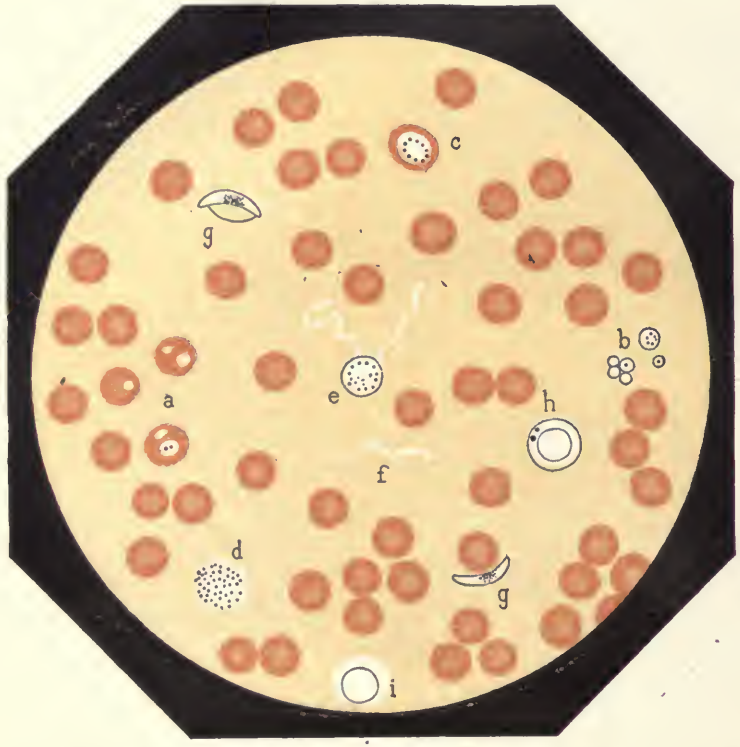


FIG. 2.

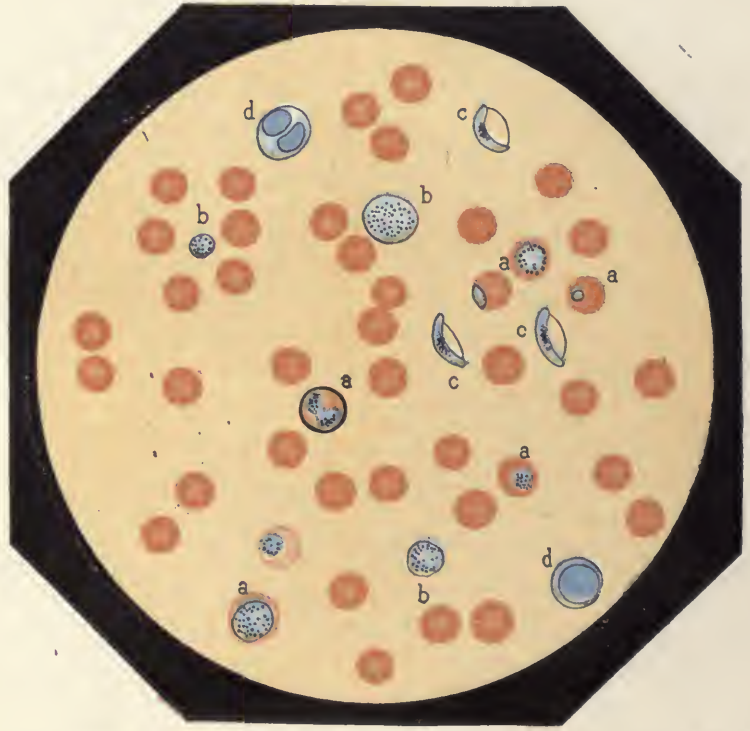


FIG. 1.—Hæmatozoa of Malaria in Fresh Blood (Laveran), × 500 diam.: *a*, Group of Normal Red Blood-cells, to which there adhere small spherical bodies; *b*, Small Free Spherical Bodies; *c*, Spherical Body of Medium Volume adherent to a red blood-cell; *d*, Spherical Body arrived at Complete Development and Free; *e*, Spherical Body with Two Flagella: *f*, Free Flagellum; *g*, Crescentic Body; *h*, Melaniferous Leucocyte; *i*, Normal Leucocyte.

FIG. 2.—Hæmatozoa of Malaria in the Blood, dried and stained with methylene blue (Laveran), × 500 diam.: *a*, Red Blood-cells, to which adhere spherical bodies in different degrees of development; *b*, Free Spherical Bodies; *c*, Crescentic Bodies; *d*, Leucocytes.

intermittent or the remittent type; thus there is a tertian intermittent fever, a tertian remittent fever, etc.

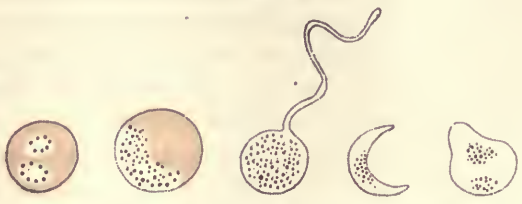
Etiology.—The cause of all malarial fevers is now supposed to be the presence in the blood of a specific organism of one or more varieties.

The Malarial Germ.—The malarial parasite is a protozoön, or a vegetable micro-organism, which inhabits the blood of man and certain of the lower animals. In the year 1716 an Italian physician, Lancisi, first attributed the origin of malarial fever to poisonous exhalations arising from marshes. In 1879, Klebs and Tommasi-Crudeli succeeded in isolating a germ—*Bacillus malariæ*—from the low-lying atmosphere over marshes and from the soil, which they inoculated into rabbits, with the result of producing a malarial paroxysm with enlargement of the spleen and pigmentation.

Laveran in 1880 closely followed with an elaborate description of the malarial germ, as discovered by him in human blood among residents of Algeria. The parasites which are obtained from human blood in the course of malarial fevers exhibit several varieties of form and size, and it is possible that there may be several species which are capable of exciting the distinct types of the disease, as tertian, quartan, etc. To what extent these various forms are related to one another as different stages of the same growth, or to what extent they represent different species, cannot in all cases be definitely decided at present. Laveran is inclined to think that the malarial germ is a single but polymorphic organism, and that the type of fever depends in part on the particular form of the germ, and also upon the condition of the patient, his tolerance of the germ, etc. Osler believes that different forms of the germ belong to distinct species, and that they are not all different stages in the development of one microbe.

Laveran describes the chief forms of his malarial hematozoön as consisting of (1) amœboid spherical bodies with nuclei; (2) crescentic shapes with nuclei; (3) rosettes; (4) flagellate bodies. (See Fig. 24). The flagellæ are very delicate. They are only to be found in fresh blood, and they are difficult to see unless

FIG. 24.



Forms of the Hematozoön of Malaria (after Laveran).

they are in motion. The other forms may be discovered in preserved specimens. His method of examination of a drop of blood was by rapid drying and fixation by artificial heat, followed by staining with a concentrated solution of methyl-blue or gentian-violet. (See Plate.) The germs may be contained in the blood-plasma or in the substance of the red blood-cells.

The name *plasmodium* has been given to the germ found in the red-blood disks. The observation of the malarial germs in human blood has been confirmed by Marchiafava, Osler, Councilman, James, Carter, and many other competent and skilled observers.

Councilman describes the flagellate bodies as being most common in blood aspirated from the spleen, although in acute cases they may sometimes appear in other situations. They exhibit from three to eight vibrating ciliæ.

In acute malarial fever the amœboid bodies are found occupying a certain number of the red blood-cells or adhering to them. They derive pigment (melanin) from these cells, and after undergoing a certain development and increase in size at the expense of the red cells, they contain this pigment in distinct granules and rods. During the paroxysm they must undergo segmentation. They vary in size, and some are as large as the red blood-disks. They are colorless and transparent.

According to the observations of Laveran, the crescentic forms of the germ are common in the blood in the quartan and irregular types of the disease and in malarial cachexia. Like the amœboid forms, they are transparent and colorless, but contain pigment-granules in the centre. They are somewhat larger than the diameter of the red disks, and the ends of the crescents may be joined by delicate lines. They are said to be more common in the autumn than at other seasons. Quinine acts upon the amœboid form of the parasite, and antagonizes or gradually destroys it. It has less effect upon the crescentic form. The leucocytes, or active white corpuscles of the blood, are believed to antagonize the parasites, and spontaneous recovery from malarial fever is attributed in part to their destructive action upon the germs. They may either act directly upon the germs or else destroy toxines produced by them.

Golgi believes that there are several distinct parasites which give rise to impaludism, and which are developed periodically or in a rhythmical manner. This theory accounts for the paroxysmal character of the symptoms of ague, which are believed to correspond with the various stages in the development of the germ. He considers that tertian, quartan, and double quartan fevers are due to germs having a period of development, or life-cycle, which corresponds with each separate variety of fever. Golgi's extensive observations upon the malarial organisms (recently published) are of sufficient interest and importance to be described somewhat at length.

In tertian fever he finds a plasmodium in the blood of patients several hours before the onset of the fever. This parasite is from one-fourth to one-fifth as large as the red blood-disks, and it exhibits more amœboid activity in its pseudopodia than is shown by the germs in other forms of malarial disease At first it contains scarcely any pigment. In the course of the second day the plasmodium increases in size and occupies the red disks at the expense of its substance, filling one-half or two-thirds of the entire corpuscle. The plasmodium appropriates the pigment of the red disk, and converts it into small rods and granules (Marchiafava and Celli). The plasmodium grows

more distinct and the red disk less and less distinct, until the latter becomes scarcely visible. Finally, all the pigment of the red disk is devoured by the organism, and the pigment-granules are grouped in its centre. Meanwhile, the organism shows a tendency to subdivide. Radiating lines mark off its substance into separate masses, and, the red disk having finally disappeared, the plasmodium liberates its pigment-granules and separates into a number of small new cells, which in turn attack other red disks, and the process is thus repeated while the paroxysms last. Golgi declares that the appearance of the radiating lines, with concentration of the pigment in the centre of the amœboid cell, is an indication that a paroxysm will occur at once. The administration of quinine makes it difficult, and finally impossible, to find the plasmodium in the blood.

In tertian ague Golgi has found the malarial organism to have a more distinct outline than in the quartan form. In the latter it is coarser, the pigment-rods and granules are thicker and long retained, and the entire development of the germ is more uniform than in the tertian fever. In tertian ague the parasite completes its cycle of development in two days, whereas in quartan it occupies three days. In tertian ague the red disks may be swollen, and they certainly are not shrivelled as in quartan fever. The two forms of germ have been simultaneously found in the blood in some instances. Malarial fever has been transmitted from man to man by inoculation with the blood of a malarial patient.

Marchiafava declares that in Italy the malarial parasites are subject to variations with the season of the year—that in summer they occupy the red corpuscles, and if the fever is pernicious they are associated with amœboid bodies, while in the autumn semilunar and filiform shapes are more common. The latter, he says, do not cause fever, but the amœboid bodies excite diurnal paroxysms. They are found also in winter and spring. Various hæmatozoa have been discovered in the blood of certain birds and other animals, especially in the blood of apparently healthy animals whose natural habitat is marshes, such as frogs, lizards, tortoises.

The germ of malarial fever taken from the human subject and injected into birds does not, however, grow, but disappears. Inoculated in rabbits, the germ produces an intermittent fever with enlargement and pigmentation of the spleen.

Mode of Infection.—The mode of malarial infection is not definitely understood. The miasm often seems to enter the system through the medium of inhalation, while at other times it apparently enters through the alimentary canal by means of contaminated drinking-water or other fluids which have been exposed to a malarial atmosphere. It is never transmitted directly from man to man or from animals to man, except by inoculation experiments, and it is not known that the germ is able to escape from the body in any manner. Presumably, it cannot do so, since it is only found in the blood. Concentration of malarial patients, therefore, has no effect upon the spread of the disease.

The exact relation of the germ to the symptoms and pathology of malarial fevers is yet under discussion. Whether the peculiar conditions of the nervous system and the alterations in the composition of the blood are occasioned by direct irritation of the plasmodium itself, or whether they are due to toxines produced by it, are questions which further investigation may solve.

It should be observed that there are still many competent observers who have not accepted the malarial plasmodium as the sole cause of paludal fever, and the whole subject is so new that it is impossible, as yet, fully to explain all the different clinical phenomena without some reserve. It is certain, however, that the germ exists in human blood during and between malarial paroxysms in number sufficient to exert powerful effects, for it can often be found in almost every drop of blood drawn. Moreover, it is never discoverable in normal blood or in disease other than some form of ague. In chronic forms of ague and in doubtful cases the presence of the germ in the blood is of great value in diagnosis.

Race.—It is sometimes stated that negroes are less susceptible to ague than are Europeans. As a matter of fact, race, apart from a certain degree of acclimatization, has but little influence upon the susceptibility to the malarial fevers. Natives of Central and Western Africa, although they may be acclimated at home, if they remove to another part of the country are quite as liable to contract malarial fevers as are foreigners living among them (Parke). In the United States negroes are somewhat less frequently affected by agues than are other persons. In India the mortality from malarial fevers among Hindus and Sepoys is as high as among the resident English soldiers.

Age and *Sex* have but little influence upon the liability to affection by malaria. It is observed at any age, and, although it is not very common among infants, it may occur in the first six months of life. Men whose occupation keeps them at work in draining marshes and upturning malarial soil are naturally more apt to be affected than women who stay at home, but with equal exposure the miasm shows no preference for one sex more than the other.

Locality.—Malarial fevers are endemic in all regions excepting in the frigid zones. They are more common and more intense in the tropics, and gradually become less common and less severe in passing away from the equator. In Europe they are centred in Italy, especially in the Roman Campagna, and in the marshes about the lower Danube. In the United States they are most severe in the South and South-west. Latent malarial fever will sometimes develop in a person who has left a malarial region for a locality where the disease never originates.

Season and Climate.—In tropical climates malarial fevers are always more or less prevalent, but in temperate latitudes they do not prevail in very cold weather, and they are most commonly developed in the early autumn, when moderate cold and dryness of the atmosphere follow a damp season. In the United States ague is more prevalent in the spring and autumn, less common in midsummer, and it is in abeyance in winter. In malarial regions exposure

to the hot sun, and afterward to a draught of cold air, often precipitates an attack of ague.

For the active development of the miasm a temperature of 60° F. or more is necessary. In temperate latitudes frequent exposure is often required in order to develop malarial fever. In tropical countries, where severe forms of ague prevail, the exposure is not necessarily prolonged in order to develop a bad attack. Malarial fevers are undoubtedly spread by winds. There are many localities in which the wind, changing to a certain direction and blowing across an infected marsh, will provoke outbreaks of fever. This is especially true in regard to the salt marshes near Rome.

The malarial germ has been found in the air to a limited extent. A heavy rainfall washes down the germs from the atmosphere. Malaria is more potent in infection at night. The reason for this is ascribed to the greater concentration of the cool night air after a hot day has rarefied and expanded the atmosphere; hence any floating germs would become more concentrated by night. Extensive fires destroy the miasm.

Soil.—The soil is regarded as the home of the miasm, and under certain conditions, especially when miasmatic earth is freshly exposed to the air, the germs are swept up by air-currents and carried off in the lower atmospheric strata. A fresh-water marsh which is occasionally overflowed by a high salt-water tide is particularly liable to breed malaria. As a rule, the malarial poison requires moist humid earth or a marsh for its development, combined with a warm temperature and exposure to the air. The miasm is occasionally found, however, under other conditions. It is met with on dry and hot sandy soil in Western India (Moore), and even on a rocky bed, as at Hong Kong (Maclean), or on the coral rocks of Southern Florida, where vegetation is very scanty. It is especially virulent along seacoast marshes in tropical and subtropical countries, and it tends to follow up the banks of rivers from their mouths. It flourishes in fresh-water marshes, but particularly in stagnant pools and in marshes near the sea tainted with brine and not cleaned freely by the tide. It has been known to develop in bilge-water in dirty vessels at sea.

Malarial fever does not always develop in proportion to the amount of decomposing vegetable matter present. Overturning the soil in a malarial region or removing the upper layers of earth usually precipitates an outbreak of malarial fever.

Malarial fever was almost unknown in the island of Mauritius up to the year 1865. At that time it was suddenly contracted by immigrants from India, who were employed in draining and filling a mud flat. Malarial fevers at times prevail throughout extensive regions for years until cultivation of the soil and drainage gradually destroy the home of the miasm. On the other hand, cultivated regions when abandoned may again become malarious, as they were before being occupied by man.

Elevation Above the Sea.—It is frequently stated that malarial fever does not occur at high elevations, but this is not strictly so. While it is true that it

flourishes along seacoast marshes, and that the most malignant types of fever are found in low-lying lands generally, it may be present elsewhere. Thus Parke speaks of encountering it near the Albert-Nyanza on a dry plain at 4800 feet above the sea-level, where there was more fever than in the lower damp forest, and he met with it again in Africa at an elevation of 10,000 feet. Ague is common in the elevated plateaus of Northern India.

Antagonism.—It has been claimed that a certain degree of antagonism exists between ague and phthisis. This, however, is not the case. Ague occurs in connection with a variety of chronic and acute affections, and when so doing exerts a distinct paroxysmal influence upon their course.

The lower animals may contract malarial fever independently of inoculation, but it is not common among them. It has been known to occur in horses, donkeys, and oxen.

Classification.—For convenience of description it is customary to subdivide malarial fevers into several groups, and clinically the different types are very distinct. It should be borne in mind, however, that anatomically, as well as etiologically, all malarial fevers are very closely related, if not actually identical, and the manner in which one variety of fever may occasionally merge into another, or in which two types coexist in the same individual, indicates rather a difference in degree or intensity of poisoning than multiplicity of diseases. The final outcome of the present study of the life-history of the malarial germs and their relations to the symptomatology of ague is anticipated with great interest in regard to the theory of the unity of malarial fevers.

Malarial diseases are usually classified under the following heads:

I. Intermittent Fever; II. Remittent Fever; III. Pernicious Intermittent Fever; IV. Pernicious Remittent Fever; V. Typho-malarial Fever; VI. Malarial Cachexia.

I. Intermittent Fever.

Morbid Anatomy.—The anatomical lesions of intermittent fever are few and simple. The spleen is engorged with blood during the febrile paroxysm. It is enlarged considerably in each attack, and at first it regains the normal size during the intervals. It soon fails to do this, and finally becomes permanently enlarged, and is called an "ague cake." In exceptional instances it may extend to the umbilicus or below it. The size of the spleen is not always an indication of the duration of the disease. In not a few cases of very long duration it is scarcely enlarged at all, while it may suddenly enlarge very much after one or two attacks. A rare lesion of malarial fever is rupture of the spleen, producing almost instant death from hæmorrhage into the peritoneal cavity. The spleen occasionally presents hæmorrhagic infarcts of various sizes. Its capsule is sometimes adherent.

The liver, like the spleen, is somewhat engorged during the paroxysm, and the hepatic area may be tender on pressure.

The heart is sometimes acutely dilated, though this accident is rare, and it

is more apt to occur in patients rendered anæmic and debilitated by protracted attacks of ague.

The blood during the febrile paroxysm contains fewer than normal corpuscles of both red and white varieties. The condition of the red corpuscles and their pigment has already been detailed in the description of the malarial germ.

The brain and spinal cord have not been found to present any typical lesions, but it is believed by many that certain of the prominent symptoms of ague are occasioned by functional disorder of these organs. In the severe types of malarial fever they are pigmented.

Incubation.—The length of the incubation period of intermittent fever depends upon the intensity of the miasm. The exact limits are not known, and are variously stated, from an hour or two up to twenty days. There are exceptional cases in which this period seems to last for several months. Eichhorst relates the case of a patient who resided for some time in a malarious region without having ague, but nine months after leaving the district and residing in a healthful locality he was attacked by intermittent fever.

During the incubation period there may be no symptoms, and the patient be in apparent health, or there may be certain indefinite prodromata, such as malaise, dyspepsia, constipation, dulness, sleepiness, irritable temper, etc.

Symptomatology.—Intermittent fever presents three distinct stages: (1) a cold stage; (2) a hot stage; (3) a sweating stage. Collectively, they constitute the malarial "paroxysm."

The cold stage is characterized by subjective sensations of cold and by rigors, while the thermometer records an elevation of the internal temperature; the hot stage is characterized by high temperature and the symptoms of pyrexia; the sweating stage, by profuse perspiration and the subsidence of pyrexia.

The individual symptoms of ague vary much in intensity in different cases and in different seasons and localities. The following is the history of a typical case of intermittent fever:

History of a Typical Case.—In an ordinary case the chill usually comes on gradually, and is preceded for two or three hours by a feeling of languor, dulness, yawning, and headache. In some cases, however, the chill begins very violently and suddenly. When it begins slowly the malaise is followed by a chilly feeling, commencing in the back and loins and gradually extending over the entire body. This is accompanied by muscular tremors, and sometimes by cramps. There are often nausea and vomiting, which are probably due to congestion of the stomach, and there is headache. The tongue is pale and coated. The chill becomes more and more violent; the teeth chatter; the surface of the body feels cold to the touch; the tips of the fingers, nose, and ears become livid; there is pronounced pallor of the features, which look shrunken and haggard; and the skin is roughened, dry, and presents the appearance of "goose-flesh," or cutis anserina, in a marked degree. There is great bodily discomfort, and the patient calls for more covering, but is not relieved by it. The muscular rigors become so violent as to shake the entire

bed, and the voice is feeble, or there is inability to speak on account of the constant chattering of the teeth. The respiration is hurried and short and there is præcordial oppression, and sometimes palpitation. The urine may be voided in increased quantity. During this time there is a gradual accession of fever. The surface temperature may be below normal, but the oral and rectal temperatures show an increase of two or three degrees. The peripheral vessels are contracted, so that the prick of the finger fails to draw blood. The blood passes in larger volume to the viscera, producing congestion of the more vascular internal organs. This congestion accounts for the gastric or enteric symptoms which accompany the disease, and it interferes greatly with the functions of the organs affected. The greater blood-pressure of the internal vessels in organs like the liver and spleen modifies their nutrition and the elimination of waste material.

The spleen is enlarged during the paroxysm, and the splenic area is often quite tender on pressure, and a sensation of fulness may be experienced there. The mind remains clear.

The cold stage continues for a varying time. It may be very mild and unaccompanied by rigors, lasting but a very brief period—ten or fifteen minutes—or it may be prolonged for an hour or two. There is no constant relation between the length or severity of the cold stage and that of the hot stage. The cold stage is said to be shorter in the quotidian than in tertian ague, but with the hot stage the reverse may obtain.

The average duration of the chill is from one-half to three-quarters of an hour. In young children the chill is replaced by one or more convulsions, or else the first stage is very mild or wanting altogether, or the child grows suddenly pale and has pronounced nervous symptoms.

The first stage passes into the second by gradual abatement of the chill and rigors, which are replaced by a feeling of warmth.

The second or hot stage is characterized by fever and high temperature. The peripheral vessels now have their constriction relaxed, and the pallor of the face and lividity give place to flushing and redness, the skin feels smooth, hot, and dry, and the thermometer in the rectum records 103°, 105°, or 106.5°, or even a higher temperature. Respiration grows deeper, and the pulse is full, bounding, and rapid—130 or more—and it is frequently dicrotic. There may be an anæmic bruit heard at the base of the heart. The patient becomes restless and irritable, but the mind is clear. The mouth is dry and the throat parched. Sometimes there is herpes labialis. There is continued throbbing frontal headache, and the vomiting may be repeated. The tongue is coated with thick white fur, and the breath is foul. There is constipation. There may be slight dizziness or a "sinking feeling," tinnitus aurium, and muscæ volitantes. This stage lasts from three to six hours, when the fever gradually declines and the patient becomes easier.

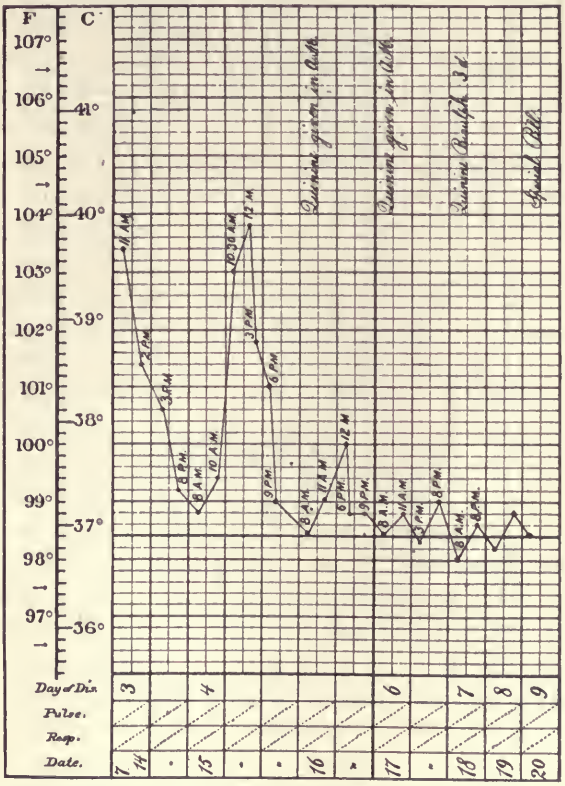
The third or sweating stage is characterized by profuse perspiration and the disappearance of the fever. The fever may subside before the perspiration occurs, or it may continue into the third stage; hence the perspiration cannot

be regarded as the cause of the subsidence of the temperature. The sweating commences on the forehead and face, and soon the whole body is profusely bathed by it, so that the bed-clothing is thoroughly wet. The pulse becomes slower and returns to the normal tension, and the spleen gradually returns to its natural size. With the subsidence of the fever the restlessness disappears, and, although the headache may continue, the patient becomes much more comfortable, and, feeling greatly exhausted, he usually passes into a quiet sleep, from which he awakens more or less fatigued.

The sweating stage lasts from two to four or six hours. The entire paroxysm lasts from six to ten or twelve hours, according to its severity. In the interval between two paroxysms the patient may feel in perfect health, but there are apt to be more or less debility, anorexia, and anæmia, with a diminished number of red blood-disks and a reduced quantity of hæmoglobin in the blood.

Special Symptoms.—*Temperature in Intermittent Fever.*—The temperature, although it may be very high for a few hours, is not regarded with the

Fig. 25.



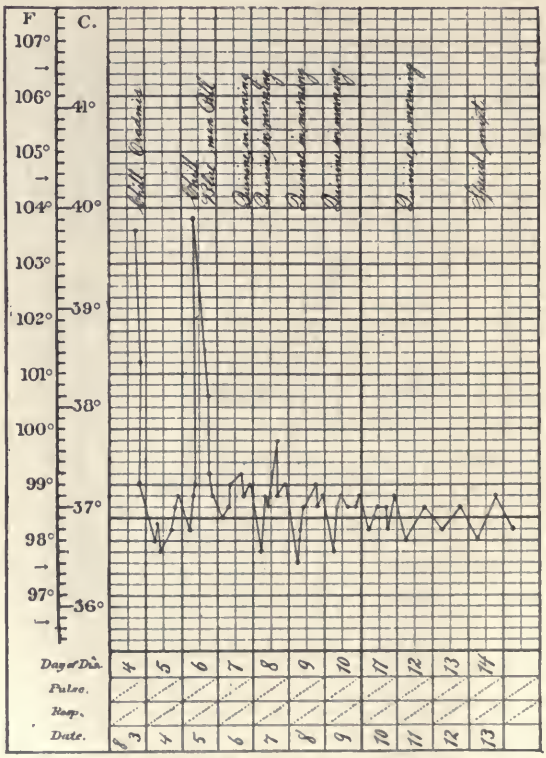
Temperature-chart of a Case of Quotidian Intermittent Fever.

solicitude that it would engender in other affections. The same degree of temperature might be fatal in a case of insolation which in malarial fever soon

subsides of its own accord. Parke[1] states that in Africa he has seen every officer of the Emin Pasha Relief Expedition "do a day's march with a temperature of over 105° F." Of course this was exceptional and required undaunted pluck, but in many other diseases the same degree of fever would mean utter prostration or delirium or coma.

The temperature rises more rapidly than it falls. It rises slowly but uniformly in the cold stage, and more rapidly during the hot stage. The maximum temperature lasts for one to three or four or more hours according to the severity of the paroxysm. The decline is sometimes uniform, but it is frequently interrupted by "steps"—*i. e.* it falls one or two degrees, remains stationary for half an hour or an hour, then falls again, and so on (Wunderlich). After the paroxysm it is quite common for the temperature to drop a degree or a degree and a half below the normal for a few hours. The accompanying temperature-charts (Figs. 25 and 26) illustrate the periodicity of the high temperature attained in the quotidian and tertian types.

FIG. 26.



Temperature-chart of a Patient with Tertian Intermittent Fever.

The Urine.—After the chill the urine is often increased in amount and is of low specific gravity. This is attributed in part to alterations in the renal blood-pressure from the constriction of the superficial blood-vessels of the

[1] *My Personal Experiences in Equatorial Africa*, p. 424.

body. The malarial fever gives rise to waste products which are taken up by the blood and eliminated by the kidneys. This elimination sometimes attains its maximum before, sometimes during, the paroxysm. There is always an increase in the elimination of urea during a paroxysm, and Jaccoud has noted that this increase commences even before the chill, so that careful quantitative estimation of urea will foretell the approach of a paroxysm. This increase of urea-excretion he observed two hours before the chill in quotidian and six or eight hours before in tertian fever. He regards the estimation of the increased urea as a reliable indication for the proper time for administering quinine in order to anticipate the chill. Rarely there is temporary albuminuria during the pyrexia. Temporary glycosuria has also been reported in a few cases.

Exceptional Cases.—In the vast majority of cases of malarial fever each succeeding paroxysm conforms in its duration and stages to the type with which the disease began, but this is not invariably the case, and there are some curious anomalies in the type itself. Thus, the cold stage may be omitted entirely or the sweating stage may similarly be absent, or they may both be wanting, leaving only the hot stage. When the chill is absent the disease is sometimes called "dumb ague." This form is more common among the older residents of a malarious region.

In severe cases of intermittent fever the several stages, and especially the hot stage, may all be prolonged, and the sweating stage of one paroxysm may overlap the cold stage of the next. In rare instances the cold stage may be replaced by various neurotic symptoms, such as neuralgic pains, general nervousness, or periodic hysteria, and, if the temperature be high, there may be drowsiness, partial coma or delirium, etc. Intermittent deafness, blindness, vomiting, diarrhœa, and asthma are all symptoms which may at times predominate or which may precede a typical paroxysm.

"*Latent intermittent fever*" is a name given to a condition among certain persons living in malarious regions. There are no definite paroxysms, but the condition is best described by the term "bilious." There are anorexia, vomiting, headache, constipation, weakness, and lassitude. Sometimes there is a very slight periodic elevation of temperature. Such persons at any time are liable to be seized with a veritable paroxysm.

"*Masked malarial fever*" is a variety of intermittent ague which is commonly neuralgic. The ordinary malarial paroxysm is replaced by violent neuralgic pain, lasting from half an hour to six or eight hours, and recurring, like the true paroxysms, at regular intervals. These attacks are usually unaccompanied by fever, which makes the diagnosis still more difficult. The nerves most often affected are the supraorbital or infraorbital branch of the trigeminus. Other branches of that nerve may be affected, or the sciatic, the nerves of the brachial plexus, or intercostals. In children there is sometimes bronchial catarrh. The term "masked ague" may also refer to malarial fevers in which other disorders having pronounced symptoms occur, such as pneumonia, dysentery, etc., and which by their greater intensity obscure the original ague.

Course.—Simple intermittent fever runs a mild course, and the number of

paroxysms may be cut short at any time by treatment, by removal of the patient, or by change of season. If left to itself, the disease may run on for several weeks or months, and in a bad malarial region at any time it may suddenly be converted into one of the severe types of ague. Quartan fevers are often more obstinate than either quotidian or tertian.

Relapses are very frequent in all forms of ague, and they may undoubtedly occur without fresh exposure to the miasm, as may be the case with sailors at sea. It is a curious feature of such relapses that they often occur on the day on which there would naturally be a paroxysm had the disease been uninterrupted. A relapse at periods of two, three, or seven weeks is sometimes noted, or the interval may be very much longer. It is said that in such cases there may be modifications in the urine with increase in urea corresponding to latent paroxysms. While there is any elevation of temperature indicated by the thermometer there is very likely to be a relapse, or if the spleen remains enlarged relapses are apt to occur.

Sometimes the spleen becomes periodically enlarged, reaching even to the umbilicus. This enlargement is accompanied by local pain and tenderness, and severe vomiting, while the chill and fever may be entirely absent. Between the attacks the engorged spleen returns to nearly normal size.

Terminations.—The majority of mild cases of malarial fever recover by themselves, but the recovery is greatly accelerated by appropriate treatment. Severe cases become more or less chronic, and repeated attacks develop the malarial cachexia. Even the graver forms of malarial fevers, such as the bilious or hæmorrhagic types of remittent fever, are frequently amenable to treatment if seen early and if treated very promptly. Otherwise they may prove fatal from various causes.

One attack of malarial fever affords no immunity, but, on the contrary, it is apt to predispose the subject to others. Secondary attacks may occur within a few weeks or after an interval of years.

The number of individual paroxysms that the same person may have at varying intervals while in a badly-infected district is sometimes extraordinary. Parke[1] states that among the Europeans who crossed the continent of Africa on the Emin Pasha Relief Expedition the average number of separate attacks was one hundred and fifty for each man in a period of three years, during which time they marched five thousand miles.

Diagnosis.—The diagnosis of malarial fevers is easy when the attack is simple and typical, and when the patient can furnish a clear history of exposure or of previous attacks. It is far more common to mistake other diseases for ague than to err in the opposite manner.

Simple intermittent fever is readily distinguished from remittent fever by the use of the thermometer, which in the latter demonstrates a continuance of the fever in the interval between two paroxysms. There are usually other symptoms besides the temperature that persist during the interval in remittent fever.

[1] *My Personal Experiences in Equatorial Africa*, p. 483.

In all doubtful cases the presence of an enlarged spleen and the effect of a large dose of quinine will aid the diagnosis. The quinine can scarcely ever be harmful, and its prompt employment often serves to clear away very obscure symptoms.

Moreover, the blood should always be examined for the malarial plasmodium. This is easily done by pricking the thoroughly cleansed finger and drawing a minute drop of blood, which is to be flattened out into a very thin layer by pressing it between a cover-glass and a microscope slide until the corpuscles are only one layer deep. The slide is then placed under a high-power lens ($\frac{1}{12}$ oil-immersion), when the germs, if present, may be detected by careful search. Osler says that the crescentic forms may be detected with a lower power, such as an $\frac{1}{8}$-inch objective.

The sudden occurrence of a severe chill and rigors lasting three-quarters of an hour, followed by a sharp, brief fever, the temperature reaching 105° or more within a few hours, is very suggestive of malarial fever. In but few other affections attended by an initial chill is the latter so protracted and severe. It is exceptional in any other disease for the temperature to reach such an elevation so suddenly and to promptly subside again to the normal.

Pneumonia may be ushered in by a severe chill and rapid rise of temperature, but the subsequent course of the disease, the development of physical signs in the chest, the sputum, and the continuance of the fever will soon confirm a doubtful diagnosis.

Deep-seated suppuration, producing sudden general septic infection, may be mistaken for malarial fever. In such cases a searching physical examination, with a careful history of the case, the absence of splenic enlargement, and the lack of regularity in the recurrence of chills, fever, and perspiration, will aid in eliminating malarial fever from the diagnosis.

Pulmonary phthisis is occasionally mistaken for ague when there is suppuration with recurring chill, hectic fever, and perspiration. In such cases the correct diagnosis can be made upon a thorough physical examination of the chest, the detection of tubercle bacilli in the sputum, the normal spleen, and the negative effect of quinine upon the hectic.

Catheterization, or the passage of a sound, occasionally produces a paroxysm resembling that of ague.

Prognosis.—The prognosis of uncomplicated intermittent fever is most favorable for speedy recovery under proper treatment, but it should always be remembered that in a region where malignant types of the disease sometimes occur a simple unchecked intermittent fever may rapidly merge into a most pernicious form with fatal issue.

Prophylaxis.—To some extent malarial fevers may be restricted by draining and filling in marshes and improving the general sanitary condition of a locality.

Favorable influence has been attributed to the eucalyptus tree (*Eucalyptus globulus*) planted along the edge of marshes. These trees grow rapidly and absorb a considerable amount of moisture, thereby drying the marsh; and

closely-planted trees, like high fences, prevent the dissemination of the heavy malarial poison in the atmosphere to a very limited degree. Experiments on a large scale, as made by the French in Africa and the Italians near Rome, have, however, failed to demonstrate any special preventive influence from the eucalyptus.

Proper attention to the general health is important. Many persons living in a malarial region who have had the fever find that when they allow themselves to become constipated for two or three days they are apt to precipitate an attack of the ague.

Excesses in eating or drinking, mental strain, over-fatigue, and exposure should be strenuously avoided. Persons are far more liable to acquire malarial fever if exposed to the miasm while fasting than after eating.

Persons living on the upper floors of buildings in a malarial region are less likely to have ague than those who occupy the ground-floor or basement. The susceptibility to the miasm is greater after sunset, at night, and in the early morning than in broad day. Sleeping out of doors should be especially avoided. By proper attention to these facts many persons can avoid exposure while residing in an infected locality.

Those who are obliged to live in malarial regions do well to take quinine in daily moderate doses—three or four grains twice a day—and arsenic is also of value as a prophylactic. Fowler's solution, in doses of four to six minims well diluted, may be taken three times a day after meals. The quinine should be taken only in the season when ague is active, for, if too long continued, the system after a time becomes accustomed to it, and very large doses are required to obtain any effect in an emergency.

Care should be exercised to maintain the general health by proper regulation of the diet, bathing, clothing, exercise, etc., and it is important to avoid constipation.

Treatment.—The chief indications for treatment are to prevent the return of the paroxysm, to restore the blood to a normal condition, and to re-establish the functions of the congested viscera.

The urgency with which treatment must be employed in the various malarial fevers will depend upon the severity of the case. Fortunately, in the salts of quinine we possess a specific for malarial fever, and in very mild cases of intermittent fever no treatment is required beyond a few grains of that remedy. In severer agues, however, prompt and energetic action is imperative in order to save life, for it may result in the rescue of apparently moribund cases of the worst forms of pernicious malarial fever.

Treatment of the Chill.—When a paroxysm of ague is expected, the patient should go to bed and keep warm. As the chill approaches a diffusible stimulant, such as aromatic spirits of ammonia, with fifteen or twenty drops of chloroform, may be given with some simple hot drink. Hot-water bottles should be placed at the feet. Warm blankets are needed. An opiate, such as Dover's powder, is often beneficial. This treatment is sometimes successful in aborting the chill, and it may lessen the severity of the entire paroxysm.

Treatment of the Fever.—In the hot stage the covering should be lessened and the patient may be sponged with cold alcohol and water in equal parts. Cooling draughts of carbonic-acid water, Vichy, or lemonade may be given. Except for the administration of quinine, described below, the temperature rarely requires any more active treatment, as its duration is brief in any event. During the sweating stage the patient is made more comfortable by having the perspiration wiped away with warm cloths as fast as it forms.

The Administration of Quinine in Malarial Fevers.—The dosage and method of administering quinine must vary somewhat with the condition of the patient, the severity of the attack, and the quantity which the patient may be accustomed to take. Some persons are put in a state of most uncomfortable cinchonism by a dose of five grains, while others are not disagreeably affected by thirty.

Small doses of quinine are often efficacious in those persons in whom cinchonism is readily produced, whereas larger doses are commonly needed by those in whom toleration is much greater. In very mild cases five or ten grains given some hours before a paroxysm will avert it, but in the severer types forty, fifty, and in very malignant types even one hundred, grains must be given within a few hours.

Quinine acts most promptly when administered in solution, but the taste is so bitter, lingering, and difficult to disguise, often causing vomiting, that it is generally preferable to give the drug in powder, in a wafer, in black coffee, or in a soft gelatin capsule. Pills are apt to become hard and insoluble. When not given in solution quinine is rendered more soluble and assimilable by prescribing ten or fifteen minims of dilute hydrochloric acid to follow each dose. Young children refuse the bitter solutions of quinine, and they cannot swallow pills or capsules: in such cases the drug may be given in solution or suppository by the rectum, or it may be rubbed into the abdominal wall as an oleate or ointment. In this way the constitutional effects are usually obtainable.

Some clinicians give quinine at stated intervals without regard to the paroxysms, aiming merely to administer a certain dose within twenty-four hours; and in very mild cases this will accomplish the desired result, but in the majority of instances it is better to prescribe one or two large doses, carefully timed to meet the paroxysm, so that one dose shall not be eliminated before the next exerts its influence. Thirty to forty grains given four to five hours before the paroxysm in a very severe case will accomplish far more than if the same amount be distributed throughout the day, for it is quickly eliminated from the system.

It is sometimes desirable to precede the quinine by a purgative dose of calomel, for the bowels are apt to be constipated, the tongue coated, and the patient more or less "bilious;" but it is not advisable ever to delay the administration of the quinine on this account. In severe cases of the pernicious form it is highly injurious to weaken the patient by purgation, and it

is a mistake to drive the quinine out of the alimentary canal by calomel before it has had time for complete absorption.

It is of no use whatever to give quinine during a paroxysm of simple intermittent fever, for it requires so long a time for its complete influence upon the system to be established that the paroxysm is over before it can be absorbed. In fact, it is very often vomited when taken during the seizure. It is from four to six hours after the administration of quinine before its maximum effect is attained. In quotidian fever quinine should be given eight hours before the expected chill, because the real onset is two hours before the chill. In tertiary fever it should be given twelve hours before, and in quartan fever fifteen or eighteen hours before, and repeated. A fifteen-grain dose of quinine given only two hours before or given during a paroxysm does not affect it, but given at the close of one paroxysm it aborts the next paroxysm either wholly or in part. It may have to be continued in this manner for four or five days before the fever entirely ceases, and quinine should be taken in smaller doses for a week or two thereafter.

The effect of quinine in intermittent fever is to prevent a second paroxysm only in a certain number of milder cases. In other cases it either postpones the next paroxysm, or, without postponing it, renders it much milder than it presumably would have been, causing the chill to be abbreviated or omitted. A third paroxysm is usually prevented by the quinine.

Many believe that opium acts as an adjuvant to quinine in controlling malarial paroxysms. Schauffler recommends the bromide of potassium in doses of forty to eighty grains to relieve cinchonism and quiet the nerves.

In certain patients quinine possesses but little influence over the fever. This may be due to some idiosyncrasy or to the fact that the system from long-continued use of the remedy has become inured to it. Warburg's tincture and arsenic may then be of service. The former is a compound remedy which has been long used in India and elsewhere. Besides preparations of cinchona, the original formula contained chiefly aloes, rhubarb, opium, and camphor. A modified Warburg's tincture is prepared by omitting the aloes and some of the minor ingredients. This remedy has a very disagreeable taste, and, since the dose is f℥ss in water, it is apt to prove nauseating. It may be given by the rectum, where it is usually well borne, or in pill form after evaporation, but the latter method is not so efficacious. Warburg's tincture sometimes succeeds in breaking up obstinate malarial fevers when quinine has failed. It is of more use in the severer forms of ague than in simple intermittent fever, and quinine may be given in combination with it. Besides controlling the fever, it has to some extent a sudorific action.

Arsenic is administered as arsenious acid, one-thirtieth of a grain thrice daily, in pill or in the form of Fowler's solution, liquor potassii arsenitis, four to six minims thrice daily, after meals, well diluted. It is often useful to combine this drug with iron on account of the anæmia, which is more or less marked. Neither Warburg's tincture nor arsenic have any effect upon a paroxysm already begun.

Many attempts have been made to find substitutes for quinine for use in those cases in which it is not well tolerated. Other preparations of cinchona, such as quinidina, chinoidina, cinchonidina, have all been used. Salicin in doses of a drachm in twenty-four hours, strychnine and nux vomica, ammonium chloride and eucalyptol, with a long list of other remedies, have been faithfully tested for antiperiodic action, but none of them can really replace quinine.

II. Remittent Malarial Fever.

This fever, from the prominence of the gastro-intestinal symptoms, is often called bilious remittent fever or gastric fever. This type of ague is characterized by the same symptoms that occur in intermittent fever, but the temperature continues elevated through the interval. It is supposed to be due to a greater intensity of action or of concentration of the miasm, or to a greater susceptibility on the part of the patient.

Morbid Anatomy.—In fatal cases of remittent fever the characteristic lesions are a deep pigmentation of the spleen, liver, and brain, and the presence in the blood of free altered blood-pigment—a condition known as melanæmia. Organs having deposits of such pigment are said to be in a state of melanosis. The pigment occurs in granules. It is found in remittent fever, in pernicious malarial fevers, and occasionally in protracted intermittent fever and malarial cachexia.

The pigment in melanæmia forms Prussian blue when tested with the ferrocyanide of potassium; hence the iron which it contains does not all exist as an organic compound. The extent of discoloration of the different organs affected varies with time. In recent cases they are slightly darker than normal, but in protracted cases they are deeply bronzed or of a grayish or bluish-black color.

The spleen is at first hyperæmic, soft, and swollen, but as the paroxysms return it fails to contract in the interval, and it gradually becomes permanently hypertrophied and firm, instead of remaining soft. There is hyperplasia of the connective-tissue elements of the organ. The pigment is found deposited within the lymphoid splenic cells in granular masses. It is also found around and in the walls of the veins.

The liver is enlarged, but in old cases it may be atrophic (Flint). It is often hyperæmic, and is strongly pigmented. It is commonly called the "bronze liver." Pigment is found in granular masses both in and between the lobules, in the vessels, and vessel-walls.

The marrow of the long bones is similarly pigmented. The granules are found in the lymphoid cells, around and within the blood-vessels.

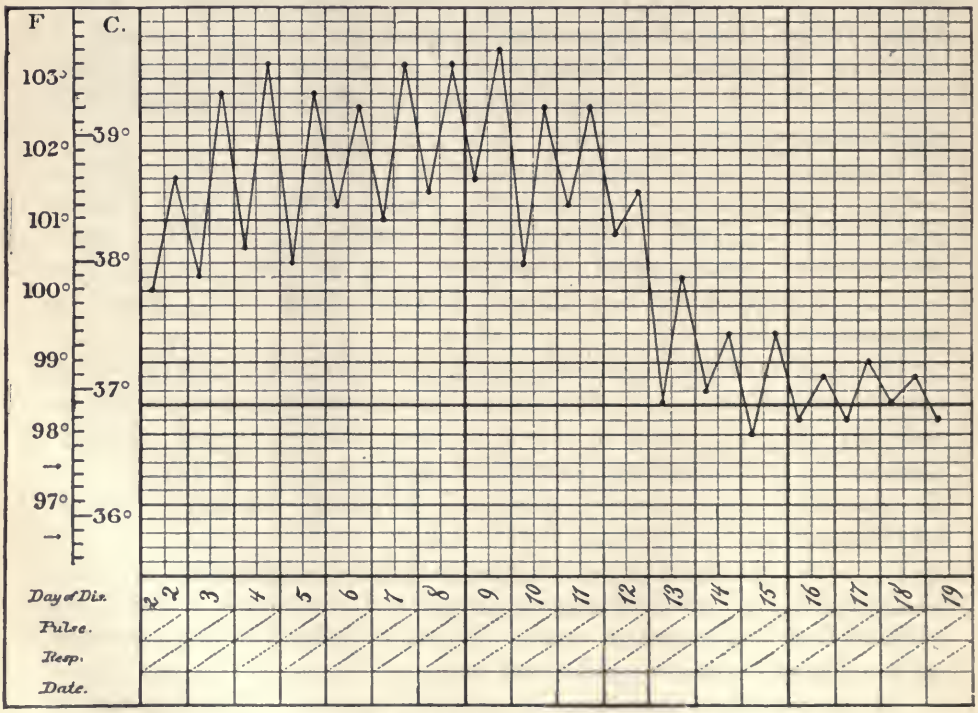
The gray matter of the brain is dark gray or almost black. In severe cases the white matter is also pigmented, and it exhibits minute hæmorrhages, which are thought to be produced by emboli of small masses of pigment which enter the capillaries and occlude them. In the brain, as elsewhere, the pigment is found in the walls and outside of the blood-vessels.

Other vascular organs, such as the pancreas, thyroid gland, kidneys, the mucous membranes, lymphatic glands, and the skin, are more or less pigmented. There may be ecchymoses in the mucous membranes of the alimentary canal.

Symptomatology.—This variety of fever is apt to begin with a more pronounced paroxysm than occurs in intermittent fever, although the cold stage may be more brief. Sometimes, however, there are prodromata, such as may precede any febrile disease, or there may first be one or two mild intermittent paroxysms.

When the paroxysm commences there is a good deal of nausea and emesis, which continue. Often large quantities of bile are vomited. There are tenderness over the epigastrium and splenic area and loose watery diarrhœa. Not infrequently there is jaundice. The temperature is high, often rising to 106° F. or higher. The second stage of febrile exacerbation often lasts for a longer period than in intermittent fever, and it may not subside before twelve or even twenty-four hours. (See Fig. 27.) In milder cases the temperature falls in

FIG. 27.



Temperature-chart of a Patient with Remittent Fever.

three hours. This is followed by a sweating stage of moderate degree, and the fever continues at 101° to 103° F. until the next paroxysm. In the second paroxysm the chill is frequently omitted.

During the interval, besides the increased temperature there may be nausea, lassitude, and muscular soreness. The frequency of the paroxysms

may correspond to any type, quotidian, tertian, etc. After about a week of severe fever the pyrexia gradually subsides, and in another week or two it disappears and the patient recovers. The fever may subside by becoming continuous and slowly decreasing, or it is not uncommon for it to pass into a distinct intermittent type and end in that manner. The remission usually commences between midnight and the early morning. Occasionally the disease ends by crisis.

Duration.—The duration of the disease can be curtailed by treatment. It may last anywhere from three or four days to three weeks, or it is still further protracted, and the patient may pass into the typhoid state. As a rule, remittent fever is more grave than intermittent ague.

Complications.—Remittent fever may occur in connection with many other diseases or be complicated by them. Among the more frequent complications are to be mentioned acute nephritis, dysentery, and lobar pneumonia.

Diagnosis.—Remittent fever is liable to be confounded with typhoid fever and with certain cases of yellow fever occurring at the commencement of an epidemic. Since this is also true of the pernicious type of ague, the diagnosis will be considered in connection with that disease. (*Vide infra.*)

Prognosis.—The prognosis for simple remittent fever is favorable. In this type of malarial fever there is more danger than in intermittent fever of sequelæ, such as extreme anæmia, "ague cake," and dropsy. The disease is much more severe in hot climates than in temperate regions.

Treatment.—The treatment involves the early production of cinchonism, as in the intermittent type. Large doses of quinine, twenty to thirty grains, should be given every three or four hours until there are ringing in the ears and throbbing of the temples. It is not necessary to await the termination of the paroxysm, but the quinine should be begun at once, for it is the prolonged hot stage which especially demands attention. As a rule, severe purgation or any depleting measures should be avoided. In certain cases, however, when the bowels are very constipated, the tongue thickly coated, and the urine diminished and overloaded with solids, a dose of calomel or of blue mass is indicated. Care must be taken not to hurry off the quinine by purgation; and, if the quinine has just been given, the bowels can be moved by a stimulating enema of half an ounce of turpentine and an ounce of castor oil in a pint of warm soapsuds.

If, on the other hand, severe diarrhœa or depressing emesis exist, such symptoms should be controlled by an opiate, and poultices or hot turpentine stupes placed upon the abdomen. When the kidneys are congested, mild saline diuretics, such as potassium citrate or bitartrate, may be given with refrigerant or effervescing drinks, such as Vichy, acid lemonade, carbonic-acid water, etc.

III. Pernicious Intermittent Fever.

Pernicious intermittent fever is also called malignant, irritative, and congestive intermittent fever or congestive chills. As the name implies, it is a very

severe type of ague. In the late Civil War in the United States this type of fever constituted nearly 24 per cent. of the mortality from disease. It is a relatively uncommon form of malarial fever, but it appears from time to time in tropical countries, and in the United States in the South and West.

The disease may commence in a malignant manner and be fatal at once, or it may be ushered in by one or more paroxysms of ordinary severity. When one paroxysm grows very severe, the following one is frequently fatal. The pernicious type soon becomes manifest through one or more of the following features: deepening coma, delirium, violent vomiting and purging with blood and mucus in the stools, intense weakness, hæmaturia with hæmorrhage from various mucous surfaces, and collapse.

The prominence of certain of these symptoms in pernicious malarial fever makes it convenient to subdivide the disease into several varieties, which will be briefly described under the following heads:

I. Bilious intermittent fever, in which the gastro-intestinal irritation with vomiting of bile is the most striking symptom;

II. The hæmaturic or hæmorrhagic variety, with bloody urine and hæmorrhages from mucous surfaces;

III. The asthenic form, with great prostration and feeble circulation;

IV. The algid form, resembling the algid stage of Asiatic cholera;

V. The comatose variety, with sudden and profound unconsciousness.

Symptomatology.—I. Bilious Intermittent Fever may attack the patient very suddenly, the first symptom being a severe chill and vomiting, or it may be preceded by dyspeptic symptoms, constipation, flatulence, a coated tongue, and offensive breath, with muscular pains. In patients who have had several attacks, often one of the first symptoms noticed is an extreme irritability of temper with great mental and bodily restlessness. There is frontal headache, the muscular pains increase, and the entire body aches. The pains are mostly in the loins and knees, and sometimes they begin quite suddenly, as though the muscles had been pounded. These pains are occasionally so severe as to require morphine for their relief. There are often cramps in the muscles of the calves. The pulse is rapid and the heart-action irritable. Pallor is succeeded by congestion of the face and injection of the eyes, which acquire a typical staring, glistening appearance. The temperature rises to 105° or 106.5° F., or even higher. The vomiting begins early and continues. Large quantities of bile may be ejected. Pressure over the stomach and liver is painful. The prostration is very great and there is rapid emaciation.

In severe cases there is a sense of fulness or constriction in the chest, and neuralgic pains may be felt in the larger nerves, as the sciatic, median, or anterior crural. The spleen becomes greatly enlarged, and the splenic region may be more or less tender. There are increasing anæmia, and decided constipation from lack of power in the muscular coat of the intestine. The urine becomes dark, of high specific gravity, is loaded with urates and phosphates, and may contain blood or bile-pigment. In some cases rapidly-deepening jaundice is a prominent symptom, coming on within five or six hours.

In certain cases of bilious intermittent fever, as in the simple form, the chill and sweating are absent, the recurrent fever being the main symptom. In other cases the chill and fever are present, but the third stage, that of sweating, is omitted.

In an intestinal variety there are frequent diarrhœal stools, with flatulence, tormina, and abdominal distension and tenderness, especially over the stomach and liver. The stools contain large quantities of mucus, and at times they become so copious and watery as to suggest choleraic discharges, or they contain blood and mucus, as in dysentery.

II. The Hæmaturic Type.—The hæmaturic or hæmorrhagic form is always very serious. The paroxysm commences with a prolonged chill and rigors, and the temperature rises rapidly. In the second stage blood-disks and blood-pigment appear in the urine, which is diminished in quantity and contains more or less albumin with granular and bloody casts. The patient appears very ill and is restless and anxious. Soon a condition of general purpura develops: there are ecchymoses into the skin, and there are epistaxis, hæmorrhage from the mouth, stomach, rectum, or vagina. The skin becomes more and more yellow, or even of a bronze hue. Suppression of urine is apt to occur, and then the toxic condition of uræmia is added to the existing blood-poisoning. The emesis continues: there are violent headache, delirium, and finally deepening coma, with Cheyne-Stokes respiration, a rapidly failing heart-action, pulmonary œdema, and death. These hæmorrhagic symptoms may occur in either pernicious intermittent or remittent fever. In less severe cases the mind remains clear throughout. When delirium or coma ensues the case becomes very critical. Death is not caused by excessive hæmorrhage, but by toxæmia or asthenia. There is intense congestion of viscera, and the hæmorrhages are believed to be occasioned by a combination of altered blood-composition, impaired nutrition of the capillary walls, and changes in the local vascular pressure in the various congested organs (Bemiss). The hæmorrhages usually bear a direct relation to the intensity of the chill, which favors internal congestion.

III. The Asthenic Type.—In this variety there are irregular neurotic symptoms, restlessness, and great weakness. The circulation becomes extremely feeble and the cardiac sounds are scarcely audible. The pulse is reduced to a mere thread and is intermittent. In other cases exhausting perspiration is a very prominent feature, which continues into the remission.

IV. The Algid Type.—The algid form of pernicious malarial fever suggests the algid stage of cholera, and is very fatal. There are the same prostration and collapse, with cold extremities, cramps, cyanosis, dilated pupils, feeble, husky voice, shallow respiration, vomiting, purging, and great thirst. There are often very profuse perspiration and offensive fluid alvine evacuations. The pulse is very feeble and irregular. The internal temperature is very high. The intellect remains clear.

In some cases the disease resembles yellow fever, and there are profound

jaundice, serious neurotic symptoms, and severe emesis. The brain and cord are usually found anæmic, dry, and firm (Hertz).

V. The Comatose Type.—The comatose variety is rare, excepting in hot climates and where the miasm is greatly concentrated. The patient is early overwhelmed with profound coma, from which it is impossible to arouse him. He may die suddenly in collapse. The coma is not necessarily due to cerebral congestion (Bemiss), although the brain and meninges may both be congested, but to general toxæmia. The surface of the body is hot; there are elevated temperature and pulse-rate; and stertorous breathing and jaundice may be present.

This variety of pernicious fever develops especially in persons who reside in regions where ague abounds, and who have neglected proper treatment in repeated attacks. It may be associated with one of the other graver forms. In the United States the congestive varieties of pernicious ague are much more common than the comatose. Should the patient survive the first attack of coma, the second is usually fatal, but third attacks sometimes occur.

In some cases, instead of coma, there is sudden, violent delirium, with cephalalgia, congestion of the face, staring eyes, and great excitement. At the autopsy there are found more or less hyperæmia and œdema of the brain.

Complications.—Complications of infrequent occurrence, but which have been from time to time noted in connection with the pernicious types of malarial fevers, are hemianopsia, transient amblyopia, optic neuritis, and hæmorrhages (Sulzer), muscular contractures and choreic or ataxic movements, convulsions (in children), local anæsthesia, transient cortical paralyses, such as aphasia with hemiplegia, spinal congestion, etc.

It has been suggested, although there is as yet no proof, that some of the neurotic symptoms and lesions may be due to embolic plugging of small arterioles by the plasmodium or by pigment-granules. In a case of pernicious malarial fever with bulbar paralysis Marchiafava found the plasmodium in the nuclei of the facial and hypoglossal nerves, with necrosis of their cells.

Periodical delirium has exceptionally been noted. In some patients there is a decided tendency to syncope, while others pass into a condition of "suspended animation," in which the radial pulse and respiratory movements almost disappear, and the cardiac sounds are so inaudible that the patient seems almost dead.

Asthma sometimes occurs, or there may be severe and sudden localized pulmonary congestion, with signs of consolidation, and sometimes rusty sputum and dyspnœa resembling pneumonia, but disappearing under the use of quinine.

Sequelæ.—Repeated attacks of the severer forms of malarial fever leave the patient in a weakened and very irritable condition. There may be paralysis, dropsy, extreme anæmia, or the malarial cachexia. (*Vide infra.*)

Diagnosis.—The different varieties of pernicious malarial fever at times resemble the following diseases, from which they are to be diagnosed: typhoid

fever; yellow fever; cholera; ulcerative endocarditis; pyæmia and septicæmia; uræmia and meningitis.

Typhoid Fever.—Simple remittent and pernicious malarial fever are distinguished from typhoid fever by the absence of the rose-colored rash, epistaxis, tympanites, and pea-soup evacuations which so frequently occur in the latter disease. Moreover, in typhoid fever the invasion is slower, and usually without distinct chill; the fever is more continuously high after reaching a maximum than in remittent fever; and the delirium, carphalogia, subsultus, and other neurotic or ataxic symptoms are more pronounced. The tongue in typhoid fever is red and small at first, afterward brown and dry. In malarial fevers it is large and more heavily coated. In severe malarial fevers there is often a history of one or more previous paroxysms and of exposure to infection, and the gastric symptoms are more pronounced at the onset. There is also a tendency to become jaundiced which is absent in typhoid fever, and the skin is sallow at the commencement of the disease. The symptoms of gastric irritation appear earlier, and are more pronounced, than in typhoid fever. The face during the paroxysm is more flushed, the eyes are congested, and the expression is more animated than in typhoid fever.

The detection of typhoid bacilli in the stools would render the diagnosis certain, but their demonstration is difficult.

Yellow Fever.—In remittent fever typical black vomit never occurs, as it may in yellow fever; the pulse is more firm; the temperature is higher; and the influence of quinine and the subsequent course of the disease are to be noted. The icteric form of pernicious ague affects those who have been long resident in an ague district, but yellow fever selects particularly the recent arrivals. The jaundice appears later in the course of yellow fever. In pernicious ague free pigment should be sought in the blood. Yellow fever is apt to be more quickly fatal.

Cholera.—The algid form of pernicious ague is to be distinguished from cholera by the free pigment in the blood, and the fact that the copious watery evacuations are often preceded by bloody stools, which is not the case in cholera. In cholera there is the presence of an epidemic and the history of infection.

Ulcerative Endocarditis and Pyæmia.—Remittent and pernicious malarial fever may resemble acute ulcerative endocarditis or pyæmia and septicæmia. In the former the physical examination of the heart and the presence of embolic infarctions will aid in establishing the diagnosis, and in the latter the diagnosis can be made by the exclusion of a source of septic infection and by the greater regularity of the paroxysms occurring in pernicious fever.

Uræmia and Meningitis.—The comatose form of pernicious fever must be distinguished from uræmia and meningitis. The presence of free pigment-granules in the blood and the enlarged spleen are to be noted, with a history of previous malarial paroxysms. In meningitis the case is of longer duration before coma appears; it is preceded by photophobia and delirium, and the temperature is lower than in comatose ague.

Mortality.—In the rural parts of the Southern Atlantic States the number of fatal cases of malarial fevers is 70.6 in every 1000 deaths. In cities the number is 11.5 (Johnston).

Treatment.—The treatment of pernicious malarial fevers demands the utmost care and promptness. Quinine must be given at once by hypodermic injection in doses of fifteen grains in distilled water, using a soluble salt, such as the tannate, hydrochlorate, or hydrobromate, combined with a grain of sodium chloride (Bocelli). The sulphate and bisulphate, which are the preparations most frequently employed when quinine is given by the mouth, are not adapted for hypodermic use on account of the difficulty of dissolving them in a small bulk of water without the use of acids, and the consequent liability to abscesses at the site of injection. Free stimulation by the mouth, rectum, or subcutaneously must be employed. Everything depends upon tiding the patient over a present paroxysm and preventing the recurrence of a second, which is so apt to be fatal. The patient must be kept absolutely quiet. Opium in full doses, given early, is often serviceable. Morphine and atropine may be given with whiskey or brandy, and diffusible heart stimulants, such as chloroform or ammonia, are required when there is any evidence of enfeebled circulation. Hypodermic injections of strychnine are also serviceable. Warm alcoholic stimulants should be given by the rectum.

Hot bottles should be applied to the surface for the collapse, while vigorous rectal and hypodermic stimulation is maintained. By stimulating and nourishing such cases even the worst of them are rendered not necessarily fatal, and everything depends upon careful attention to all the details of the treatment. In the violently congestive type, with delirium and a full pulse, venesection has been employed; but it is of doubtful efficacy, for the patient is soon in greater need of stimulation than depletion. Saline laxatives, cold sponging, cold applications to the head, and sedatives, especially opium, are indicated for this type of fever.

Vomiting and purging must be controlled by opium. Warburg's tincture may be given in the intermission or remission, but cinchonism must be steadily maintained. Patients have become both blind and deaf under the excessive use of quinine in these cases, but fortunately such results are almost invariably temporary accidents, and the patient's life depends upon the prevention of another paroxysm through the agency of this invaluable specific.

In the hæmorrhagic form the vomiting may be controlled by morphine, and very mild diuretics and diaphoretics are serviceable. Ergot and turpentine have been given with the idea of checking the hæmorrhages, but such remedies are of very doubtful efficacy.

With signs of improvement the patient's strength must be supported by a nourishing and concentrated fluid diet and nutrient enemata. Beef juice, beef peptonoids, egg-albumin in sherry, and milk should be given in small quantities repeated every hour or two. As convalescence advances, tonics, such as simple bitters, iron, quinine, and hypophosphites, will be required to build up the impoverished blood and restore the greatly reduced system.

IV. Pernicious Remittent Fever.

This type corresponds so nearly with pernicious intermittent fever, excepting in regard to the temperature-curve, that a separate description is superfluous. The fever is sometimes called African fever, jungle fever, etc.

None of the severe types of ague correspond as closely in regard to the time and duration of the paroxysms as do the simple forms of paludal fever, and it is therefore less easy to separate them with distinctness. When death occurs early in the disease it is difficult to ascertain whether the type were intermittent or remittent.

V. Typho-malarial Fever.

The name typho-malarial fever must not imply a specific disease, but rather a combination or coexistence of the two diseases, typhoid fever and malarial fever, in the same individual. It is at best a misleading term, and ought to be abandoned. Nevertheless, the name has entered medical literature extensively, and it is still in common use in a large section of this country.

Typho-malarial fever should not be confounded with the "typhoid condition" which may supervene in protracted and severe remittent fever.

Etiology.—Typho-malarial fever may occur in malarious regions where men are crowded in camps or prisons under bad hygienic conditions and with a water-supply contaminated with sewage. For this reason it is sometimes called "camp fever." In the United States this fever occurs chiefly in the autumn and in the Southern Atlantic and Gulf States. To produce the fever there must be a double infection with the typhoid bacillus and the malarial plasmodium. Unfortunately, positive evidence that these two germs can coexist in the same individual has not yet been obtained, since the natural history of both germs has been studied for only a brief decade. Autopsies, too, are infrequent, for many of the patients recover, and hence the description of the double disease is based solely upon clinical features. In 1888, Johnston[1] published an exhaustive paper containing researches on the question of the separate existence of a typho-malarial fever. The paper was based upon answers received from three hundred and fifty physicians living along the Atlantic and Gulf coasts of the United States and in other malarial regions, and their belief was about evenly divided as to the existence and non-existence of the disease in question as an independent fever.

Accumulating evidence is very convincing that the majority of cases reported as typho-malarial fever are simply modified or irregular forms of typhoid fever, without any malarial admixture. Whatever view is taken, however, of the etiology of so-called typho-malarial fever, it is an undoubted fact that in a malarious region a number of cases occur from time to time which present, from a clinical standpoint, the features of both diseases in combination. These symptoms are as follows:

Symptomatology.—The onset is often more abrupt than in typhoid fever. There are one or more severe chills, and the temperature rises suddenly, and

[1] *Transact. Assoc. Am. Physicians*, vol. iii., 1888, p. 20, *et seq.*

not gradually as in enteric fever. As in typhoid fever, there are frontal headache, epistaxis, sometimes a rose-colored eruption of maculæ upon the abdomen and lower chest or back, tympanites, low muttering delirium, subsultus, diarrhœa. If the disease commences with the typical temperature of typhoid fever, rising slowly during the first week and maintaining a fairly uniform maximum for the next week or ten days, it is soon broken by decided remissions of from two to four degrees which recur at regular intervals. If the malarial type predominate, there may be decided chills, with a maximum temperature of 106.5° F. and sweating, and the remissions are very marked. The spleen is much more enlarged than it often is in enteric fever, and there is more severe gastro-intestinal disturbance, with vomiting of bile, jaundice, and hepatic congestion. There frequently are severe pains in the back and extremities. As a rule, the typhoid symptoms predominate in other respects than the invasion and the temperature, but, contrary to expectation, the supposed union of the two diseases does not appear to be as fatal as enteric fever occurring uncomplicated. According to Woodward, it is less fatal in the proportion of about 1 to 4.

Treatment.—The treatment is a combination of the treatment employed in both diseases. Quinine must be given as in remittent fever. If the fever be high, cold sponge-baths, or if the patient be sufficiently robust cold tub-baths, may be given, with a fluid diet and stimulants. The stools must be carefully disinfected, and complications are to be met as in the case of simple typhoid fever. For further details the reader is referred to the article upon *Typhoid Fever*.

VI. Malarial Cachexia, or "Chronic Malaria."

This condition may occur as a result of repeated attacks of intermittent or of remittent fever, or it may originate in persons living in a malarious locality who have never had the paroxysmal fever in any form. It is more apt to occur among the older residents of an ague region than among new-comers. The condition is quite typical. The face is pale, and the skin has a muddy or yellowish hue. There may be slight jaundice. There is decided anæmia, with attendant disordered digestion. The spleen is melanotic and enlarged, and may be distinctly felt by abdominal palpation. It may even extend as far as the umbilicus. The tongue is large, pale, flabby, and covered with a thick white coating, giving a bad taste and a bad breath. The thin margins are often indented by the teeth. There is more or less gastric indigestion, and the bowels are sometimes constipated or sometimes diarrhœa exists. The circulation is inactive, and the hands and feet are cold. There are dulness, more or less mental depression, and lassitude.

In bad cases there may be dropsy or general anasarca, or a scorbutic condition may develop, with epistaxis, ecchymoses, etc. The temperature is not infrequently subnormal or there may be slight fever of an irregular type. The liver may be enlarged and melanotic, like the spleen.

In obscure cases the detection of the crescentic forms of the malarial germ and of flagellæ in the blood is of great value in diagnosis (Osler).

Secondary diseases, such as dysentery or tuberculosis, may attack cachectic patients, in whom they are very apt to become fatal. While remaining in a malarious region the cachectic are always more or less liable to have a sudden seizure of ague in a pernicious form.

Treatment.—The condition of malarial cachexia requires tonic and hygienic treatment. Such cases are quite anæmic, and iron, arsenic, and cod-liver oil are indicated. There may be constipation, which should be relieved by regulation of the diet and laxatives. Cascara or a pill of aloin gr. $\frac{1}{10}$, belladonna gr. $\frac{1}{10}$, and podophyllin gr. $\frac{1}{10}$, may be given at night for the constipation, and an occasional ten-grain dose of calomel is beneficial. Many cachetic patients make very slow progress while remaining in a malarial district, but improve as soon as they begin to travel, and in very obstinate cases change of locality is the only measure that is effectual in producing a cure.

The spleen is often greatly enlarged, and for this condition ergot is sometimes given internally, and the ointment of the biniodide of mercury or a belladonna plaster may be applied locally. General tonic treatment seems to be more efficacious than any local treatment designed to reduce the size of the spleen.

CHOLERA.

By W. GILMAN THOMPSON.

Definition.—Cholera is an acute infectious disease, occurring both endemically and as an epidemic, and characterized by violent vomiting, purging with serous stools, and by collapse. It is caused by a bacillus, and it has been recently defined as a "specific intestinal putrefaction, with the production of a specific toxine."[1]

Synonyms.—Cholera algida; Cholera Asiatica; Cholera maligna; Cholera infectiosa; Epidemic cholera.

"Cholerine" is a misleading name which is applied either to very mild cases of true cholera, in which the collapse and later typhoid symptoms are absent, or the word is used to designate severe and often fatal cases of diarrhœa accompanying a cholera epidemic. The former usage is German, the latter French.

Recent Epidemics.—Cholera originated in India, in which country alone it is endemic. It has been definitely described since the tenth century, although it was doubtless known long before. The invasion of India by various foreign powers served to extend the limits of the disease, and to-day the enormous religious Eastern pilgrimages are the chief means of spreading the contagion. In the sacred cities of Mecca and Medina, where these pilgrimages are made, sanitation is quite unknown, and cholera frequently becomes epidemic. In Mecca 150,000 sheep are annually slaughtered for ceremonial rites, and the waste portions of the animals rot in the sun and favor the development of fevers, dysentery, and cholera.

For centuries frequent epidemics of cholera have occurred in India, Persia, Mesopotamia, Egypt, and in Russian provinces bordering on the Black and Caspian Seas. The sacred city of Hundwar is the great focus from which cholera radiates throughout India, and Alexandria is a focus for the propagation of European epidemics.

In Bengal the habits of the natives in regard to bathing and washing their utensils in filthy water, and storing water for a long time in unprotected tanks, greatly favor the extension of cholera epidemics.

Cholera first appeared in Europe in 1832, when there were 120,000 victims in France. In the same year it was imported in an emigrant vessel to North America, and the first case appeared at Quebec, whence it spread to New York and elsewhere through the United States. In New York City 3500 died.

[1] *Report on Cholera in Europe and India*, 1890, Edward O. Shakespeare, M. D., United States Cholera Commissioner.

Eighteen years later the disease was again introduced—at New Orleans from abroad. It extended rapidly, and in the ensuing year there were 5000 deaths in New York. Five years later, in 1854, it again developed in that city, causing 2000 deaths. It last visited the United States as an epidemic in 1873. It then occurred simultaneously at several remote localities where it had been conveyed by immigrants. In the United States the epidemics of late years have become gradually milder.

During the period between 1884 and 1887 cholera appeared at various points in Europe, and finally reached Paris, but it was kept out of England by improved sanitation. In 1883 another severe epidemic occurred in Egypt, in which there were 50,000 deaths, and over 600 natives died daily in Cairo alone (Parke). In 1886–87 an epidemic of cholera in Chili caused over 22,000 deaths. In 1890 epidemics of cholera occurred in Natal, Corea, Japan, India, Italy, Spain, and Asiatic Turkey. In Abyssinia 4000 deaths from cholera took place in a fortnight during September, 1890.

During the early summer of 1892 a severe cholera epidemic originated in Meshed in Persia, and extended to Russia by way of the Trans-caucasus railroad. At the present time (August, 1892) it has invaded Nijni-Novgorod, Moscow, and St. Petersburg, and has been carried to Hamburg and Havre. The epidemic is also advancing along the borders of the Black Sea. Meanwhile many deaths from "cholerine" have occurred in the environs of Paris.

Etiology.—Predisposing causes are privation, famine, debauchery, fatigue, debilitating diseases, alcoholism, mental distress, eating decomposing meat or spoiled fruit, drinking unwholesome water. These are merely the common causes which favor the spread of any infectious or contagious disease, and beyond this fact they have no special influence upon the spread of cholera, except in the case of unwholesome food and water, which, in addition to disordering the digestion, may convey the cholera germs.

Cholera has never been known to spread more rapidly than the ordinary rate of human travel by land or sea, and it is essentially a disease of densely populated districts. It advances along the seacoast from town to town, follows the lines of traffic by land and along great rivers, and lingers in crowded cities and encampments. The infectious principle is conveyed by ships, in foul water, soiled clothing, filth of any kind, vehicles, baggage, etc. It has been carried by a box of clothing from Europe to the Mississippi Valley. It is possible that flies serve as carriers of some of the contagion, and it is therefore well to protect food from them and to keep them from access to excrement.

Climate and Season.—It is now generally believed that cholera is not propagated through the atmosphere, but to aid its extension the locality and season must be favorable, for alterations in its intensity are effected by certain meteorological changes. Its spread is not affected, however, by prevailing winds, as it would be if the germ were borne through the atmosphere.

Cholera has been known to occur in all climates excepting the arctic, and

at all seasons, but as a rule its progress is arrested completely by cold, while a warm, moist season favors it. In a given locality an epidemic may cease with the onset of winter, only to be revived in the spring.

Age and Sex.—The disease is of commonest occurrence between twenty-five and fifty years of age. It may occur in childhood, and it is rare in old age. Cholera attacks males somewhat more frequently than females.

Race.—In India, Hindus and Mohammedans are more susceptible to cholera than are Europeans, but this is no doubt due in great part to differences in hygienic surroundings rather than to race influence.

Social Position.—Cholera is essentially a filth disease, and is therefore commoner in the lower strata of society, among the very poor and ignorant. Its chief victims are found in the slums and dirty tenements of the over-populated quarters of large cities. Those who are convalescing from other diseases are liable to be attacked by cholera. Thus at Helonan, near Cairo, in 1883, 9.34 per cent. of such cases were afflicted, but only 2.63 per cent. of the previously healthy were seized (Parke).

The agent which causes the disease is capable of very rapid increase, both within the body and apart from it, and it is discharged from the body in the stools. The evacuations of preliminary choleraic diarrhœa, as well as the characteristic serous stools, are highly infectious. The cholera bacillus undoubtedly enters the system through the medium of contaminated food and drink, especially the latter.

The extension of cholera is so far controllable by proper drainage and sanitation that if ideal hygienic measures could be realized, quarantine would be unnecessary and the disease might be almost exterminated.

In Calcutta the spread of epidemics has been greatly diminished of late years by a more copious supply of water to the city.

Physicians, nurses, and others in attendance upon cholera patients are not especially liable to be attacked by the disease, provided that they are not overworked and that they take proper precautions. They are, however, by no means exempt, and many cases are recorded where the disease has been transmitted by direct contagion. Nurses are more apt to be attacked than physicians, because they are constantly with the patients and have to remove the evacuations. When patients, debilitated by other diseases, have been allowed to remain in wards with cholera patients, inhaling the emanations from their evacuations, they have been frequently attacked by the disease. Those who wash the soiled clothing of cholera patients are often seized, proving that the disease can be conveyed by fomites. It has been communicated through the medium of the mails.

The Cholera Bacillus.—Animals were at first thought to be immune to cholera, but recent careful experiments upon dogs and guinea-pigs have proven the contrary, and the disease has been demonstrated in them after inoculation (Pasteur).

Koch,[1] in 1884, was the first to describe a distinct bacillus associated with

[1] *Berliner klinische Wochenschrift*, March 31, 1884.

cholera. In 1885 he announced that he could reproduce cholera in guinea-pigs without making the inoculations directly into the intestine.

It is believed that, whether inhaled in the mouth or swallowed with food or drink, the germ must always first reach the intestine by way of the stomach before becoming active, and that the alkaline intestinal contents constitute its most favorable habitat. It is found most abundantly in the ileum, on its surface, and within the tubules of the mucous glands. The bacillus which was discovered by Koch in the stools of cholera patients presents the following characteristics: It is from one-half to two-thirds the size of the tubercle bacillus, but thicker and somewhat curved, resembling a crescent, comma, or half circle, or a double curve like an S. (See Fig. 28). When freshly obtained from the stools the length of the bacillus seldom exceeds 1μ, and many are only $.5\mu$ long. The germs are frequently aggregated in small groups or arranged in spirals, when they resemble the genus Spirillum.

Fig. 28.

Comma-shaped Bacillus of Cholera (Koch).

The bacillus grows rapidly in and upon various culture media. It thrives in alkaline nitrogenous media, such as milk, meat juice, or peptone, and it grows well in a slightly alkaline gelatin, which it causes to liquefy. It also grows upon solid substances, such as potatoes, damp dirty linen, and moist earth. A decided acid reaction of the culture medium stops its reproduction; hence it develops in the intestine, and not in the stomach. It is destroyed by drying for a short time and by a temperature of 143° F. (Sternberg), but not by freezing to even —10° C. It exhibits one or two cilia at one end, and is actively motile in the fluids in which it grows. It reproduces by fission with enormous rapidity, and spores have not been identified with it. The bacillus thrives in foul water, especially briny water, and Koch considers the Delta of the Ganges to be its natural home. This bacillus is so constantly associated with cholera stools, and so constantly found in the intestines of those dying of cholera, that it may be regarded as causative as well as pathognomonic of the disease.

According to Klein and Gibbes, who investigated the cholera in India on behalf of the British government, there are several allied species of the bacillus, which differ in their size, mode of growth, and effect on the lower animals.

Bacilli which closely resemble the cholera bacillus in appearance have been found in the saliva and in some healthy stools, and in the alvine discharges of diarrhœa and dysentery; hence the cholera bacillus may be overlooked, for, while it only occurs in Asiatic cholera, it is not present when the stools become normal, or even in the diarrhœa of convalescence. At the latest, it is to be

detected ten days after the commencement of an attack, and it often disappears by the fourth day of the disease. It is apparently shorter-lived than many bacilli. To facilitate its detection cultures should be made from the stools and frequently repeated in order to keep the germs alive for any length of time.

Microscopic examination alone does not establish its identity, but it may be cultivated and inoculated in the lower animals, and through such inoculation, together with its behavior in the various nitrogenous culture media, it is possible to demonstrate that the germ is typical. Since the germ multiplies rapidly in the intestine (which contains little or no free oxygen), it follows that it is anaërobic; but it has been proved that immediately after leaving the intestine it is more easily destroyed by various agents, such as the acid of the gastric juice, than after it has been exposed to the air for some time, when it becomes aërobic. Hence the practical importance of the immediate disinfection of all choleraic discharges as soon as they are voided.

As is the case with several other infectious diseases, when a warm, dry season closely follows a very wet one cholera becomes more active and virulent. This is explained by the fact that such conditions promote putrefaction and fermentation and furnish favorable products on which the germs thrive. It needs but the access of a few germs to such a soil to soon contaminate a very extensive area.

The facts which support the belief in the bacillus of Koch as the cause of cholera are as follows:

I. It is the almost invariable accompaniment of the stage of collapse.

II. It is not found apart from the disease, and disappears with it.

III. It occurs in the stools and in the small intestine, which is the region particularly affected by the disease.

IV. When inoculated in certain of the lower animals it produces symptoms similar to those of cholera, with collapse and death.

Since the germs are not found in the tissues generally throughout the body, it seems probable that they produce a poisonous substance, a toxalbumin, or ptomaïne in the intestine, which, on being absorbed, occasions the constitutional symptoms of the disease. Similar action has been clearly demonstrated in enteric fever and diphtheria. Ptomaïnes and toxines have been isolated from cultures of cholera germs, which, injected into animals, cause fever, cramps, diarrhœa, and collapse.

Gamaleia and Lowenthal have succeeded in rendering certain animals immune to cholera by the use of attenuated cultures.

The following is a summary of the present beliefs in regard to the nature of cholera which have been discussed in the previous pages:

The disease is due to a specific virus—namely, a germ which enters the body through the alimentary canal and attacks the small intestine, where it develops ptomaïnes, which on being absorbed into the system produce constitutional symptoms. The disease is propagated by fomites and by direct contact with the stools. The chief agent for its dissemination is contaminated drinking-water. The contagion multiplies with extreme rapidity both inside

and outside of the body, and it thrives especially in warm, moist putrefactive organic matter.

Morbid Anatomy.—The local action of the morbific agent of cholera is chiefly directed against the epithelia and subjacent tissues of the small intestine, especially its lower end. The general or constitutional lesions are not distinctive. They are produced through the influence of poisonous material absorbed from the intestine which chiefly affects the vaso-motor centres and other parts of the nervous system.

In an autopsy made half an hour after death in a typical case of cholera Milles noted the following appearances: The small intestine was of a rose-red color, and distended as if paralyzed. It contained a typical clear stool. The mucous membrane was swollen and denuded of its epithelium. The follicles were filled with epithelial detritus and comma bacilli, and their orifices appeared as red spots. Comma bacilli were also discovered in the subepithelial tissue, to which they are supposed to penetrate by their own activity.

In those cases in which death occurs very early in the course of the disease there are no pathological changes. Rigor mortis appears early, and lasts during a longer period than usual. There may be post-mortem spasmodic muscular twitchings, lasting for two or three hours. In typical cases there is dryness of all the tissues, including the muscles, connective tissues, and skin. As a result, decomposition proceeds very slowly. The serous outpouring in the intestine must be regarded as a simple transudation, rather than as the result of a true inflammatory process.

In typical cases the mucous membrane of the small intestine, especially the ileum, is congested or soft and œdematous, and it is frequently the seat of ecchymoses. The villi are swollen, stripped of epithelium, and the blood-disks in their capillaries are destroyed and free pigment is found (Sutton). The congestion may extend over a large part of the intestine, or it may nearly surround the swollen agminated or solitary glands. There is sometimes croupous inflammation of the large intestine, with necrotic changes at the surface of the membrane. The mesenteric glands are enlarged. There may be more or less gastric catarrh, with congestion and abrasion of the mucous surface. The serous membranes, such as the pleura and peritoneum, are dry or covered with a layer of sticky albumin. The brain and its membranes may appear normal or very dry, and the pia may be œdematous or ecchymotic. The cerebral sinuses contain thickened, dark blood. The spleen and liver are either normal or anæmic or the seat of parenchymatous degeneration. Anæmia of the liver results from the drain upon the intestinal division of the portal system and paresis of the vessels. The kidneys present the appearances commonly produced in the course of infectious fevers. The tubules contain desquamated epithelium and hyaline, granular, or fatty casts. The cortex is often thickened and the pyramids are congested. The bladder is contracted and empty. The heart is soft, and there may be ecchymoses in the pericardium. When the patient has died of asphyxia the left ventricle, as usual in such cases, is comparatively empty, while the right ventricle is over-distended

with dark blood. The thickened blood coagulates more slowly than usual, and, owing to diminution in the quantity of fibrin, the clot is less firm. The solid ingredients are present in quantity one and a half times greater than normal (C. Schmidt), and the chlorides are found to have transfused into the intestine, leaving a relatively larger quantity of phosphates behind. The red blood-corpuscles appear shrivelled, and both red and white corpuscles are apparently increased in number on account of the diminution in serum. The lungs are contracted, dry, pale, and anæmic. Pulmonary œdema is rare. Congestion is sometimes found at the bases of the lungs. The lungs may weigh as little as twenty ounces (Sutton).

Symptomatology.—The latent period or incubation which intervenes between the time of infection and the development of the first symptoms is not accurately known. In a majority of cases it is two or three days; it may, however, last for a fortnight.

The symptoms of cholera may be due in part to toxæmia and in part to the sudden and extreme drainage of water from the system. Thus by some observers the final collapse is attributed to toxines in the circulation which cause vaso-motor spasm and impeded pulmonary circulation (Johnson), while others believe it to be due to the exhaustion occasioned by the profuse watery evacuations which cause desiccation of the nerves and other tissues of the body. It is convenient to divide the symptoms into four typical stages, as follows:

I. The premonitory diarrhœa;
II. The stage of serous diarrhœa;
III. The algid stage, or stage of asphyxia or collapse;
IV. The reaction.

These stages usually occur in the order mentioned, but any one may be omitted. Thus, the premonitory diarrhœa may be absent when the disease begins with the second stage, or it may be the only symptom present in certain cases during the progress of an epidemic.

Whether this stage develop or not, the onset of the disease is usually sudden, and in the majority of cases the invasion occurs in the night. More rarely there is an indefinite prodromal period of a day or two, with more or less prostration, vertigo, anorexia, and gastric oppression with flatulence.

I. The *first stage*, when present, commences with a diarrhœa, and the stools are alkaline, watery, yellowish or pale, very profuse, and frequently voided. There may be fifteen or twenty within twenty-four hours. These stools are quite as dangerous as regards spreading the contagion as are those of the fully-developed disease. There are borborygmi, but no severe colic. There is frontal headache, and there are nausea and possibly vomiting. There are apt to be mental depression and a feeling of dread. The tongue is clean, pale, and moist at first, but later becomes dry. Thirst is present and the voice grows faint. These symptoms may continue for a day or two, or even for four or five days, and either end in recovery or merge into the second stage.

II. The *second stage* presents very active and alarming symptoms. If diar-

rhœa has pre-existed, it continues or it begins anew. In either case the stools are very copious, alkaline, and watery, and their passage is painless. They are often excited if the patient turn over in bed or if pressure be made over the abdominal wall. The stools of the premonitory diarrhœa are sero-mucous and contain more or less bile and fæcal matter. They are soon followed in the second stage by more typical evacuations. The typical cholera stools are voided after the bowels have been emptied of their ordinary fæcal contents, and are usually described as having the appearance of "rice-water" or "macaroni-water." They have no odor or only a faint "meaty" smell. They are frothy, and contain no bile or fæces, but show, in suspension in an opalescent fluid, whitish flakes which are composed of desquamated intestinal epithelium. They deposit considerable sediment on standing. It is observed by Milles that the stools are almost transparent, and thus differ in appearance from rice-water, which is opaque. They are occasionally colored by extravasated blood, in which case they are described as resembling the lees of wine. The specific gravity of the rice-water evacuations is 1005–1013 (Flint). The reaction is alkaline from ammonium carbonate, but the principal salt is found to be chloride of sodium. The quantity of the fluid discharged in a single evacution may exceed two quarts, and its passage often is followed by a temporary feeling of ease.

The typical cholera stools contain a lesser variety of micro-organisms than is found normally, and they yield an almost pure culture of the comma bacillus. The germs are not found in any quantity until the stools become characteristic. Later, in the stage following the collapse, the stools become darker, even brown or black or bloodstained, and they are slimy and very malodorous. It is believed that the cause of the excessive transudation is a paralysis of the intestinal nerves (Brunton). As soon as a considerable quantity of fluid has been drained off in the frequent serous evacuations the thirst becomes more and more intense, so that drinking water does not relieve it. There is rapid emaciation, and in a few hours the victim, who may have been previously robust, presents the aspect of an emaciated and old man, with loose, wrinkled, inelastic skin, sunken, glaring eyes, and a parched, dry tongue. The scrotum is markedly retracted. The stomach is highly irritable and rejects nearly everything which is swallowed. There are nausea, sudden vomiting, and epigastric distress. The ejecta consist at first of the food or other contents of the stomach, then become bilious, and finally are clear and transparent, with flocculi of mucus resembling the rice-water stools. The quantity of fluid vomited exceeds that which is drunk. The vomiting may occur only at the commencement of the disease, but it usually continues. The urine is thick, turbid, and contains an excess of urea, and later more or less albumin, with granular and hyaline casts. Urea is said to be also eliminated by the stomach, and in increased amount in the perspiration. The pulse becomes small and feeble, and arterial tension is diminished. The diarrhœa is finally accompanied by abdominal cramps, and the pains extend down the legs and become agonizing. The patient is restless, anxious, and distressed, and becomes more and more

feeble and prostrated. The pulse becomes very weak and accelerated, and the respiration may be shallow and somewhat increased in frequency. All this occurs in a few hours' time. The temperature of the surface is low and the skin feels cold to the touch, but the internal temperature is elevated. A cold, clammy perspiration frequently covers the entire surface of the body.

The mind, as a rule, remains unclouded, but the voice is feeble, husky, and high-pitched. More or less complete suppression of urine ensues, due either to a loss of water or to local action of the poisonous products of the disease.

There is an occasional variety of cholera which is very fatal, and in which the diarrhœa is wanting but the other symptoms are present. This is known as "dry cholera" or "cholera sicca." In these cases the intestine is found at the autopsy to be greatly distended with serous exudate. Hence the amount of diarrhœa is not an infallible indication of the severity of the disease. Sometimes in these cases the patients are seized with great prostration. While walking about they suddenly become faint, dizzy, and unable to stand. There are headache and mental confusion. In the worst cases the prostration rapidly increases, and the patient dies in two or three hours.

In the serous stage the amount of water lost from both stomach and intestines is very great. It comes away in gushes, frequently from both stomach and rectum simultaneously, or it may flow from the rectum in an almost continuous stream. There may be spasm of the diaphragm, producing hiccough, and often the abdominal muscles become tense. The spasms and cramps of the various muscles become extremely painful, particularly in the legs. These spasms may be explained in one of three ways: they may be due to toxic products in the blood, to reflex gastro-intestinal irritation, or to desiccation of the nerve-centres (Weir Mitchell). The latter explanation is probably the most correct. There are sometimes involuntary contractions of the flexors and extensors of the digits. The serous stage lasts during one to three hours or longer.

If the symptoms are very violent the second stage will be more brief, on account of the exhaustion of the patient. It is followed sometimes by reaction, but more frequently by the stage of collapse.

III. *The Stage of Collapse.*—In this stage the prostration, emaciation, and enfeebled heart-action continue. The face becomes shrunken and expressionless, the cheek-bones are prominent, the cheeks are depressed, the nose is sharp, the eyes are dry and hollow, and the whole physiognomy is highly typical of the disease. The deeply sunken eyes are half closed and surrounded by dark circles. The forehead is wrinkled, the lips are thin and set, the fingers are shrivelled, and the radial pulse is scarcely perceptible. The skin is dusky or blue, and feels very cold to the touch. A sudden increase in the diarrhœa or vomiting is apt to be accompanied by a rapid fall in the surface temperature (Shakespeare). The oral temperature falls to 90° or 95° F., and it has been observed as low as 79° F., and the axillary temperature may fall to 75° F., but the fact of a rise of deep internal temperature has been confirmed recently by a number of competent observers (De

Renzi, Guterbock). A thermometer carefully passed high up into the rectum may record an elevation of temperature amounting to two degrees above the normal. The patient complains of a sensation of internal heat. The fever is said to be of a remittent type with evening exacerbations. It is often overlooked on account of the stone-cold feeling of the surface and from the difficulty of taking the temperature in the rectum while the stools are being almost constantly voided. The vomiting and diarrhœa finally cease, apparently from exhaustion or because there is no more material to be discharged. The tips of the fingers and toes become livid and rigid, and the breath feels cold. The respirations are shallow and irregular, and dyspnœa is often extreme. The loss of so much fluid from the blood through the alvine evacuations causes diminution in all other secretions of the body, excepting sometimes the perspiration. The tears, saliva, and bile are withheld, and menstruation is checked. The dryness of the eyes may result in their inflammation from exposure to the air (Stillé). The suppression of urine continues, and it may become complete. If any urine is voided, it is albuminous, and frequently contains sugar.

The mind remains intelligent, but the patient is too feeble and too greatly prostrated in every way to speak or to take note of his surroundings, and lies as though dead. The blood, thickened by deprivation of so much water, flows but slowly through the capillaries and lingers in the veins until it becomes highly venous all through the body, and imparts a cyanotic hue to the entire surface, and the condition of asphyxia ensues, which Brunton believes is due to spasm of the pulmonary vessels preventing the free flow of blood through the lungs. The post-mortem appearances of the lungs confirm this view. There is less interchange of the gases of respiration than there should be, and elimination of carbon dioxide is diminished. The pulse is feeble, not usually above 100 or 120, and it may fail entirely at the wrist. The second sound of the heart is inaudible. The entire body is shrunken almost beyond recognition.

This stage lasts for several hours as a rule, or it may be protracted for a day or two, and it either terminates fatally or in a reaction with recovery. Death may be due to asthenia or to asphyxia, but profound coma is uncommon. It may come suddenly, without premonition, and patients in whom the symptoms have not been very severe have been known to jump out of bed and walk about just before death overtook them. More often death supervenes gradually with progressive coldness of the surface and insensibility. When it results from asphyxia from stagnation of the blood, the temperature usually rises and may reach 108° F. In other cases, after death the temperature may continue to rise to 106° F., and there may be post-mortem contractures of the muscles of the extremities and of the facial muscles, producing grimaces.

It will be observed from the foregoing account that nearly all the symptoms of cholera are induced by the excessive loss of fluid from the blood. This occasions the dryness of all the tissues, the diminution in biliary, renal,

and other glandular secretions, the paralysis of the nervous system, the exhaustion, and the asphyxia.

IV. *The Reaction.*—The great majority of patients who recover pass through a reactionary febrile stage. The reaction follows either the stage of serous diarrhœa or the collapse. The external temperature rises, while the internal temperature falls, and the condition of the circulation is gradually restored to the normal. The face regains its natural expression. The cramps and vomiting cease. The pulse becomes slower and of better volume, and thirst is no longer complained of. The stools become of firmer consistence, and finally resume their fæcal character and contain bile-pigment, except in those cases in which there has been extensive denuding of the epithelial surface of the intestine, when the stools may be hæmorrhagic for some time. The secretion of the urine is gradually restored. In some cases convalescence is interrupted by absorption of septic matter from this denuded surface, and a typhoid condition or septic fever results, with considerable elevation of temperature, a dry tongue, delirium, and coma. Various cutaneous eruptions may accompany the fever. If, on the other hand, convalescence be not delayed by typhoid symptoms, there are usually pronounced anæmia and prolonged irritability and feebleness of the digestive organs and of the nervous system. There are severe frontal headache, and often vertigo and fainting. In still other cases uræmic symptoms develop, the function of the kidneys not having been restored. In such instances nervous symptoms predominate, and there is delirium with convulsions.

In some cases the intestines fail to recover their tone, and an exhausting diarrhœa still further debilitates the patient. Relapses may occur, either as a result of indiscretion in diet or exertion or without assignable cause.

In the variety of disease called "cholerine" the symptoms are comparatively mild, although they may last for a week. Asphyxia, cramps, and total suppression of urine are sometimes absent.

Complications and Sequelæ.—Diarrhœa, dysentery, and malarial fevers are apt to prevail in the same localities with cholera and at the same time with cholera epidemics, though the latter disease is so acute that it is rarely complicated by other affections unless they already exist in the individual attacked. Various exanthemata, such as roseola, urticaria, etc., may occur during the reaction or the convalescent period. Sometimes furunculosis and ulceration ensue, especially upon the emaciated extremities. Bed-sores are apt to occur. Excessive perspiration, with elimination of increased urea, is sometimes observed. There may be painful swelling of the parotid glands, rarely progressing to the formation of abscess. Sometimes a painful contraction of the muscles of the extremities resembling tetanus takes place, and lasts for several hours or a day or two. Gangrene and peritonitis have been rarely present as sequelæ. Corneal ulcers sometimes appear. More or less gastro-intestinal irritability, with gastralgia and anorexia, is apt to remain, and it may last for many weeks, greatly retarding convalescence. There may be enfeebled circulation, with cold extremities and wakefulness. Various kidney

lesions have sometimes followed, and cerebral congestion may be a sequel to the reactionary period. Pneumonia is an occasional complication.

Duration.—Fatal cases end usually in two or three days. Death has occurred within two hours after the first typical stools have appeared. In such cases all the symptoms of the algid stage occur with incredible rapidity. The duration of a single epidemic is often brief, and it seldom remains a month in any one locality.

Diagnosis.—Cholera is liable to be confounded with one or two other diseases in its early stage and before the epidemic has been recognized. After the establishment of an epidemic the disease is, however, readily identified by the typical stools, rapid emaciation, great thirst, prostration, and algid condition. The diseases and conditions with which cholera may be confounded are septicæmia, typhoid fever, pernicious malarial fever of the gastro-enteric variety, cholera nostras, and ptomaïne or mineral poisoning.

In the typhoid or reactionary stage of cholera the patient is really in a septic condition, and the prostration, emaciation, and general ataxic condition suggest enteric fever. The latter disease, however, has a protracted history: there is a characteristic temperature curve, a rose-colored abdominal eruption, and diarrhœa, if present, is of a different sort, and the stools are less watery and colorless than in cholera. The gastro-enteric variety of malarial fever is often so severe as to resemble cholera in its earlier stage. In the former the temperature is high, 106° or 107° F.; the stools may be bloody at first; vomiting, if present, is more painful, with decided retching; and free pigment is found in the blood, with possibly the malarial plasmodium.

Very severe cases of cholera morbus may prove fatal in one or two days, and every symptom of cholera may be present, rendering diagnosis extremely difficult. Fortunately, such cases are quite exceptional. Usually in cholera nostras, or sporadic cholera, the symptoms of extreme cyanosis and total suppression of urine are wanting. The alvine evacuations are loose and watery, but unlike the typical "rice-water" stools. The disease is less rapid than true cholera in its progress, much less severe in its symptoms, and recovery is more frequent. The cases are isolated and non-contagious. The typical cholera bacillus is absent. The cramps are apt to be more severe in the stomach, but less severe in the legs, than in true cholera.

Asphyxia from coal-gas (CO) poisoning may produce symptoms resembling the asphyxia stage of cholera, but the absence of intestinal irritation and of the typical choleraic stools will at once confirm the diagnosis.

In cases of mineral poisoning there may be visible corrosion within the mouth, a metallic taste, and the epigastric pain and burning is very pronounced. The stools are bloody or fœtid, instead of clear and watery. Among such cases the symptoms produced by arsenic are the most difficult to differentiate from those of cholera, for in both instances there may be great prostration, collapse, thirst, cramps, and suppression of urine. In acute arsenical poisoning there is usually constriction felt in the throat or œsophagus, and there is epigastric pain of an intense burning character.

The symptoms commonly follow very soon after the taking of drink or food.

In any doubtful instance the discovery of the cholera bacilli in the stools will decide the case.

Prognosis and Mortality.—The prognosis depends upon the severity of the epidemic, the sanitary condition of the environment, the habits of life, and the promptness with which the patient comes under treatment. For cases seen very early the prognosis is good. It is bad in densely-populated districts, and worse near the seacoast than inland.

The mortality from Asiatic cholera remains practically unreduced by every effort of treatment, although if seen early and faithfully treated many cases may be brought to recovery. The disease, once established among those of filthy habits, is, however, nearly as fatal as ever. The total number of cases occurring in a given locality is diminishing where hygienic laws are duly respected. Thus, in 1868 the cholera mortality among foreign soldiers in India was 18.6 per 1000, while to-day it is only one-sixth as great. The general mortality varies between 20 and 80 per cent. with different epidemics, but it is always high, and the hospital death-rate may often exceed 60 per cent. It has been as grave as 90 per cent. The worst mortality occurs during the earlier and middle period of an epidemic: toward the end the cases are less fatal. The disease is very fatal in childhood and old age, but is less common among such subjects; hence the mortality is greatest in adult or middle age. An epidemic may affect a very large number of persons, and yet the death-rate may be lower than in a less extensive epidemic.

Prophylaxis.—The prophylactic treatment consists in a rigid enforcement of sanitary rules and personal hygiene. All healthy persons should, as far as practicable, be removed from the infected district. Great importance attaches to immediately stopping any diarrhœa which occurs while an epidemic of cholera is prevalent, and to further this end it is advisable for the local government to appoint special medical inspectors to go from house to house. In this manner many lives may be saved.

The digestion should be particularly cared for, and some advise the internal use of dilute acids to maintain a moderate hyperacidity of the stomach, which is thereby rendered inimical to the germs. Fatigue and excesses of all sorts should be strenuously avoided. All sewers, privies, cesspools, and water-tanks should be thoroughly cleansed and disinfected. Drinking of impure water should be avoided, and all water should be thoroughly boiled before use. During the prevalence of an epidemic all public funerals or large gatherings of people should be absolutely interdicted.

Treatment.—No one drug or system of treatment has proved of much avail for cholera. The objects of treatment are, therefore—(1) to support the strength; (2) to allay pain and fear; (3) to relieve the severity of certain symptoms, notably the thirst, emesis, diarrhœa, and cramps; (4) to prevent thickening of the blood and suppression of urine. The treatment must be adapted to each stage of the disease.

Treatment of the First Stage.—If promptly taken in hand at the outset and carefully watched and nursed throughout the disease, a certain proportion of cases may be saved, and in some the disease is apparently aborted. With the first indication of diarrhœa the patient must go to bed and remain there, warmly covered. He must be kept absolutely quiet throughout the disease. Hot stupes may be placed over the abdomen and food should be withheld. A dose of laudanum or chlorodyne is to be given at once, and then salol or salicylate of bismuth may be administered every two hours. It is believed that the salol acts as an antifermentative and prevents the absorption of ptomaïnes from the intestine. In many cases this prompt treatment will stop the diarrhœa, and the disease may advance no farther. In the early stages of the disease it is useless to attempt to give food. The stomach is too irritable, and broths and milk serve only as culture media for the further development of the bacilli.

Treatment of the Second Stage.—If, on the other hand, the diarrhœa progresses, and the alvine discharges become serous and are accompanied by cramps, more active measures are imperative, and every effort should be made to keep up the patient's hope and courage for the struggle which is before him. The abdomen may be wrapped in flannel, or turpentine stupes are useful if the abdominal pains are severe. For the vomiting morphine should be given hypodermically, and a mustard paste placed over the epigastrium. Cracked ice, cold lime-water, carbonic-acid water, or iced champagne sometimes afford relief. When it can be obtained, fresh lime-juice, iced, is very serviceable. Cocaine in small doses sometimes allays the gastric irritability. The cramps in the calves of the legs and other muscles may become so intense as to require inhalation of chloroform for their relief. Kneading the muscles is sometimes of use, as well as rubbing them with mustard-water and applying hot-water bottles and turpentine stupes to the legs.

It is strongly recommended by certain writers of extensive experience to place the patient in a hot bath, at 106° or 108°, for twenty minutes during the stage of cramps and commencing serous diarrhœa. The patient is then put back to bed, rubbed dry, wrapped in warm blankets, and is given warm, stimulating, and aromatic drinks. Of course he should be moved as little as possible, and should be lifted into and out of the hot bath. Should his condition warrant it, the bath is to be repeated once in two or three hours. It quiets the nervous system, arrests the vomiting, controls the painful cramps, restores the skin to a more natural condition, and stimulates the circulation; besides which it is usually very grateful to the patient. For the agonizing thirst cold water, acidulated with a little dilute hydrochloric or phosphoric acid or lemon-juice, should be given. Cracked ice is useful, but the relief afforded by it is slight. Carbonic-acid water or seltzer may also be given. Fluid held in the mouth for ten minutes at a time affords more relief than when immediately swallowed, by giving a sort of local bath to the parched tongue.

The serous diarrhœa is not readily controlled by remedies administered by the mouth, because they are scarcely absorbed at all, but enemata of ten

grains of lead subacetate or of tannin, in four ounces of water with fifteen or twenty minims of tinct. opii, may be employed with advantage.

In order to replace the fluid which drains away in such large amount in the serous stools, and to restore the balance of the blood-pressure, volume, and density, it is necessary to put back fluid into the body in some manner. The stomach, owing to excessive irritability, is useless for this purpose, and two other methods have therefore been recently advocated by Cantani of Naples, and extensively tried by himself and others with very favorable results. The first is termed "entero-clysis," and is adapted to both the second and third stages; the second, "hypodermo-clysis," is adapted to the third stage. Entero-clysis is performed by injecting the following solution through a long, flexible rubber rectal tube, which may be passed up carefully for a foot or more into the gut:

℞. Boiled water or infusion of chamomile, 2 litres;
Tannin, 5–10 gr.;
Laudanum, 30–50 gtt.
Powdered gum arabic, 50 gr.—M.
Sig. Inject per rectum immediately after an evacuation (Shakespeare).

Treatment of the Third Stage by Hypodermoclysis.—This operation is described by Shakespeare as follows: The object is to inject large quantities of fluid beneath the skin for absorption into the blood-vessels and lymphatics. A large fold of skin is raised between the thumb and finger in the inframammary or ileo-costal region, and a canula is inserted between the skin and subcutaneous fascia. The canula is connected by rubber tubing with a fountain syringe, and the contents of the latter are allowed to slowly flow in by gentle force of gravity until one or two litres of fluid have been injected. The skin over the site of the injection may be rubbed a little in order to distribute the fluid. The injection should be made slowly, and twenty minutes or half an hour will be required for the process. Of course the ordinary precautions taken in any transfusion operation must be observed as regards prevention of entrance of air and antiseptic cleanliness of the instrument employed. A stopcock should be arranged to control the volume of fluid. The salt solution injection is prepared as follows:

℞. Chloride of sodium, c. p., 80;
Sodium carbonate, 6.
Sig. Dissolve in two litres of boiled water, and inject at the normal temperature of the blood.

The indications for the use of this method of treatment are the signs of lack of water in the vascular system, cramps, a cold, discolored skin, rapid emaciation, and copious serous discharge. It is said to produce very striking effects, and to arouse patients from profound collapse and cure them. The water thus supplied to the system through the rectum and the skin aids in

washing out the waste matter formed in the body and in eliminating the toxic principles which have been absorbed and are overwhelming the system. The kidneys are thus restored to their natural activity. Should a favorable reaction not follow almost immediately, and if the skin remain cold, the subcutaneous injection must be repeated.

Atropine has been combined with opium, with the idea of counteracting the ptomaïnes produced in the intestine, which resemble muscarin in the intensity of poisonous effect (Brunton). Salol, naphthaline, creolin, and allied remedies have been given internally, with the object of checking the diarrhœa, of controlling intestinal fermentation, and of preventing the formation of ptomaïnes. Of these remedies salol is the most useful.

In the stage of collapse every effort should be made to maintain the circulation until the danger is passed, and most prompt and vigorous action is necessary or the patient will die in two or three hours. Hypodermic stimulation may be pushed with sulphuric ether, strychnine, brandy, camphor, caffeine, or morphine. Hot-water bottles, hot-air baths, and hot fomentations are required. The perspiration should be wiped off as it forms. Internally, warm alcoholic stimulants, camphor, and ammonium carbonate may be given, provided the stomach will retain them. Intravenous and intraperitoneal injections have been attempted, but with little result. Inhalations of oxygen or of amyl nitrite have proved useful in some cases of collapse.

In convalescence fluid nourishment only should be given, and this at frequent intervals and in small amount, increasing the quantity and diminishing the frequency as the patient improves. The stomach remains weak and irritable for so long a period that solid food must be postponed for many days, and milk, beef peptonoids, and nutritious broths, with champagne, should be the dietary limit at first. Strychnine and other bitter tonics are helpful.

Frequent examinations of the urine should be made, and attention must be paid to the restoration of the functions of the kidneys. Mild diuretics and saline effervescent waters may be indicated for this purpose.

The disinfection required after a case of cholera has terminated is extremely thorough. Rooms and hospital wards should be fumigated; the floors and walls and furniture should be washed with solutions of corrosive sublimate, 1 : 1000, or other disinfectant; and the walls should be rubbed down with bread to remove any dust or germs which may cling to them. The patient should be bathed and then sponged with a 1 : 1000 corrosive-sublimate solution (care being taken to keep it from the eyes). Bedding and clothing should be destroyed by fire when possible or disinfected in superheated steam. Linen must be boiled.

It must be constantly borne in mind that cleanliness is absolutely essential, and too much reliance is not to be placed upon disinfectant materials. The stools and vomited matter must be treated with strong disinfectants. All dejections should be received in a vessel containing either a 1 : 1000 corrosive-sublimate solution or a 5 per cent. solution of carbolic acid to which a little crude hydrochloric acid has been added. This solution should equal

half the volume of the stool. Schauz advises the addition to the stool of one-sixth of its volume of a solution made by adding 100 gr. of crude sulphuric acid to a litre of water. After disinfection the stool should be buried in a trench or mixed with sawdust and burned (Stillé), for the acid is ruinous to drain-pipes. It should be remembered that the germ is relatively feeble when first voided, and the stools are therefore rendered more thoroughly inert if disinfected at once. Soon after exposure to the air the germ increases in virulency.

The question of quarantine has given rise to much discussion, but it cannot here be argued in detail. It has often proved ineffectual, and many believe that, so long as their clothing and luggage are disinfected, apparently healthy persons coming from an infected district need not be detained at all. On the other hand, it is an established fact that the disease may be spread through the agency of the premonitory diarrhœa before the individual really feels ill at all, and the fact that quarantine is often inefficacious is no argument against employing it as rigidly as possible.

When there has been an outbreak of cholera upon a vessel it should be most thoroughly fumigated and cleansed, and all persons on board must be removed and strictly isolated for not less than a week or ten days, while all their clothing and luggage is disinfected.

YELLOW FEVER.

By W. GILMAN THOMPSON.

Definition.—Yellow fever is an acute, highly infectious, but non-contagious, disease, characterized by a sharp febrile paroxysm, black vomit, jaundice, and suppression of urine. It is both endemic and epidemic, and is confined within certain geographical limits. The first recorded epidemic occurred in Barbadoes in the year 1647.

Synonyms.—Black vomit; Yellow Jack; Bronze John; Febris flava; Hæmo-gastric fever; Gelbfieber (Ger.).

Etiology.—The mode of propagation of yellow fever, as well as its behavior as a disease, leaves no reasonable doubt that it is of germ origin. Several bacteria have been described as pathognomonic, and in Havana since 1854 and in Brazil protective inoculations have from time to time been practised on man, but the most experienced bacteriologists have been unable to confirm the specific character of these germs, and we are still in ignorance as to their nature and appearance. Several times organisms found in yellow fever which were supposed to be specific have been subsequently identified with germs occurring in other affections. Various micro-organisms have been isolated from the liver and kidneys of patients dying of yellow fever, and Sternberg found a bacillus somewhat resembling that of cholera. He reports finding in the liver and intestines, after death from yellow fever, one germ which he considers unique. Cultures of the germ must be made very soon after death, and comparatively few specimens are found. It is supposed that yellow-fever microbes may enter the lungs through the air and be transmitted thence to the blood, or possibly, entering the mouth, may convey the germ to the saliva, which furnishes a culture medium. Thence they may enter the alimentary tract and be absorbed.

There are some features of close analogy between yellow fever and the Southern cattle plague or Texas fever, which is also a germ disease.

It is claimed in Havana that mosquitoes after biting a yellow-fever patient are capable of inoculating with the disease the next healthy person whom they attack (Finlay).

The true home of yellow fever is in the tropics. It extends to the temperate, but never to the frigid, zone, and it is more restricted in its distribution than any other acute infectious disease, showing at times very curious localization. It is common in Mexico, and is endemic in the West Indies, Panama, and on the east coast of South America, where it periodically becomes epidemic, and thence is conveyed in ships or by merchandise to other

countries to promptly establish a local epidemic. It is thus occasionally found in Southern Europe, the west coast of Africa, and on the southern and eastern coasts of the United States, where it is not endemic or sporadic.

When introduced by vessels into the United States it prevails chiefly in July and August. There was a very severe epidemic in Florida in 1887–88, and one in Brazil in 1889. In 1890 it prevailed in Brazil, Costa Rica, Cuba, Mexico, the United States of Colombia, and cases were detained by quarantine in New York and elsewhere. Cases have been carried as far north as Quebec. In the United States the disease has never become really endemic, although at New Orleans it has been imported in forty-eight different years out of the first sixty years of the present century (Sternberg).

A single epidemic may last a few weeks or two or three months, until its energy is spent or cut short by frost. The average duration of yellow-fever epidemics is forty-eight days (Barlow).

The following facts are admitted by those who have had the most extensive experience with the disease: The infected area extends rapidly, but is checked by streams, high walls, and sometimes by thoroughfares. Hence the poison keeps near the ground. Immunity increases with elevation above the sea, and it is almost complete at 3000 feet. The majority of cases occur below 750 feet. Exceptionally, however, the disease has appeared at much higher elevations—for example, at Cuzco in the Peruvian Andes, which is over 11,000 feet above the sea.

The virus is destroyed by extreme heat and by cold. One or two frosts stop an epidemic immediately, but freezing does not necessarily destroy the germ. Epidemics stopped by a severe frost have spread again with renewal of a warm temperature, as was the case at Memphis, Mississippi, in 1878–79. The disease has existed when the mean temperature was as low as 50° F. (Forrest), and epidemics may continue at 60° to 70° F., but they are more virulent at a temperature five or ten degrees higher. They sometimes cease from lack of fresh subjects to affect, and break out again when strangers arrive.

The disease invades seaport cities and towns rather than rural districts, but it follows up the course of navigable rivers.

It is believed by the majority of the most competent observers that the germ is not directly contagious from one individual to another, but that it lodges in the air, or upon the surface of neighboring objects, where it matures. Those who apparently have contracted the disease from the person of yellow-fever patients have been shown to have been infected rather through fomites contained in the same house or ship, and not by direct transmission from one body to another.

Substances which readily act as fomites are hair, feathers, cotton, wool, linen, etc. By these agents the poison is carried to great distances in the clothing, luggage, or merchandise from an infected district. When protected it preserves its vitality for a long period—at least a year—and when by opening a cesspool or cleaning out a drain or renovating an old house it is subjected

to favorable conditions, its activity is renewed and it gives rise to a fresh epidemic of the disease. These conditions are moisture and a minimum temperature of 72° F. Emanations from decaying animal and vegetable matter or fæces and poor sanitation also favor the spread of the disease. It might conceivably be exterminated by perfect public and private hygiene. It is not conveyed by food or drinking-water, and it loses its toxicity in abundant fresh air, and consequently the disease is not spread far by the atmosphere alone, and heavy rains, by clearing the air and cleansing the soil, diminish the force of an epidemic.

Provided that the clothing be disinfected, the disease is not conveyed by either the dead or living body. Yellow fever may break out on ships after sailing from infected ports. If the patients on reaching a northern port are disinfected and all fomites are removed from them, they may be safely treated on shore without fear of exciting contagion or epidemic.

Although transmitted for short distances through the atmosphere, the virus tends to become greatly concentrated and violent in effect in certain crowded localities, where it may remain confined, leaving other districts in the same city in perfect immunity. It prefers low-lying regions along the coast, especially the mouths of rivers, and, like malarial poison, it is more virulent by night than by day. Typhoid and malarial fevers are apt to be prevalent during yellow-fever epidemics, but they do not occur together in the same individual.

Age.—Very young infants usually escape the disease or have it in such a light form that it is overlooked. It has been reported, however, in a child only ten weeks old. Infants are less exposed than adults to the conditions which favor the spread of the fever, and they may escape on that score. The very old, for a similar reason, may escape entirely, but the disease has been observed at all periods of life up to eighty years.

Sex exerts no decided influence on the spread of yellow fever, but it is especially fatal among white males between the ages of twenty and forty years. Males are more apt to visit infected localities, and a larger number of males than females may contract the disease on that account.

Race.—White robust adults in the prime of life are especially subject to the infection, and creoles are not exempt. Among negroes there is far less susceptibility to the disease and less fatality than among whites. Billings suggests that negroes may have had a mild form of the disease in infancy, and may have secured an apparent immunity in that manner.

Immunity.—The statement is often made that one attack renders the individual exempt for life from a second. While this is true in the vast majority of cases, there are many well-authenticated instances of a second attack occurring in the same person after an interval of years. A person who has had one attack is said to be "acclimated" or "protected." These expressions do not refer to those who have escaped the disease entirely.

In cities where yellow fever has existed for a long time in endemic form the residents acquire immunity to a remarkable extent, so that the disease is

really kept in force by infants and strangers from northern climates, who are alike unacclimated. Thus, of 55 unacclimated physicians who went to Memphis to labor in the epidemic of 1878, 54 contracted yellow fever (Bemiss). The immunity or acclimatization is lost to a great extent by subsequent prolonged residence in a northern climate.

Fear, worry, and panics favor the acquisition of the disease by reducing vitality and resisting power. The same is true of fatigue, constipation, exposure to a hot sun, and debauchery.

Morbid Anatomy.—In mild cases, and in severe cases which die very early, no characteristic lesions are found. In cases of ordinary severity post-mortem examination reveals hyperæmia, extravasation, and degeneration. Cadaveric rigidity occurs promptly and is well marked. The important changes are in the liver and gastro-intestinal mucous membranes.

The *liver* contains less blood than normal, unless death has occurred very early, when it is congested. It is usually unaltered in size and is friable. The color varies from pale yellow (*café au lait*) to bright yellow or almost an orange hue. This color is uniform or shaded in patches, and the surface may be mottled with punctate hæmorrhages. The liver presents the changes of parenchymatous hepatitis. The hepatic cells in certain parts are filled more or less completely with granular matter and fat, and many of them are swollen and fused, and their nuclei are indistinct or absent. In other portions the cells may be normal. The small bile-ducts are filled with swollen degenerated epithelium. The gall-bladder contains a very little dark bile.

The skin.—The icterus, which deepens after death, and is regarded as owing to altered pigment, which colors the skin in a certain proportion of cases, appears at about the commencement of the second stage of the disease. It is derived from the red blood-disks, forming hæmatogenous jaundice. Later, at the end of the second period, or in convalescence in a smaller number of cases, the color of the skin becomes of a saffron, mahogany, or even orange hue, and the urine is loaded with bile-pigment. It is believed by Féraud and others that this form of jaundice is due to bile-pigment contained in excess in the circulation. It affects the entire body, but is deepest on the arms and chest. The urine, serous fluids, and tissues generally are stained yellow. The blood is dark, coagulates poorly, and many red corpuscles are disintegrated. It quickly decomposes.

Scattered throughout the entire body, especially on serous and mucous surfaces, are small hæmorrhagic spots. They are found on the pleura, pericardium, meninges, between the muscles, in the mucous lining of the gall- and urinary bladders, and in the stomach and intestines. Petechiæ, vesicles, or large irregular ecchymotic spots are also found on the skin, especially in dependent portions of the body. Larger hæmorrhagic infarctions occur sometimes in the lungs and other viscera. The heart-muscle is pale, soft, and the seat of granular and fatty degeneration. The spleen is not enlarged, but is dark and friable.

The *kidneys* present a more or less advanced parenchymatous nephritis.

There is cloudy swelling of the epithelia of the tubules with granular fatty degeneration. There are granular casts in the tubules. The whole alimentary canal is the seat of acute catarrh, but the gastric mucous wall especially is soft, turgid, and ecchymotic, and it may present erosions and contain "black-vomit" material. Similar material is found in the small intestines.

The *brain* is hyperæmic, especially the pons and medulla, and the meninges are congested. Schmidt describes certain degenerative changes in the sympathetic ganglia, with disappearance of the nuclei of the nerve-cells.

Symptomatology.—The incubation period of yellow fever lasts from twenty-four hours to six days: it may even extend over ten days. It is short in severe epidemics.

STAGES.—The disease has three stages: I. The "paroxysm," consisting of a cold period, followed by a febrile reaction; II. A remission or "stage of calm;" III. A uræmic stage, or a second exacerbation or collapse.

First Stage.—The invasion is in all cases sudden, and it may occur at any hour. It is characterized by a chill, rigors, frontal headache, vomiting, lumbar pains, pains in the calves of the legs, and great muscular prostration. There is capillary congestion, and the patient soon acquires a typical expression, with shining, staring, watery eyes and congestive cheeks and conjunctivæ. There is photophobia. There may be excessive sweating. The mind, as a rule, is clear, delirium being exceptional.

In children the disease often begins with convulsions. The cold period is followed by one of pyrexia, with a rapid rise of temperature, 104° or 105° F. being the maximum reached in twelve or eighteen hours. The temperature, as in malarial fever, begins to rise during the chill. The fever, which is seldom very high, gradually subsides after the maximum is reached, and after three to five days, with very slight remissions, the temperature reaches the normal degree. There is continued vomiting, first of mucus, then of bile, sometimes of blood. The stomach is intensely irritable, and the vomiting is of the projectile type. Pressure over the epigastrium excites emesis. The bowels are costive. The gums are sore and swollen, the mouth is dry, the tongue is red at the tip, and is often narrow. The skin may be dry and hot throughout the febrile stage, or it may be bathed in a profuse perspiration, which emits a peculiar sickly, disagreeable odor.

There is scanty acid urine, which shows a trace of albumin on the third day. There are great restlessness and possibly delirium. On the third or fourth day the conjunctivæ, and later the entire body, begin to show an icteroid hue. The pulse is slow in proportion to the fever, seldom rising above 110, and frequently keeping within 100. It is often described as "gaseous," or highly compressible and feeble. It may grow slower before the fever declines. Sometimes the cold stage is inappreciable, and the disease seems to commence with the fever. The symptoms thus far described constitute the "paroxysm." On the fourth or fifth day the fever and other symptoms abate and the "stage of calm" is reached.

Second Stage.—The temperature, having attained the normal degree by

lysis, may become subnormal and the patient feels greatly relieved. In mild cases convalescence begins from this time. The "calm stage" rarely exceeds two days in duration, and frequently lasts but a few hours, when the patient becomes much worse again, grows deeply jaundiced, and passes into the third stage. In severe cases there is sometimes a reactionary fever of remittent type and irregular duration (Sternberg). The urine is diminished in amount and there is albuminuria.

Third Stage.—The striking features of this stage are tendencies to hæmorrhages from all the mucous surfaces and to complete suppression of urine. The temperature either rises again for a day to 103° or 104° F., or it remains normal while symptoms of uræmia develop. The pulse-rate may be abnormally slow, falling sometimes to 40 per minute. Bleeding occurs uniformly from the stomach as "black vomit," and in addition it may take place from any other mucous surface or into the skin. The "black vomit" is present in about one-third of the fatal cases, and is due to passive hæmorrhage, but is not by itself pathognomonic of yellow fever, as it may occur in other affections. The stomach at first ejects whatever food it may contain, then mucus tinged with bile, and finally brown or black semifluid acid material resembling coffee-grounds, and consisting of red blood-corpuscles, pigment-granules, degenerated mucous and epithelial cells and leucocytes, fatty matter, and serous fluid. The acid gastric juice, acting on the blood-pigment, makes it dark brown or black. The quantity of this fluid ejected varies from a few drachms to several pints. It is acrid and irritates the fauces and mouth. The blood, altered in composition, oozes from the capillary walls of the congested mucous membrane of the stomach. The fluid is not always vomited, but in fatal cases it is very exceptional not to find it accumulated in the stomach. The intestines may be the seat of similar hæmorrhages, giving black diarrhœal stools. The swollen gums bleed readily, and there may be epistaxis. Rarely there is hæmorrhage from the respiratory tract, the nose, the ear, and the urethra. Females who are capable of menstruation bleed profusely from the vagina and uterus. During pregnancy yellow fever causes miscarriage. The jaundice deepens and the skin becomes of a dark olive or mahogany hue. If there be perspiration, it stains the linen yellow and emits a cadaveric odor. The urine becomes more and more scanty. It is acid, of high specific gravity, and it may be stained by the altered blood-pigment. The chlorides are diminished. It contains granular and hyaline casts, and its suppression adds uræmia to the existing toxæmia of the yellow fever. Sometimes cutaneous eruptions appear, such as roseola, pustules, or herpes labialis.

Exceptional cases are those (*a*) in which the cold period is omitted, and fever inaugurates the first stage; (*b*) cases in which no fever follows the cold period, or the febrile reaction is delayed, while the patient becomes stupid or semi-comatose, and the skin is congested and livid, the pulse being extremely feeble, albuminuria occurring on the first day, and the patients dying within three days; (*c*) cases commencing with delirium or maniacal excitement.

Should recovery take place, the jaundice continues for several days, grad-

ually fading out. If the case be fatal, the jaundice persists after death. Other expressions are used in describing various types of yellow fever in which certain symptoms predominate, such as ataxic, algid, adynamic, congestive, etc.

Duration and Terminations.—In a violent epidemic a patient may die suddenly from collapse within the first few hours of the disease, being stricken down while walking or at work. Death is most apt to take place from the third to the fifth day. Relapses are infrequent, but they may occur. If the patient do not improve or die before the fourth or fifth day, he passes into a typical typhoid state, with sordes, hard, dry, black tongue, muttering delirium, diarrhœa, petechiæ on the skin with large ecchymotic patches, extreme albuminuria, increased epigastric tenderness, black vomit, and finally suppression of urine, followed by convulsions or coma and death.

In cases which recover the average duration of the disease is six days. Death occurs from the poison of the disease itself, exhaustion, uræmia, or black vomit. It is not very common for patients to die from the hæmorrhage alone, though it should always be borne in mind that death may result suddenly from this or other cause at almost any period of the disease. It may occur in syncope after violent maniacal excitement.

Complications.—There are no special complications of yellow fever. It may exist in the course of various chronic diseases. The black vomit and jaundice in many cases do not appear at all, but when present they are symptoms rather than complications.

Sequelæ.—The sequelæ of yellow fever are few. Convalescence, always slow except in the mildest cases, may be further retarded by general furunculosis, suppurative parotitis, hepatitis, or by a very weak and irritable stomach and diarrhœa. Errors in diet have been known to produce fatal gastric hæmorrhage two or three weeks after establishment of convalescence. The heart is apt to be feeble, and reparative and nutritive processes proceed slowly. Phlebitis and thrombosis of the femoral vein sometimes follow. The stomach often remains irritable for a long time. Relapses occasionally occur during early convalescence.

Diagnosis.—The diagnosis of yellow fever is based upon the following features: its portability by fomites, the sudden invasion by chill and rapid rise of temperature, with a slow pulse, pains in the forehead, lumbar region, and calves, tenderness over the epigastrium, redness of the eyes, excessive gastric irritability, black vomit, jaundice, and diminished urine with albuminuria. Typical cases presenting all these features are unmistakable. In mild cases a correct diagnosis may be difficult. In any doubtful case occurring in a malarial district at the commencement of an epidemic of yellow fever it is well to give a large dose of quinine with a purge, and favor free diaphoresis by hot diluent drinks and a warm bath. If the temperature yield to these measures and the patient improve, he has not taken yellow fever. The jaundice itself is a very misleading diagnostic feature, for it is often absent in yellow fever, and may be present in malignant types of malarial fever. It may occur only

as a post-mortem pigmentation in yellow fever, and even in the severe cases it is not common before the fourth or even fifth day. Mild cases and the earlier cases of an epidemic may be confounded with relapsing fever, severe malarial fevers, such as bilious remittent or pernicious malarial fever, acute yellow atrophy of the liver, or jaundice of local hepatic origin attended by fever.

Relapsing fever has a typical spirillum found in the blood, is non-contagious, the spleen is enlarged, there is no black vomit, there is a typical relapse, and both temperature and pulse are higher than in yellow fever.

Malarial fevers are non-portable, are controlled by quinine, the spleen is enlarged, the fever is distinctly periodic, either remittent or intermittent, albuminuria is much less frequent, and one attack of the fever favors subsequent ones.

In bilious remittent fever there are usually several paroxysms instead of one; the remission commonly occurs abruptly, within twenty-four hours instead of upon the fourth day; the tongue is coated heavily, and is broad, flabby, and indented by the teeth, instead of sharp, dry, and pointed; the spleen is large; the splenic area is tender; delirium is more common; and copious vomiting of bile occurs, in distinction from the mucus and black vomit of yellow fever. Albuminuria is rare, and the ague is more apt to occur in inland rural districts than in seaport towns and cities. Free pigment and the malarial plasmodium may be found in the blood. Death, if it takes place, comes earlier than in yellow fever.

Hæmorrhagic remittent malarial fever, accompanied by jaundice, sometimes resembles yellow fever quite closely, but previous attacks of ordinary ague will have occurred, the jaundice appears very early, and the symptoms fluctuate with the temperature, and melanuria appears with each paroxysm.

Phosphorus-poisoning has some features in common with yellow fever, but the odor of the drug is obtained in the breath and traces of poison may be found in the ejecta.

In acute yellow atrophy, which is non-portable and not epidemic, the spleen is enlarged, the liver is reduced in size, black vomit is absent, and the disease begins slowly without fever or pain.

In local jaundice with pyrexia difficulty in diagnosis may arise, but careful attention to the special diagnostic symptoms of yellow fever above enumerated will seldom fail to decide the case.

Prognosis.—The prognosis depends upon the severity of the epidemic. As in many other epidemics, the maximum mortality in yellow fever is usually attained in a middle period, the earlier and later cases being less severe. The hospital mortality is always worse than that of private cases. It is seldom less than 20 per cent. among unacclimated adults, and it may exceed 50 per cent. This is in part due to the fact that the cases come under treatment late, many being brought in moribund. In mild epidemics 1 patient in every 15 or 20 dies; in severe epidemics 1 in 3 dies. In the epidemic of 1878, 36,000 cases occurred in Louisiana with a mortality of 16.66 per cent. (Bemiss), and in New Orleans the mortality at the Charity Hospital was 50

per cent. Among the fatal cases nearly three-fourths of the deaths occur during the first week.

The prognosis is particularly bad—(1) if the initial paroxysm is unduly intense; (2) if severe gastric irritability is persistent; (3) if black vomit occurs, but especially if there are passive hæmorrhages from various mucous surfaces; (4) if albuminuria increases and the volume of urine diminishes; (5) if jaundice appears early and is intense; (6) if patients have been greatly worried or fatigued, or if they are suffering from inanition or cachexiæ; (7) in pregnancy and the puerperal state; (8) if capillary congestion of the skin is excessive in the first stage; (9) if there are delirium and irregular pulse and respiration.

Complete suppression of urine is more fatal than black vomit. The combination of black vomit and complete suppression of urine is certainly fatal. If necessity for removing the patient arise after the attack has begun, the prognosis is rendered worse thereby. Prognosis is bad in certain rare cases in which the earliest symptom is delirium or stupor.

Prognosis is favorable when the gastric irritability and the amount of albumin in the urine diminish. Ordinary bilious vomiting is not an unfavorable sign.

The disease is more fatal among men than among women and children, and more fatal among alcoholic and plethoric subjects.

When the temperature remains below 103.5° during the paroxysm the course of the disease will probably be mild. If a certain locality has enjoyed long immunity from yellow fever, the epidemic is apt to be severe, because there will be more unprotected persons exposed to the disease.

Treatment.—While there is no specific for yellow fever, which is a self-limited disease, many lives may be saved by prompt and vigorous measures. There is no disease of which this can be said with greater emphasis, and as much depends upon the faithfulness and efficiency of a thoroughly trained nurse as upon the physician. Maintaining the patient's courage and hope is of great service.

The indications for treatment are—

I. To adopt prophylactic measures by rigid quarantine, etc.;

II. To keep the patient absolutely quiet;

III. To control the emesis and prevent the suppression of urine;

IV. To support the strength until the crisis is past.

I. *Prophylaxis.*—When an epidemic breaks out all persons whose duty does not keep them with the sick do well to leave the infected district immediately. It is well to avoid the presence of fever-stricken patients when suffering from fatigue, loss of food or sleep, or depressing emotions,—all of which factors render one more liable to the disease. Nurses and attendants should secure all the fresh air possible, and hospital patients are often best treated out of doors in tents. The patient should be quarantined, and all care must be taken to prevent the distribution of clothing, bedding, or any personal effects which have not been most thoroughly disinfected by strong heat, fumigation, or

other measures. The disease has been transmitted by a lock of hair from a diseased patient, and frequently through the medium of the mails.

Quarantine based merely upon a time limit is ineffectual, and it should depend rather upon the perfection of sanitary arrangements. The cardinal principles involved in prophylaxis during any epidemic are summed up in the oft-quoted words, "Isolation, disinfection, and depopulation."

As in the case of cholera, strict sanitary cordons around an infected area as a rule prove useless and practically impossible, and prompt depopulation, with thorough destruction of all fomites, is both more effectual and more humane. All excreta should be disinfected and afterward buried, although their infective power is doubted by some observers; and Freire advises the cremation of the bodies of those dying of the disease.

If yellow fever extends to a ship in an infected port, the vessel should put to sea and reach a healthy port as speedily as possible, when the crew having been isolated, it must be thoroughly fumigated, scraped, cleaned, and painted.

II. *Early Treatment.*—As soon as the diagnosis of yellow fever is made the patient must be put to bed and kept from visitors or excitement of any kind. Cheerful surroundings and abundant fresh air are most important. Medication and fluids must be given through a tube or with a teaspoon to avoid raising the head. The bed should be kept clean and the linen changed without moving the patient unnecessarily or exposing him to draughts. A bed-pan should be used, and, if the urine cannot be voided while the patient is recumbent, it is better to use a catheter than to let him run the risk of rising. If the patient be seen very early and indigestible food has been taken, the stomach should be unloaded by an emetic of ipecac. If there be constipation, a laxative or a purgative enema should be given. Excessive purgation is to be avoided, although it is a very common domestic practice to give castor oil on the first day. It is well to promote diaphoresis by a hot mustard foot-bath; the patient is then kept covered by blankets, and is given a hot lemonade or hot alcoholic drink. The neuralgic pains in the calves and back are relieved by twenty grains of quinine *per rectum* (Bemiss). Salol in five-grain doses every two hours is sometimes useful for its favorable action in the intestine.

III. *Treatment of Emesis and Suppression of Urine.*—When emesis becomes severe attempts should be made to control it by a mustard paste over the epigastrium and the internal administration of any of the following: dilute hydrocyanic acid; cerium oxalate; cracked ice; light acidulated and effervescing draughts, or plain lime-water in frequent half-ounce doses; cocaine in doses of one-sixth of a grain. If these measure fail, it is best to let the stomach have absolute rest, employ the rectum for subsequent medication, and give a small hypodermic injection of morphine. No hæmostatics have any specific control over the black vomit or the purpura, and no unnecessary medicine should be given.

With evidence of suppression of urine the lumbar region should be cupped and poulticed or covered with a large mustard paste or a turpentine stupe. The effervescing draughts should be increased. Apollinaris, Seltzer, Vichy, iced champagne, lemonade with bitartrate of potassium, are all useful. If the

stomach will not tolerate these beverages, a pint of salt water should be injected into the rectum every two hours. Digitalis, camphor, or ammonia in whiskey may be given *per rectum*. Diaphoresis should be encouraged in every way possible without too much disturbance of the patient. The temperature seldom demands special attention. If very high and prolonged, it should be controlled by frequently sponging the body with equal parts of alcohol and water.

IV. *Supporting Treatment.*—In ordinary cases during the febrile paroxysm it is best to give no food at all, for the stomach will only be disturbed by it. In cases accompanied by great prostration the immediate administration of stimulants by the rectum and hypodermically may be necessary. If extreme restlessness, insomnia, or delirium be wearing out the patient's strength, morphine should be given hypodermically or opium, sulphonal, chloral, or bromides *per rectum*.

After the paroxysm is over the greatest care must still be exercised in regard to diet. No solid food should be given for ten days or a fortnight in bad cases. At first teaspoonful doses may be given of peptonized milk, koumyss, or iced champagne, followed by beef-juice, and later by nourishing broths and gruels. Stimulating or nutrient enemata may be given once in two hours. They should be injected high up by the use of a flexible rubber tube. A few drops of laudanum will often secure their retention if the rectum prove irritable. In this way peptonized milk, beef peptonoids, brandy, egg-albumin, etc. are given, and enough is absorbed to maintain life until the stomach retains food. Children even earlier than adults usually require rectal stimulation. Large doses of opium should be avoided on account of the tendency to suppression of urine.

Exceptional and very fatal cases are those in which no reaction follows the initial cold stage. For these it is recommended to give hot mustard sponge-baths and vigorous stimulation. Other cases, occurring especially among the young, show violent neurotic symptoms at the first, such as convulsions, delirium, with alternate flushing and pallor of the face. For this type Bemiss advises the application of leeches to the temples or back of the neck, cold applications to the head, calomel, chloral by enema, morphine by the skin, and the inhalation of chloroform if the convulsions are severe.

Sternberg, who has been employed by the United States government to study recent epidemics of yellow fever, advocates the use of sodium bicarbonate to make the highly acid urine neutral or alkaline, and thereby diminish the imminent danger of nephritis with suppression. He has also had some success with the internal use of corrosive sublimate.

The fact that epidemics of yellow fever are arrested by frost has led to the experiment of treating patients by surrounding them with an atmosphere of artificially cooled air (Garcia); but, as might be expected from the fact that the reduction of the internal body-temperature to any marked degree is incompatible with maintenance of life, this treatment has yielded discouraging results, besides being very expensive and uncomfortable for the patient.

TETANUS.

By JAMES T. WHITTAKER.

Tetanus (τετᾰνός, τεινῶ, to stretch); Trismus; Lockjaw; Opisthotonos (ὀπίσθε, backward, τεινῶ, to stretch); Starrkrampf, Wundstarrkrampf (Ger.), —a grave, often exquisitely acute, infection, caused by a specific bacillus, the tetanus bacillus, introduced through a wound or some break of the surface, characterized by excessively heightened reflex under the action of toxines, which induce spasmodic contraction of the voluntary muscles, first and especially of the jaw (trismus, lockjaw), face, and neck, and extensors of the spine (opisthotonos), of short duration, often of rapidly fatal termination. Among the larger animals the horse, sheep, and goat are especially liable to the disease.

The clinical features of tetanus are so coarse and obtrusive as to have been remarked in the most ancient times. Some of the finest descriptions of Aretæus were based upon observations of tetanus. Hippocrates devoted a whole section to its treatment, and certainly appreciated the gravity of the disease. "Such persons," he says, "as are seized with tetanus die within four days, or, if they pass these, they recover." Aretæus declared tetanus to be a spasm of an excessively painful nature, very swift to prove fatal, and not easy to be removed. "It supervenes," he declares, "on the wound of a membrane or of a muscle or about punctured nerves, when, for the most part, patients die; for spasm from a wound is fatal." . . . . Women are more disposed than men; children are frequently affected, but less fatally. "In all the varieties," he says, "there are pain and tension of the tendons and spine and of the muscles connected with the jaws and cheek, so that the jaws could not easily be separated even with levers or a wedge." No such graphic description of the symptomatology of the disease as detailed by Aretæus has ever since been written. The distortion and suffering are so great as to make the spectacle painful even to the beholder. "The physician," he declares, "has no power over the disease; he can merely sympathize. This is the great misfortune of the physician."

Most of the contributions of later times have been presented by the surgeons Laurent, Larrey, etc. Curling wrote his famous treatise on tetanus (Jacksonian Prize Essay) in 1834; Rose (E.) made the most valuable clinical contribution of modern times to the *Handbuch der Allgem. u. Specielle Chirurgie*, Pitha u. Billroth, Bd. 1, Abtheil. A., 1870. Nikolaier discovered the bacillus of tetanus in 1885.

Brieger (1887) obtained from sterilized cultures of the tetanus bacillus a toxine which, in mice, in the smallest doses, produced the typical symptoms

of trismus and tetanus with fatal termination. Besides this body, Brieger eliminated various tox-albumins with specific properties.

Etiology.—Tetanus is now known to be a specific disease. It arises in no case spontaneously, and demands for its development a break of the surface through which its specific cause may be introduced; hence tetanus follows most frequently in the course of and in consequence of some external injury. Though the extent and severity of the injury stand in no direct relation to the disease, the seat and character of the wound have much to do with its development. For, while tetanus may occur in consequence of any kind of wound, it does occur much more frequently after contused wounds with penetration of foreign bodies. It is therefore very frequent after gunshot wounds, and is especially frequent in wounds of the extremities. Wounds of nerves are also attended with special liability.

Tetanus may follow a lesion as trivial as the extraction of a tooth, a venesection, the sting of an insect, a simple scratch of the surface, the application of a blister, a slight wound of the foot as from a nail in a shoe. It occurs not infrequently in the newborn from lesions of the umbilical cord, and has been repeatedly observed after a wound of the cervix uteri, as after parturition. The intrusion of a splinter of wood, the lodgment of a fish-bone in the throat, have broken the surface sufficiently to introduce or give entrance to the cause of the disease; but, as the cause comes from without, tetanus occurs in the great majority of cases in wounds of the extremities. Curling found wounds on the extremities in 111 of 128 cases, and Thamhaym in 395 cases found the locality of the injury in the hand and finger 119 times.

Though the frequency of tetanus varies at different times, it is on the whole a comparatively rare disease. True, Lind saw 5 of 6 cases of amputation die of tetanus, and once in modern times—namely, at the battle of Lyon in 1834—12 of 277 wounded died of the disease. The experience of Blanc, who saw 30 cases in 810 wounded, is also unusual. More in accord with the rule is the rarity of the disease in the Civil War in America and the Franco-Prussian War, in one corps of which there were observed but 45 cases among 24,262 sick and 7182 wounded.

In civil life the disease is still more rare. Thus, at Guy's Hospital in thirty-two years there occurred but 1 case of tetanus to 1570 patients; in Vienna in ten years, but 1 case to 4798 patients. Rose states that the mortality of tetanus in Berlin was but .04 per cent., and this included 266 cases in newborn infants.

The disease is most frequent in hot countries. Aside from attack of the newborn, the period of greatest liability is between ten and thirty.

The fact that the disease occurs after minute, almost undiscoverable, injuries as readily as after the most extensive lesions, long ago excited suspicion of its infectious nature. Carle and Rattone (1884) furnished the first proof of communicability of the disease by the inoculation of rabbits with pus from the wound in a case of human tetanus. Nicolaier in the following year discovered widely disseminated in all kinds of earthy matter bacilli which, introduced

subcutaneously into mice, guinea-pigs, and rabbits, produced typical trismus and tetanus with fatal termination. Rosenbach in the next year (1886) demonstrated the tetanus bacillus for the first time in man, and a number of competent observers confirmed these demonstrations in other cases, including tetanus neonatorum (Beumer, Piper), including also castration tetanus and tetanus traumaticus in animals (Bonome). Thus was established the genetic relation to the disease of the bacillus of Nicolaier.

The tetanus bacillus is a delicate rod, a little longer than the bacillus of mouse septicæmia. It occurs in irregular masses in the affected tissue, and is recognized by the characteristic development of its spores. One end of the bacillus swells to show an oval, sharply-defined, shining spore, and presents the appearance of clock bell-strikers, drumsticks, or, better, pins. This spore formation occurs in great abundance in the body of the animal as well as in artificial culture. The bacilli are easily colored with methyl-blue and fuchsine. Artificial culture is difficult. The bacillus is a strict—*i. e.* an obligate—anaërobe, so that in artificial culture particles of infected matter must be introduced into the deeper layers of blood-serum to secure growth. The culture is so commonly contaminated as to often require subsequent separation to obtain it pure.

The bacilli and spores of tetanus are so widely disseminated in soil and dust as to be almost ubiquitous. They abound most on the surface of inhabited soil, and are not entirely absent in uncontaminated virgin soil. The rubbish and dust of streets and houses are soils of predilection. The wide dissemination of the parasites accounts for the cases of apparent spontaneous or idiopathic tetanus, while the fact that the free access of oxygen prevents its growth furnishes explanation of the comparative rarity of the disease and greater liability in case of penetrating wounds.

The tetanus spores found in the earth develop virulent cultures upon serum in the course of sixteen days. Of 23 soil-tests taken in Copenhagen, 16 proved virulent in the inoculation of animals; 7 tests taken at a depth of two to four feet all produced tetanus; 4 of 5 soil-tests, taken from gardens outside of the city, showed no spores and produced no infection. The bacillus is innocuous in the stomach.

In an examination of 25 specimens of earth Verhoggen and Baert found the genuine tetanus bacillus 15 times, demonstrated in all cases by inoculation. The bacillus may not, however, be demonstrated in the blood. The injection of the substance of the spinal cord of animals dead of tetanus produced tetanus in other animals only when introduced under the dura, and never when introduced under the skin. The same results were observed with the use of strychnine, which has much the same effect as the poison of tetanus. The disease is sometimes conveyed by contact with horses affected with tetanus, though the bacillus is found much more frequently in the soil than in the body of animals. Tureina demonstrated in the dust of the floor of three wards of a military hospital, as well as in the dormitories, the presence of the tetanus bacillus. The demonstration was made by means of the inoculation of rabbits.

Dor inoculated rabbits with the cerebro-spinal fluid of a man dead of tetanus. The animals quickly succumbed without showing any pronounced picture of tetanus. Pure cultures were made with the tetanus bacillus obtained from the spinal cord of these animals. Rabbits inoculated with these cultures showed the distinct picture of true tetanus. The bacilli perish very rapidly after the death of the patient, hence the difficulty of their detection. Pure cultures are best obtained by great dilution in sterilized water and stroke inoculations of the serum of horses' and sheep's blood.

Brieger demonstrated the presence of tetanine both by chemical analysis and physiological experiment. Particles from an infiltrated arm which showed, under the microscope, tetanus bacilli, other long bacilli, staphylococci, and streptococci, were introduced under the skin of mice, guinea-pigs, and rabbits, with the result that tetanus occurred in every case. A dog proved refractory both to this substance and to the injection of tetanine. A large abscess developed in a horse. Injections of large doses of tetanine produced long, persistent, violent muscular contractions, but the rigidity characteristic of tetanus in the horse did not develop. Beumer first succeeded in producing the characteristic picture of tetanus by the inoculation of particles taken from a wound at the umbilicus in a child dead of trismus neonatorum. The demonstration was thus offered that trismus and tetanus in the newborn may be no longer looked upon as a neurosis, but must be regarded as a traumatic infection. Kischensky examined three cases of tetanus neonatorum in consequence of an omphalitis. Inoculation of the pus produced tetanus in one case. In all three cases the streptococcus was found in the pus, and in one case it was also found in the internal organs. Nissen succeeded in demonstrating toxines of like effect in the circulating blood of a patient affected with tetanus. The blood, withdrawn by venesection twenty minutes before death, showed itself free of tetanus germs in agar cultures, but the injection of six mice with but .03 ccm. of blood-serum produced a fatal tetanus within a few hours, while other mice injected with the blood-serum of healthy or non-tetanic men remained unaffected.

Pestana concludes that the poison of tetanus is absorbed by the blood, and is thence taken up by and retained in the lungs, the spleen, the kidneys, and, above all, the liver. The toxine is conveyed by the urine in imperceptible quantities. It can be demonstrated in nerve- and muscle-substance. Faber secured a filtrate, by means of Chamberland's filter, entirely free of bacteria —a sterile, clear, yellowish, nearly alkaline fluid of equal virulence with the culture-soil itself. Inoculation of this substance is followed without local signs—*i. e.* spasms—by general tetanus which begins with trismus. The tetanus shows itself sooner than after infection. The filtrate loses its virulence entirely after heating for five minutes at 65° C. Introduced into the alimentary canal, it has no poisonous effect.

Kitasato observed that the filtrate perfectly free of germs produced the same tetanic effect as the culture of tetanus bacilli; hence tetanus is not a question of infection, but of intoxication by a specific product of the tetanus

bacillus. Of the animals experimented upon, the most sensitive were guinea-pigs, then mice, then rabbits. Tetanus sometimes shows itself at once, at the latest on the third day. The inoculation of organs of animals dead of tetanus into other animals remained without effect, but the inoculation of blood or transudations from the chest-cavity, though free of germs, always produced tetanus in mice. The tetanus poison therefore penetrates to the blood and produces here its toxic effect. A filtrate exposed to daylight at a window loses its virulence in the course of several weeks, but when kept in a dark room it is, after three hundred days, as virulent as when fresh. Direct sunlight absolutely destroys the poison of tetanus in fifteen to eighteen hours. Dilutions with water do not affect it.

Morbid Anatomy.—Tetanus shows no distinct and definite lesions. The cause of the disease often disappears to leave no trace, and, since this cause has been determined to be of chemical nature, questions of morbid anatomy have lost interest. Lockhart Clarke mentions the discovery of areas of fluid or of granular disintegration in the gray matter and in the white columns of the spinal cord. Coates found the same appearances in the bulb and the pons. Dickinson looked upon these changes as exudations. Recent necropsies show extensive hyperæmia, which in the course of time entirely fades away.

Bruscattini studied the condition of the different parts of the organism after inoculation. He made inoculations with emulsions of the central nervous system, kidneys, liver, blood. The animals having been killed when the symptoms were at their height, the blood and kidneys were found virulent, the liver and suprarenal capsules innocent. The poison is disseminated gradually along the course of the nerve-substance, and rather in ascending than descending direction, whether it be injected directly, subdurally, or subcutaneously, after the manner of the poison of hydrophobia.

Symptomatology.—The period of incubation varies from one to two weeks. Of the 75 cases recorded by Faber observed in the course of thirty-five years, the period of incubation could be accurately established in but 64. In 74 per cent. of these cases it ranged from seven to eleven days, never less than four or more than twenty-two days. In 11 of these 75 cases no contact with tetanus could be observed; 28 of the remaining 64 cases were infected by the soil, 11 by contagion in the hospital.

The disease begins, as a rule, with spasm of the muscles of mastication. Contraction of the masseters locks the jaws to produce the condition known as trismus, lockjaw. Contraction of the muscles of the neck occurs at the same time or may precede the contraction of the jaws. Rose delares that the contraction of the masseters may be felt by the insertion of the finger within the mouth, and that the stiffness of the muscles of the back of the neck is best recognized, as in cerebro-spinal meningitis, by attempts to lift the body by the head. The affection of the muscles of the face soon produces a peculiar physiognomy. The lips are usually stretched over the closed teeth to produce the characteristic smile, the *risus sardonicus*, so graphically

described by Hippocrates. Fagge speaks of the case of a girl who was reprimanded by her mother on account of a singular grinning expression of the face, over which she had, of course, no control. This alteration of the physiognomy gives to the patient the appearance of age. Farr says a man aged twenty-six was taken for sixty.

The disease begins usually mildly and increases gradually and progressively. There is, in association with the stiffness of the neck or diminished mobility of the jaw, some difficulty of deglutition. The muscles are affected from above downward. The spasm extends to involve the muscles of the back. Implication of the groups of great muscles in the spine soon distorts the body. The whole trunk is stiffened like a statue (orthotonos), or is more frequently arched with its convexity upward (opisthotonos). It is said to be sometimes arched forward (emprosthotonos), or laterally (pleurosthotonos). The forearms and hands are spared for a long time. Motion, either active or passive, is soon inhibited or lost altogether under the board-like induration of the muscles. During these states of rigidity convulsive attacks occur with shocks like strokes of lightning. They show themselves in consequence of effort, even of involuntary effort, or as the result of any outside irritation, and express the intense reflex excitability of the spinal cord. In the interval the body assumes the position of rigidity from which it has been distorted by the violence of the spasm. The suffering of the patient at this time is indescribable. The spasms are attended with excruciating pain. The mind is perfectly clear, but is weak from loss of sleep and anxiety. The patient may not satisfy either hunger or thirst on account of the locking of the jaws. The arching of the body from contraction of the muscles of the spine (opisthotonos) prevents a proper decubitus. Individual muscles, especially the recti abdominis, have actually ruptured under the powerful contraction, to discharge masses of blood at their divided ends. Difficulty of breathing, cyanosis, a sense of distress and danger, with lancinating pains at the bottom of the chest, indicate the spasmodic contraction of the diaphragm. Fever may be entirely absent. There is generally some elevation of temperature, which is liable to sudden exaggeration, often without discoverable cause, probably due to the influence of the nervous system. Extreme elevations of temperature to 110° or 112° F. are pre-agonal. Sometimes there is an elevation of temperature after death. The pulse is, as a rule, but little affected. It may be retarded during the paroxysm or it may be increased ten to twelve beats. Rapid increase to 170 to 180 usually precedes a fatal termination. Liston declares that the vessels may be so much contracted as to prevent the escape of a drop of blood in amputation of a member. The skin is usually covered with sweat, a point often of diagnostic value. The bowels are constipated. There is often suppression, and more frequently retention, of urine.

Diagnosis.—The diagnosis largely rests upon the early appearance of trismus. Lockjaw from sore throat, mumps, synovitis, rheumatism at the temporo-maxillary articulation, should be easily distinguished by the most superficial examination. The feel of the rigid masseters inside the mouth and

the associate stiffness at the back of the neck speedily dissipate doubts. Hysteria and hystero-epilepsy may show the typical opisthotonos of tetanus, but hysteria is, as a rule, unattended with trismus, and when trismus is simulated by the fixation of the jaws, hysteria is recognized by the fact that the intervals of attack are irregular and always entirely free from spasm or pain.

The regular invasion of tetanus from above downward, first of the muscles of the face and neck, later of the trunk, distinguishes the disease from the spasmodic contractions of spastic myelitis. Cerebro-spinal and basilar meningitis, which have in common with tetanus stiffness of the neck and opisthotonos, almost never show trismus. They have also a different origin and history, are epidemic or tuberculous, with associated symptoms, such as vomiting, headache, hyperæsthesia, herpes, etc., not seen in tetanus.

Tetany is distinguished by its typical spasms of days' and sometimes weeks' duration, and absolute intermissions; by the peculiar contraction or position of the hand, which may be called out by long pressure upon the nerves or arteries of the arm, the so-called Trousseau phenomenon; by the frequent laryngo-spasm; and by the increased mechanical and galvanic excitability of the motor nerves.

Hydrophobia, which has in common with tetanus spasm of the muscles of deglutition, is distinguished by the much shorter period of incubation, by the trismus and opisthotonos of tetanus, and by the psychical exaltation and anxiety of hydrophobia.

By far the most important question in differential diagnosis concerns the recognition of poisoning by strychnine. The poisonous effects of this alkaloid are most closely simulated by the effects of the toxines of tetanus. The diagnosis rests upon the following points: The history of origin where it may be ascertained, the existence of a wound, the period of incubation. Signs of strychnine-poisoning supervene at once. Tetanus begins with trismus, and gradually descends, sparing as a rule, except in children, the arms and hands. Strychnine often shows its first signs in irritation of the stomach, and in the affection of the muscles seizes by preference upon the extremities. In tetanus there is persistent rigidity; in strychnine-poisoning there are intervals of absolute relaxation. Thus, in the interval between the paroxysms the mouth remains closed in tetanus, but may be freely opened in strychnine-poisoning. The reflex spasms of tetanus occur later in the course of the disease, and increase in intensity, while those of strychnine occur at once, intense from the start. Strychnine-poisoning is quickly terminated by death or recovery; tetanus is protracted to days and weeks. Golding-Bird reported the case of a boy affected with tetanus with spasms for fifty-one days, with subsequent persistent rigidity and death on the one hundred and seventh day.

Eiselberg establishes as a difference between tetanus and other wound infections the fact that in tetanus local wound reactions are entirely absent. So-called cases of rheumatic tetanus are therefore really of traumatic origin.

The **prognosis** is exceedingly grave. Death may occur in any attack of convulsions. The heart has, actually under observation, suddenly ceased to

beat. Death occurs, as a rule, before the end of the first week, so that, as Hippocrates said, "patients die within four days, or, if they pass these, they recover." In exceptional cases, however, the fatal termination may not occur for three weeks. The disease rarely lasts longer in children than two or three days. The prognosis is so grave in the newly-born that Bauer declares that the occasional cases of recovery have been looked upon as probable errors in diagnosis.

The prognosis may be determined in some degree by the length of the period of incubation—that is, the interval between the injury and the appearance of the trismus; for an interval of less than ten days gives a prognosis of 96.6 per cent., while the general prognosis, inclusive of the cases of long and short interval, ranges from 84 to 87.5 per cent. The prognosis stands in direct relation to the frequency of the paroxysms and the rapidity of increase of rigidity. Death may take place in cases of rapid recurrence and short intervals in the short space of two to ten hours. According to Rose, 63 per cent. of cases die within the first five, and 88 per cent. within the first ten, days. The relief of the later periods is probably to be explained by at least partial elimination of the toxines. Rigidity may persist for some time, even for months, after recovery. The ability to sleep is always a favorable sign.

Prophylaxis.—In prevention of tetanus it is to be emphasized that the minutest wounds soiled with earth, dust, or foreign bodies, as splinters, are to be scrupulously cleaned and disinfected. The minutest fragments of splinters must be removed immediately. With regard to the fact that the secretions of the wounds of patients contain bacilli, and that the poison has such great resistance to desiccation, it is further strictly enjoined that all materials in contact with the wound, dressing, bandages, etc., are to be destroyed by fire—that separate intruments are to be used for such patients, and the patients themselves are to be isolated from other surgical cases.

In prophylaxis of the newborn it must be observed that the wound at the navel is attended with the utmost care. The aseptic treatment already recommended by various authors meets thus with scientific justification; for all the investigations concerning the origin of the tetanus bacillus demonstrate that it has an unusually wide ectogenous dissemination. Unclean hands, the use of bandages not sufficiently aseptic, and the raising of dust in the cleaning of the puerperal room, suffice in the observations of Beumer to convey the infecting agent.

Treatment.—As in hydrophobia or other disease characterized by excessive hyperæsthesia of nerve-centres, the patient should be kept perfectly quiet. He should be put in a dark room and isolated from curiosity and officious or meddlesome ministration. The most absolute silence should be enjoined on the part of the patient as well as the attendants. On account of the locked jaw the food should be fluid, but should be as nutritious as possible. Milk, soft-boiled egg diluted with hot water, nutrient soups, stimulants, as wine, whiskey, or brandy, should be regularly administered. Where the act of deglu-

tition excites spasm the patient may be anæsthetized, and, according to the suggestion of Rose, fed through a tube, which may be, as in the case of insane or refractory patients, inserted through the nose. Foreign bodies should certainly be immediately extracted and irritated nerve-trunks excised. Angry wounds, "festering sores," may be treated with the powerful antimycotics, as carbolic acid, corrosive sublimate, or with the actual cautery. More extensive exsection, and especially amputations, are surgical barbarities of the past. Spasmodic contractions are best relieved by the administration of anodynes. Opium, on account of its associate discomfort and distress, is better substituted in our day by chloral. A large dose, 1 drachm at first, may be followed by smaller doses, 15 to 30 grains every hour or two, or as often as necessary to subdue spasm. Calabar bean and curare have been administered with success in individual cases, sometimes of questionable diagnosis, but these remedies have failed, as a rule, to secure other than temporary relief.

Baccelli recommended the injection of 1 cg. of carbolic acid every hour or two until the spasms entirely ceased. Caliari reported the case of a child three years of age which cut itself in the left thumb with a kitchen knife. The father stopped the blood with cobweb. Tetanus set in in twenty-seven days, and was treated by the method of Baccelli: 1 gramme of a 1 per cent. solution of carbolic acid was injected subcutaneously three or four times a day. At the same time there were administered clysters of potassium bromide, 0.75, and chloral hydrate, 0.25, with warm baths. Perfect cure was secured in twenty-seven days. Proof that the cobweb carried the tetanus was established by experiments on a guinea-pig and a rabbit, in whose bodies was introduced cobweb taken from the same place. The animals died in three days. The rabbit showed exquisite tetanic symptoms.

Tizzoni and Cattani, as the result of a large number of disinfection experiments with pure cultures of the tetanus bacillus, found the most effective substance to be the nitrate of silver, which destroys the spores of the tetanus bacillus in a 1 per cent. solution in one minute; in the proportion of 1 : 1000, in five minutes. Sublimate solutions of the same strength require ten minutes. Creolin in 5 per cent. solution destroys tetanigenic spores in five hours; iodine in six hours; carbolic acid, 5 per cent., in eight hours; permanganate of potassium, 1 per cent., in ten hours. The injection of mytilo-toxine, the active principle of the poisonous mushroom (toadstool), as a therapeutic measure proved valueless.

The hope of successful treatment lies in the use of the antitoxines, derived from the blood-serum of immune animals (dogs) or from the bodies of the bacteria themselves.

Behring and Kitasato concluded, first, that the blood of rabbits rendered immune to tetanus possesses antitoxic properties; second, these properties exist also in extravascular blood, and are demonstrable in the serum of such blood free of cells; third, these properties are so permanent that they remain effective in the organism of other animals, so that it is possible by means of the transfusion of blood—*i. e.* serum—to secure therapeutic effects; fourth,

the antitoxic properties do not exist in the blood of animals which enjoy no immunity to tetanus, and the tetanus poison introduced into the bodies of such animals remains demonstrable in the blood and other fluids after the death of the animals. A normal rabbit succumbs to the injection of 0.05 ccm. tetanus poison. A protected animal may be inoculated with 10 ccm. without injury. Such an animal has immunity not only against the tetanus bacillus, but also against the tetanus poison, and may receive without damage twenty times as much poison as would be absolutely fatal to normal rabbits. In ancient times the transfusion of blood was regarded as an heroic, but in certain cases an extremely valuable, remedy. The results obtained by these experiments with the serum of blood furnish new proof that "the blood is a very peculiar juice."

Vaillard was able to confirm the conclusions of Behring and Kitasato concerning immunity from tetanus. The serum of rabbits rendered refractory to tetanus possesses protective properties; but immunity secured in this way is not permanent. It begins to diminish in the mouse in fourteen days, and disappears in the guinea-pig between the eleventh and fourteenth days. Neither aqueous humor nor the spleen of refractory animals extracted during life possesses the properties of serum. The fowl is insensitive to large doses of tetanic poison, yet the serum of the fowl has no antitoxic effect. The serum of a rabbit which had resisted every effort at inoculation had not the slightest antitoxic effect. This effect occurs only in animals to whom immunity has been given artificially. It is conferred by the injection of a large quantity of a filtrated culture. Thus it may be imparted to the serum of the fowl by the intraperitoneal injection of 15 to 20 ccm. of filtered culture.

Schwarz reports a case of cure of tetanus traumaticus with the antitoxine prepared by Tizzoni and Cattani. This case, after failure of other remedies, yielded to the injection of antitoxine 20 ctg. The patient had been previously put under chloroform. A part of the wound had been excised and the wound disinfected with a 3 per cent. solution of sublimate and a 4 per cent. solution of nitrate of silver. The antitoxine was injected during anæsthesia, and was repeated on the following day. The patient left the hospital perfectly cured. The author quotes from Gagliardi a similar unpublished case in which 1 gramme of the agent sufficed to remove all symptoms of tetanus and bring about a complete recovery. Paschini recorded a third, Casali now a seventh, case rescued in this way. The treatment consists in the injection of the tetanus antitoxine obtained from the blood of a dog rendered immune to the disease, 25 cg. being injected twice a day. Such improvement occurs in the course of a week as to render the further use of the remedy unnecessary, and the treatment is usually concluded with the hydrate of chloral. Unfortunately, all the best observers do not confirm these conclusions. Kitasato was not able to get immunity by tolerance nor by the use of filtrates attenuated by heat. Rabbits were rendered immune in 40 per cent. of cases with the trichloride of iodine, but the immunity was lost in the course of two months. Immunity is conferred upon mice by the injection of the serum of

immunized rabbits, but this immunity is lost in forty or fifty days. The fowl is by nature immune to tetanus, but the blood of the fowl does not confer immunity upon other animals.

By the second method, Ehrlich, Brieger, and Wassermann utilize the antitoxines developed by the bodies of bacteria themselves, after the manner of Koch with tuberculin. These antitoxines or protective bodies are to be obtained in the milk of parturient animals previously rendered immune in pregnancy by the inoculation of an attenuated culture which is gradually increased in virulence. The protective principle remains in the whey after coagulation and separation of the casein, so that it may be preserved indefinitely. Some of the most sensitive of the lower animals, mice, goats, etc., have already been protected in this way, but at the time of the present writing no account has been published of any work with man.

ACTINOMYCOSIS.

By JAMES T. WHITTAKER.

Actinomycosis (*ακτίς, ακτινος*, ray; *μύκης*, fungus), Big jaw, Swelled head, Bone tumor; Ger. Kinn-beule, Holzzunge, Knochenkrebs,—a peculiar infection of cattle communicable to man, caused by the ray fungus, *actinomyces*, characterized by development of the fungus in mass with excessive overgrowth of the soil in which it grows, attended by metastases to different organs, marked by symptoms of pyæmia and marasmus, and distinguished always by the detection of particles of the fungus itself in the mass, in its metastases, and in its discharges.

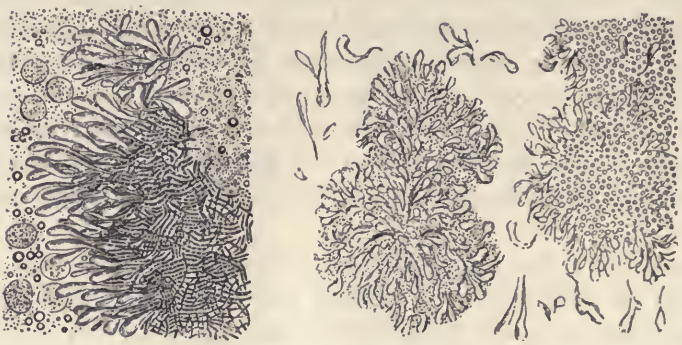
Bollinger (1877) first saw the fungus as the cause of the disease known as the big jaw in cattle. In this affection, which the veterinary surgeons had considered hitherto a purely local disease, especially of the jaw, and sometimes also of the tongue, throat, stomach, etc., and which they had called big jaw, wooden tongue, throat boil, bone cancer, etc., Bollinger discovered for the first time an extraordinary fungus as its cause. He took a specimen of it to Harz, a botanist of Munich, who gave it the very appropriate name it bears—actinomyces, ray fungus. Hereupon Bollinger designated the disease which it produces as actinomycosis.

Israel of Berlin also saw the parasite in man in the year of its discovery in cattle, and described it as a new mycosis of man. Ponfick (1879) established the identity of the disease caused by it in man with the actinomycosis of cattle. In the first observation in man the disease ran its course as a peculiar form of chronic pyæmia. Israel succeeded in distinguishing the parasite itself during life in the discharges from various abscesses from the skin, the largest of which, as post-mortem examination subsequently showed, communicated with the left lung. Fragments of the parasite, varying in size from a millet-seed to an apple, were discovered, also after death, in the liver, kidneys, spleen, and intestine. Israel subsequently encountered the same formations in other cases of local abscesses, evidently extending from carious teeth, as well as in the root-canals of the teeth themselves; whence he inferred that the mouth was the avenue of entrance, and that the parasite was carried by aspiration to the lungs, from which point it was disseminated by metastases to internal organs and to the skin. Israel now recognized that a case in the practice of Langenbeck, a fatal prevertebral phlegmon, had depended on the same cause. In subsequent observations the author demonstrated the origin of the disease not only from teeth, but also from tonsils, in whose crypts frag-

ments were found. These observations were speedily confirmed by others, so that the disease immediately took a recognized place in pathology. Belfield of Chicago first recognized the parasite in cattle in our own country as the cause of the disease known as swelled head, technically as jaw sarcoma.

Actinomyces constitutes a mass so large as to be visible to the naked eye. It consists of a conglomeration of innumerable threads of mycelia about a central mass of the same structure, from which the threads radiate in every direction to construct the ray shape. The mycelia can be always recognized by their clubbed extremities (see Fig. 29), and the mass, on an average about

FIG. 29.



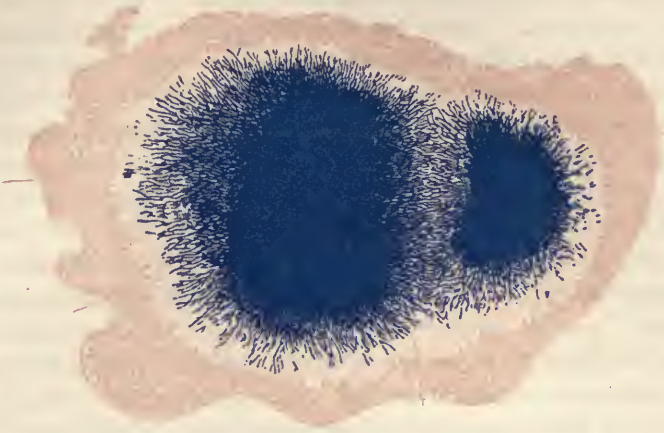
Actinomyces (V. Jaksch).

one-fortieth of an inch, is as large at times as one-tenth of an inch in diameter. Agglomerated masses may be as big as a fist. Fragments detached and discharged have a tallowy consistence and a distinctly greasy feel. Peripheral protrusions divide dichotomously, and show, as stated, distinctly clubbed or pear-shaped extremities, to resemble in certain fragments the appearance of a hand or glove with outstretched fingers. The peripheral radiation from a central mass gives, under the microscope, something of the appearance of an aster or sunflower. Many deviations, however, may occur from this classical type. The size of the individual mass may vary from barely visible granules up to masses of measurable diameter.

Besides the typical yellow color, particles may be seen colorless, transparent, greenish, or brown. The young granules are whitish-gray, the very youngest gelatinous, almost diffluent; the older colonies are opaque, and the oldest yellowish-brown and yellowish-green. The surface may be granulated, mulberry-form. Harz and Johne tried in vain to cultivate it. Israel finally succeeded with coagulated blood-serum, but with such different appearance from the normal structure as to make it impossible to decide upon the exact botanic relations of the microphyte. Boström succeeded best with granules floating free in pus or lying loose in granulation-tissue. Wolff finally inoculated the disease with pure cultures of actinomyces. The mass is colored with difficulty, though the mycelia at the periphery absorb the aniline dyes, especially gentian violet, and retain them. Fine pictures are made with dou-

ble colorations, as by the method of Gram, and subsequent stain with eosine. (See Fig. 30.)

Fig. 30.



Actinomyces: Double Stain by Gram's Method.

The **pathology** of the affection differs in man from that of the lower animals in that the process in the animal is a local swelling, a so-called granulation tumor, while in man the tendency is toward a suppurative process with metastatic dissemination, so that the disease in man runs its course with the formation of multiple abscesses and showing the characteristics of chronic pyæmia. The difference is explained by the belief that the process is not pure in man, but is attended with mixed infections, especially with the penetration of the pyogenic micro-organisms. Of the 9 cases reported by Baracz, in only 1 was there a pure actinomycosis: in all the others there was subsequent infection with the micro-organisms of pus.

The suppurative process in man is attended also with a distinct tendency to extensive fatty degeneration. Preparations of the granulation-tissue show great accumulations of fatty degenerated cells.

The most frequent avenue of entrance in man is, as stated, the cavity of the mouth, and especially the teeth whose surface is broken with caries; next the bones of the jaw; less frequently solutions of continuity in the pharynx and tonsils. More than half of all the cases hitherto observed in man have arisen in this way. The origin of the disease is ascribed to the ingestion of vegetable food, especially certain cereals. The fact that the disease occurs so frequently in cattle excites suspicion in this direction. Prickly fodder breaks the surface for the reception of the fungus. Johne found granules upon certain grains, especially of barley, whose free end was covered with a mycophytic growth which presented much similarity to a mass of actinomyces. Jensen, a veterinary surgeon of Denmark, observed an endemic of actinomycosis in cattle after feeding them with barley which had lain for some time upon moistened soil. Endemics after floods and inundations show the effect of moisture

in developing the fungus. Piana discovered fibres of various cereals in the actinomycotic growths removed from the tongue of a cow affected with the disease. These fibres were surrounded with characteristic vegetations of actinomyces. Brazola saw masses of fungus on fragments of barley in the gums of individuals affected. Baracz reported 9 cases of human actinomycosis—2 acute and 7 chronic. One individual was said to have chewed the ears of barley; one patient lived in the vicinity of stables for horses; a third drank daily of lukewarm milk in the stable. In others no possible contact could be discovered either with cattle or grain. In these cases the disease developed itself in the region of the lower jaw—in one at the point of the tongue, and in one on the neck in the region of the larynx. The avenue of entrance in man bespeaks the same origin—that is, from some vegetable source.

Symptomatology.—The disease demonstrates itself as a torpid and but slightly painful growth, which finally perforates the skin with sinuous tracts and various fistulous orifices. Some, if not most, of the great tumors or masses in the region of the lower jaw formerly diagnosticated as cases of angina Ludovici, which constituted in ancient times a much-dreaded malady, were certainly cases of actinomycosis of the lower jaw. Besides the penetration of the teeth, the parasite finds entrance into the body of man by way of the bronchi, and also by way of the intestinal canal. Thus, there is an actinomycosis of the jaw, of the lungs, and of the intestine. The disease distinguishes itself by its gradual encroachment upon tissues hard and soft in its vicinity. It expands bone, enlarges the natural outlines of the neck, converting the skin into a mass of cicatricial tissue, finds its way at times into the anterior mediastinum, and finally, after a lapse of months or years, causes the death of the individual by a slow process of suppuration or by a quicker suffocation or occlusion of larger vessels.

It may be distinguished upon the surface by the mass of cicatricial tissue; by the formation of abscesses with subsequent discharge without offensive odor, often through fistulæ of sinuous tracts; and absolutely by its yellowish granules of the size of grains of sand, visible to the naked eye, greasy to the feel, which, when placed under the microscope, reveal the distinctive characteristics of the growth.

Entering the bronchial tubes, it produces a peculiar form of bronchitis, most closely allied to putrid bronchitis, save that the offensive discharge which is expectorated separates into two instead of three layers—an upper supernatant, and a lower turbid fluid containing the actinomyces. In the lungs proper the disease gives rise to the symptoms of tuberculosis, and has been not infrequently mistaken for this disease. The gradual decline of health and strength, the progressive emaciation, cough, suppuration, night-sweats, make it closely resemble tuberculosis. In cases of more rapid progress the disease may simulate pneumonia with its glutinous muco-purulent or rusty sputa, dulness to percussion, and bronchial respiration. Metastatic processes from these centres disseminate the parasite to distant organs, most frequently to the subcutaneous and intermuscular connective tissue, and also to the various viscera—

liver, kidneys, intestine, heart, and brain. The irruption into the various serous cavities, pleura, pericardium, peritoneum, meninges, quickly causes fatal inflammation. Cases which escape these calamities survive to succumb to amyloid degeneration with anasarca and more protracted marasmus, the disease lasting, mayhap, two or three years.

In the intestine the mucous membrane shows whitish patches covered with yellowish granules, firmly adherent to the membrane upon which it rests. Various swellings appear, therefore, in its course, some of which suppurate and discharge their contents at times into the peritoneal sac, or, after agglutination to the parietal peritoneum, with subsequent discharge externally. Metastases, which are rare on account of the size of the growth, carry the disease to the liver, where the growths may attain considerable magnitude. So metastasis through the jugulars has developed masses in the lungs and heart.

Diagnosis.—The disease may be distinguished from ordinary affections of the jaw by its long duration, its tedious suppuration, its recurrence after incomplete exsection, its periods of quiescence, and its defiance of all ordinary treatment. In the lungs it affects the posterior and lateral portions, rarely the apices, and in the intestine it reveals nodular masses which may, at times, be felt beneath the surface. Neither the enlargement, suppuration, nor general symptoms, however, absolutely declare the disease, whose nature is only definitely established by the recognition of fragments of the parasite with the eye and its characteristic elements under the microscope. Certain apparently inscrutable cases of crypto-genetic infection have been unveiled as actinomycosis. One of the most remarkable of these cases was that mentioned by Bollinger of an apparently primary actinomycosis of the brain. Fischer remarks that the presence of vegetable fibres in any purulent discharges should excite suspicion of the etiology of the disease.

Prophylaxis includes the supervision of the food of animals; the avoidance of thorny or prickly twigs and plants, as well as of moist or wet food; the absolute destruction, as by fire, of all actinomyces in diseased organs of slaughtered animals; and enjoins above all things the most scrupulous care of the teeth and mouth.

Treatment.—The treatment is almost entirely surgical. It consists in the complete exsection and enucleation of the entire mass with the knife or its thorough eradication and destruction with caustic. The parasite seems to be singularly susceptible to the nitrate of silver. Köttnitz cured four cases with the solid stick, applied and inserted freely in every direction. Favorable results—*i. e.* death of the growth and rescue of the patient—have been secured in individual cases by injections of the ferric sulphate, tincture of iodine, carbolic acid, or corrosive sublimate, as also by cauterization with zinc chloride and the internal use of potassium iodide. Billroth succeeded in curing a case with tuberculin.

ANTHRAX.

By JAMES T. WHITTAKER.

Anthrax (*ἄνθραξ*, coal), Carbuncle, Malignant pustule, Splenic fever, Bloody murrain; German, Milzbrand; French, Charbon, Pustule maligne; Russian, Jaswa (boil-plague),—an exquisitely acute, often fatal infection, caused by the bacillus anthracis, and characterized by the formation of a boil with a black centre (anthrax), extensive circumjacent infiltration, and subsequent sepsis; in internal form by rapid toxicæmia and the development of metastatic carbuncles in the skin. Anthrax existed in the most remote antiquity. It is recognized that most of the fatal plagues which formerly affected animals, and not infrequently men, correspond to the symptomatology of anthrax. The plague of murrain, with boils and blains on man and beast, mentioned in Genesis, is believed to be of this nature (Blanc).

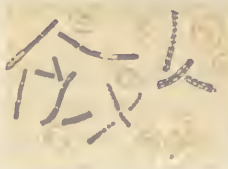
The disease is universal, but is manifest in intensity more especially under the primitive agriculture of the lower civilizations as connected with the nature of the soil and the food. In its internal or intestinal form it is exquisitely infectious and fatal. In San Domingo, in 1770, 15,000 persons perished in six weeks from eating the bodies of animals dead of the disease. Law declares that in the worst anthrax years in some of the Siberian steppes as much as one-fourth of the whole population was attacked with anthrax. Kircher ascribes the death of 60,000 people in the vicinity of Naples in 1617 to the same cause.

The bacillus anthracis is famous as the first micro-organism discovered as the actual cause of an infectious disease. It is the longest known and best studied of all the micro-organisms. The real acquisitions of modern bacteriology, with attenuations, involutions, toxines, antitoxines, have been made mostly with the anthrax bacillus. This bacillus was first recognized by Pollender (1855) and Brauell (1857), but was regarded as a lifeless crystal by the opponents of the germ theory because it showed no motion. Davaine demonstrated its infectiousness, and Koch the growth of the rods in long threads, the formation of endogenous spores, the liberation of these spores, and their development into new rods. When it was opposed to all these disclosures that the symptoms of the disease were produced by a chemical substance, anthracin, independent of any micro-organism, it was immediately demonstrated by Koch that the disease arises only from such substances as are evolved from the bacillus anthracis and its spores.

The milzbrand bacillus is a motionless rod of elongated, jointed cells .005–.0125 mm. in length—*i. e.* two to ten times as long as a red blood-corpuscle—.001 to .0015 mm. broad. (See Fig. 31.) Under proper con-

ditions it forms in the culture-soil, but never inside of the body or tissues of the living animal, endogenous spores, in which process it requires absolutely an abundant admission of free oxygen and a definite temperature ranging

FIG. 31.



Bacillus Anthracis.

between 18° C. and 34° C., best at 30° C. It is easily colored by any of the aniline dyes and readily yields its color. The spores are colored with great difficulty, so that double coloration is easy. Anthrax bacilli, like all endogenous bacilli, are not very tenacious of life, but the spores are extremely resistant and constitute the permanent forms. The bacilli perish under desiccation in several days; the spores resist it for many years. They can withstand a 5 per cent. solution of carbolic acid for thirty-seven days, while the bacilli are destroyed by a 1 per cent. solution in ten seconds. Decomposition or the action of the gastric juice quickly destroys the bacilli, but fails to attack the spores. The ingestion of meat free of spores produces no infection; the ingestion of meat with spores infects infallibly. This destruction of the bacilli is probably peptic—*i. e.* metabolic. It is certainly not due, as formerly believed, to the action of the hydrochloric acid of the gastric juice, for Dyrmont demonstrated that milzbrand bacilli maintain their virulence forty-eight hours in a 1 per cent. solution of hydrochloric acid; whereas the gastric juice of man contains at most but 0.2 per cent of hydrochloric acid. Freezing affects neither the bacilli nor the spores.

Anthrax infects chiefly herbivora, next omnivora, among which is man, and least of all carnivora. The disease is therefore not quite so dangerous in man as in some other animals. The bacillus anthracis is a saprophyte. It goes through with all its phases of development outside, and makes only accidental incursion into the body of man. Martin succeeded in extracting from cultures certain chemical products: first, proto- and deutero-albumose; second, an alkaloid; third, small quantities of leucin and tyrosin. Mice injected with the proto- and deutero-albumose were affected with œdema at the place of injection, and with a sufficient quantity (0.3 gr. for a mouse weighing 22 gr.) they were killed. Similar symptoms were produced with the alkaloid, 0.1 gr. being fatal to a mouse weighing 15 gr. Hankin also found an albumose which he injected as a prophylactic against the disease. Anthrax is peculiarly malignant in small animals. It is so surely and quickly fatal to mice, guinea-pigs, and rabbits as to make of their bodies the best physiological tests in case of doubt as to the nature of a micro-organism.

Anthrax is usually conveyed to man by contact with a diseased animal or by the ingestion of its flesh as food. Individuals most closely connected with

cattle are chiefly affected—butchers, stable-boys, shepherds, veterinary physicians, etc. On account of the great tenacity of the spores people who come in contact at any time with the skins, hairs, bristles, cloths, horns, or hoofs—as tanners, brushmakers, upholsterers (horse-hair), wool-sorters, rag-sorters, glue-makers, etc.—may be affected through open wounds in the skin or through inhalation of dusts.

Since Bollinger demonstrated the bacillus in the stomach of carnivorous flies, and with Raimbert and Davaine produced the disease by inoculation with the stomach, legs, and feelers of these insects, it must be admitted that malignant pustule may be conveyed by insects. It had long been remarked that malignant pustule occurs more especially on the exposed parts of the body, face, and hands. Bell of Brooklyn found 56 of 60 cases on the face, 2 on the hands, 1 on the wrist, and 1 on the forearm. It was evident that the bite of a fly or mosquito had often originated the disease. Extensive epidemics have been caused, as stated, by the ingestion of raw or insufficiently cooked flesh. Animals rarely contract the disease from each other; they get it from the soil. It has often been observed that certain regions are centres of infection wherein the disease shows itself year after year. The superficial burial of carcasses leads to infection of the soil, which, once produced, is seldom eradicated. The disease is spread chiefly in the warm months of summer, when the soil is softer, by animals grazing upon its surface, and is transported by streams of water, which convey the infected soil to a distance. Floods may disseminate the disease to places previously free. Stable utensils, fodder, hay from anthrax fields, litter, harness, surgical instruments, have been known to convey the disease. The fœtus is not infected as a rule. The placenta when sound acts as a filter. Exceptional cases have been accounted for by lesion of the placenta. Immunity is not secured by a single attack.

Morbid Anatomy.—There is usually marked cadaveric rigidity, sometimes, but seldom, cyanosis. Decomposition occurs early. The blood, which is black, thick, and uncoagulable, shows, especially in the lungs, liver, kidneys, and spleen, abundant bacilli or spores. The skin, when the disease has located itself in its structure, shows the signs of an extensive destruction of tissue, with intense œdematous infiltration, sometimes with gangrene. The outlying lymphatics are swollen and hæmorrhagic. In the internal mycosis the surface may show metastatic carbuncles and petechiæ. The spleen shows constant lesions (hence the terms splenic fever, milzbrand). It is increased to double or quadruple its natural size, and is distended with blood, often to rupture. Sometimes it shows gangrene. When the affection originates in the intestine this structure shows hæmorrhagic infiltration and gangrene. The retro-peritoneal lymph-glands and mesenteric glands are hyperæmic and hæmorrhagic. Hæmorrhages into the serous sacs, degeneration of the heart-muscle, of the liver, and of the kidneys, belong to this disease, as to all the exquisitely acute and grave infections.

Symptomatology.—The disease presents itself in two distinct forms—one as it originates externally, the other internally. The external disease is the

anthrax, malignant pustule, or charbon, with its lesions in the skin and subjacent tissues: the internal is the intestinal or thoracic mycosis, which is recognized by the general signs of toxicæmia, the nature of which may be, if unsuspected, overlooked. The external disease is confined to individuals; the internal may assume, as stated, endemic and epidemic proportions.

The period of incubation varies from one to several days. Symptoms may show themselves in a few hours after inoculation; they may be delayed as late as four days. A slight itching, prickling, or burning sensation is first perceived on the face or neck at the site of inoculation. Sometimes the patient feels as if he had just been stung by an insect. Very soon there appears a papule with a central vesicle, the rupture of which discharges bloody contents, to be converted into a dark red-brown or black crust, the anthrax. Smaller vesicles may appear about it. The parent nucleus, as Virchow called the first eruption, rapidly extends, the skin swells about it, becomes indurated, livid, and hard. The subcutaneous tissues are extensively infiltrated with serum. The appearance is characterized as a "brawny œdema," which rapidly spreads to involve a mass of tissue, the whole of one arm or of one side of the neck, in the course of a few days. Lymphangitis and swelling of the lymph-glands with phlebitis are frequent complications. For the first day or two there may be no disturbance of the general health. The patient may even continue at work, but toxic signs set in, as a rule, by the end of the second day with delirium, diarrhœa, sweating, vomiting, and collapse, and so the patient may die of heart failure in five to eight days. This result, however, is not so frequent as was formerly supposed. In the majority of cases the local inflammation begins to abate in the course of a few days. The anthrax sloughs off and the subjacent ulcer closes over by granulation.

A subvariety of this condition was first described by Bollinger as *anthrax œdema*. In this form the local lesion is absent. The poison seems to be introduced more deeply into the tissues, and chemical products produce an œdematous state of wide area. This variety is most often noticed in the region of the eyelids.

The internal mycosis announces itself more distinctly as an infection. The disease begins suddenly with chill, pain in the head and joints, vomiting, and diarrhœa. The case looks like a poisoning, which it is. Free hæmorrhage may occur from the mouth, nose, and kidneys. Nearly always (exceptions being noted by Bouisson) there is an outbreak upon the skin of small, phlegmonous, carbuncular inflammations, the so-called metastatic carbuncles. There is usually but little fever. There may be much delirium, convulsions, sometimes opisthotonos. There is often præcordial anxiety and intense dyspnœa. Cyanosis and heart failure usually precede the termination, which may occur in the course of a very few days.

Where the disease originates in the chest, respiration soon becomes difficult, though auscultation reveals, as a rule, only the signs of a light bronchitis. Diarrhœa is usually absent. The nervous system may be depressed or so little affected as to lead patients to decline medical advice even a few hours before

death. The case bears the aspect of a rapidly-spreading pneumonia with heart failure. Most of these cases succumb in three to five days. Bell declares that they who survive for a week recover. This form of the disease has been observed more especially among the sorters of wool. Most of the fatal cases have been hitherto unrecognized. Bell thinks that many of the cases diagnosticated as pneumonia, bronchitis, congestion of the lungs, etc., occurring among workers in carpets, blankets, furs, etc., are really cases of thoracic anthrax. It is not improbable that some of the cases ascribed to poisoning by mushrooms, meat ptomaïnes, etc. are really cases of intestinal anthrax.

Diagnosis.—Anthrax is distinguished by its origin as a red papule with a dark centre and its rapid extension with brawny œdema. The black central crust is absent, and any extensive surrounding inflammation is absent in a common boil or furuncle. Carbuncles show themselves much more frequently on the back of the neck, trunk, and extremities; anthrax occurs on uncovered surfaces. Anthrax spreads from one central point or parent nucleus; carbuncle results from the coalescence of a number of points. Anthrax œdema in the absence of a central papule is distinguished by its sudden appearance, its yellowish-green hue, and septic symptoms. Erysipelas is more superficial, has no anthrax or parent nucleus, and shows no bacteria in the blood.

The diagnosis of intestinal and thoracic anthrax is sometimes reached only by exclusion: the nature of the avocation, the exposure to the cause, is the most common index to the condition. The sudden occurrence in the midst of health of the intense signs of a grave infection—headache, nausea, and vomiting, dyspnœa, cyanosis, convulsions, free hæmorrhage, especially skin carbuncles—in connection with the history of the exposure, should lead to the recognition of the disease. In any case of doubt the diagnosis may be established by the examination of the blood under the microscope or by a physiological test. A rabbit, guinea-pig, or a mouse shows dyspnœa, dilatation of the pupils, and convulsions, with death in the course of two or three days after inoculation. The blood of these animals swarms with bacilli.

The **prognosis** is always grave; that of malignant pustule depends upon the stage of its recognition. The disease can be always eradicated at first. In places where its picture is familiar and where the disease is attacked at once, the mortality is reduced to 5 or 9 per cent., and even this mortality is ascribed to delay in treatment. Under neglect the mortality may reach 50 to 60 per cent. Intestinal and thoracic anthrax, being recognized only after general infection, have always, at least at present, a fatal prognosis.

Prophylaxis consists in the proper disposition of the bodies of dead animals by deeper burial or by cremation; in the avoidance of the use of the hides or other products of these animals; in the destruction of their discharges, as by fire; in shutting off affected pasture-fields, damming up streams of water, etc.; in the abundant use of disinfectants—carbolic acid, chloride of lime, corrosive sublimate—in handling suspected wools, horn, and other products; and in the protective inoculation of cattle and sheep with attenuated cultures or antitoxines.

Treatment must be radical. Every local manifestation must be attacked promptly and powerfully. Before absorption a diseased mass may be excised, or incised as by crucial incision, and thoroughly and profoundly cauterized, by the actual cautery, by caustic potash, or by a concentrated solution of carbolic acid or corrosive sublimate. Carbolic acid may also be injected subcutaneously—5 to 10 per cent. solutions—especially in a case of anthrax œdema. Cauterized surfaces should be dressed with weaker solutions of these or similar antiseptics, as of iodized phenol, 1 : 100, or creolin, 1 : 50.

Camera best expresses the principle of treatment with the most successful practice in countries where the condition is most frequently encountered, as follows: The mass is to be circumscribed by a deep incision and penetrated by numerous crucial incisions. In the bottom of all these cuts is to be strewn corrosive sublimate itself in powder, gr. 0.04–0.15. The liquefaction of the sublimate produces extensive, thoroughly penetrating destruction of the entire mass. Where the surface is so great as to lead to the fear of poisoning by the sublimate itself, its action may be modified and poisoning prevented by admixture with a proportion of calomel. Weil first anæsthetizes the mass with cocaine, scoops it out, and applies to the wound dressings saturated with a 1 per cent. solution of corrosive sublimate. Contento injects into, under, and about the mass, subcutaneously, 3 per cent. solutions of carbolic acid. In the œdematous form the whole infiltrate must be abundantly scarified, cut deep down to the healthy tissue in the same way, and dressed in solutions of iodine and carbolic acid.

In cases of general infection metastatic carbuncles are to be treated in the same way, and the patient supported with brandy or subcutaneous injections of ether, camphor, or other analeptic. Deformities about the nose and lips, which may follow destruction of tissue, may be subsequently relieved by plastic operations.

The therapy of internal anthrax is wellnigh hopeless. Where it is known that poisoned meat has been ingested, the stomach should be immediately washed out or a powerful emetic administered, followed by a purgative dose of castor oil. For an internal mycosis it has been recommended to administer carbolic acid in doses of 3 to 5 drops three or four times a day. It might be better to saturate the blood with creasote, as in the treatment of the sepsis of tuberculosis, and with alcohol, as in poisoning by snake-bites. Not much hope is to be entertained of either plan. The hope which seemed justified by the experiments of Fodor regarding protection by saturation of the blood with an alkali has proven futile, according to the subsequent investigations of Chor. Future success must be obtained by means of toxines or antitoxines. Hankin of Cambridge finds defensive proteids in the serum of the blood of certain animals. There is a protective albuminoid, a non-dialyzable globulin, insoluble in alcohol and water, in the blood and spleen of a rat, which renders a mouse immune against the most violent anthrax. The same matter from susceptible animals has, however, much less destructive effect, and does not confer the same immunity upon mice. Wild rats, which enjoy natural immunity, lose

it when put upon a diet of bread, and, losing it, lose also protective proteids in the blood. Very young rats, which are susceptible to anthrax, contain only traces of the protective proteids. Kostjurin and Kräinsky reached the conclusion that certain toxines from decomposition, introduced at the proper time into the bodies of rabbits affected with anthrax, totally prevent the development of the disease. The toxines must be obtained from decomposing extracts freshly prepared and well protected against the influence of light and air, else they inhibit, but do not prevent, the development of the disease. The injection must be made in five to eight hours after the inoculation, though it may be sometimes effective after twenty-four hours. More perfect results are obtained by repetition of the injection on the third or fourth day. The dose for the first injection is 0.1 gr.; for repeated injections, half of this amount, 0.05 gr. The essential principle in the extract is not the product of a definite micro-organism, but a number of them. The addition of the smallest amounts (0.1–1 per cent.) to the culture media totally destroys the virulence of anthrax bacteria without in the least hindering vegetation.

Ogata and Jasuhara claim that the blood of immune animals—*e. g.* dog and fowl—contains a ferment which, injected subcutaneously in but one- or two-drop doses, acts as a certain preventive and curative remedy. This ferment also prevents the development of the cholera and typhoid bacillus.

These disclosures of much promise have not yet been utilized in the treatment of anthrax in man.

HYDROPHOBIA.

By JAMES T. WHITTAKER.

Hydrophobia (ὕδωρ, water, φόβος, fear); Greek, Lyssa, λύσσα, rage; Latin, Rabies; French, La rage; German, Wuth, Hundswuth; Italian, Rabbia; Swedish, Hundsjuka,—is an intensely virulent infection of the lower animals—dog, fox, wolf, cat, and skunk, in the order of decreasing frequency—communicable also to man, having the most variable, often the longest known, period of incubation. It is distinguished by melancholia, terror, intense hyperæsthesia of the medulla, evinced as a spasm of the pharynx and larynx excited by attempts to swallow or the presence or the mere thought of liquids, and a subsequent very short stage of paralysis, and almost inevitable death.

The name is appropriate as expressing the most prominent symptom of the disease in man, but is inappropriate for the lower animals, as precisely this symptom, the fear of water, so obtrusive in man, is in them entirely absent. Emphasis should be laid upon this point at the start. The gravest errors have arisen in consequence of ignorance or disregard of it. Rabid dogs have been considered safe because they drank water. Rabid dogs love water. Rabies is with them a hydrophilia rather than a hydrophobia.

It is strange that while the disease appears to have been known to the ancient Indians, Egyptians, and Israelites, Hippocrates makes no mention of it. Aristotle (322 B. C.) recognized it unmistakably in dogs: "Dogs suffer from rabies. This induces a state of madness, and all animals who are then bitten by them are likewise attacked by rabies." Democritus considered it an inflammation of the nerves allied to tetanus. It is mentioned by Virgil, Horace, Ovid, Plutarch (130 B. C.). Celsus, who first uses the word, speaks of it as the disease which "*ὑδρόφοβιαν Græci appellant.*" The wound should be sucked out, he says, by means of dry cups, and should be afterward destroyed by the actual cautery. If the wound be not so treated, hydrophobia ensues—"a most deplorable malady, one in which no hope of recovery can be entertained." Galen declares hydrophobia to be the worst of all diseases, and recommends excision of the wound in protection against infection. Cælius Aurelianus discusses its modes of origin and absorption, the differential diagnosis from inflammation of the brain and mania, the course of the disease, and its treatment.

A thorough elaboration of the symptomatology in the lower animals as well as in man is chiefly due to English observers, especially to Youatt. Pasteur has connected his name with hydrophobia for all time by his studies of prophylaxis—studies which established the nature of the disease as an infection whose

symptoms are due to toxines from some as yet undiscovered micro-organism, and which fixed the fact of the first importance that rabies may in no case arise spontaneously, but always and only from itself.

Hydrophobia, like syphilis, is communicated by inoculation through a broken skin, and, while it may be transmitted by any animal, it is actually communicated to man in the great majority of cases, 90 per cent., by the bites of rabid dogs. It is therefore essential to a true understanding of rabies, as well as to the prevention of the disease, that some knowledge should be had of its main features in the dog. Rabid dogs are mad, but mad dogs are not necessarily rabid. Mad dogs may be only angry or insane, for dogs are very near to man in nervous organization. The popular idea that a dog in a fit is mad is wrong. Epilepsy is not rabies. The idea that rabies is more common in summer is not incorrect, though the ratio of cases is not greater than 7 to 15 per cent., and this increase is not due to temperature, but solely to the increased number of inoculations. It is a period of rivalry and wrangling, intensely heightened by the cruel disproportion of sex. The preponderance of male dogs affected (10 to 1) has always been observed, and is readily understood, for dogs are actually more considerate to their females than are men. It is now known, however, that rabies is not due to lack of sexual congress.

Rabies is communicated by the saliva, but is not confined to that secretion. Paul Bert found bronchial mucus virulent. Eckel and Lafosse communicated the disease with the inoculation of blood—Lafosse from dog to dog, Eckel from goat to sheep and from man to dog. Saliva has been repeatedly successfully inoculated from numerous animals, as by Berndt from ox to sheep, by Eckel from goat to sheep, Rey from sheep to sheep, Lessone from ox to horse and sheep, Youatt from horse and ox to dogs, and Ashburner from ox to fowls, King from cow to fowls, Earle from man to rabbits, Majendie and numerous other observers from man to dog. The disease has been communicated accidentally from horse to man, from sheep to shepherd, and from man to man (Law).

The average period of incubation in the dog is from thirty to fifty days. It varies, however, from six to two hundred and forty days. It is certain that the animal may communicate the disease during the whole of the period of incubation. As a rule, there are no symptoms until the end of it, when there is observed some change in the disposition; and any change of this kind is to be regarded with suspicion. In some cases there is unusual dulness and indifference, and in other cases unusual vigilance and nervousness. A morbid appetite, which leads the animal to pick up foreign bodies or devour its own excrement, is very characteristic. A dog which, hitherto affectionate, becomes morose and resentful should be regarded with distrust. *Per contra*, a sudden excess of affection in a dog hitherto lacking in this regard may betray the disease. If a social dog seeks seclusion or bears punishment without a cry, he is to be strongly suspected. "Barking without object, constant moving and searching and scraping, a disposition to tear wood, clothing, etc. to pieces, and, above all, absence from home for a day or two, should beget grave apprehensions" (Law). A dog in this stage of rabies is in a state of suppressed excitement,

to which, with uplifted head, he gives vent from time to time in a hoarse and muffled howl, a cross between a bark and a howl, the so-called rabid bark or howl, wherein one loud sound is followed by several others in diminishing force. It is impossible for a mad dog to keep quiet; he must wander; he makes long excursions, it may be of many miles, flying at any animal or man he meets as if possessed by demons. In a state of evident mania he is seized with paroxysms of wicked fury or is at intervals affected with evident hallucinations. A mad dog will often glare into vacancy, then suddenly collect himself, as from some horrid dream, with a violent start, jump to his feet, rapidly open and close his eyes, wrinkle his forehead, snarl and snap at an imaginary foe, or viciously attack any object, animate or inanimate, that he can reach, or he will gnaw to shreds an offending paw or tear off parts of his own body. He will seize and hold a stick of wood or iron bar until his teeth are broken or dislodged. Finally, exhausted by his efforts or in the further course of the disease, he gradually sinks into a state of paralysis, shown first as a paraplegia, a weakness of his hind legs, with swaying motion in walking, and by the fall of the lower jaw, which permits the escape of viscid saliva, which he still makes frantic efforts to detach. The manifestation of paralysis presages death, which occurs in the course of eight to ten days from the beginning of the disease. Throughout this whole period there is, as stated, never any hydrophobia. The dog suffers intense thirst, which he attempts to allay by plunging his head in water and lapping every fluid he meets, including his own urine. So far from showing aversion to water, he rather seeks it, and in his journeys will swim a river rather than turn from his course.

In about one-fifth of the cases the second stage—that is, the rabid stage—is entirely absent in the dog. The disease passes at once from melancholia to paralysis. In these cases there is an absence of the desire to destroy and to bite, as well as of the impulse to wander away. Paralysis may set in in the course of a single day, to show itself first in paresis of the lower jaw, which drops to permit the more or less constant escape not only of saliva, but also of everything taken into the mouth. The animal is at first able to close the mouth under powerful effort, as after extreme irritation, but rapidly loses the power altogether. Paraplegia soon sets in, and the animal dies within two or three days.

Timid animals, like foxes or badgers, lose their shyness; wolves become still more ferocious; cats are less liable to attack, but do not hesitate to use teeth and claws on occasions; infected horses and cattle bite and kick, and even fowl show disposition to inflict wounds with the beak. Animals affected with rabies are therefore truly said to be "mad."

The disease prevails very much less in some countries than in others, though statistics in the same country vary at different decades or centuries. Prevalence or absence in a country is of course wholly a matter of introduction and inoculation. Thus, hydrophobia was most common in Prussia in the last century, in one decade of which there were reported 1666 deaths, whereas at the present time the disease is actually unknown in that country, owing to the rigid

enforcement of muzzling dogs. The disease was formerly very rare in France. Trousseau declared that in his time there were not two cases for each million of inhabitants. It is now more frequent, partly, of course, on account of the importation of cases for treatment at a stage too late to be benefited. There was no hydrophobia in the isle of Cyprus or in Tasmania and Madeira, countries which abound in curs of low degree. There was no hydrophobia in Cochin-China up to 1880, since which time the disease has become so frequent as to justify the establishment of a Pasteur institute for its treatment. The disease is very rare in South America, and is by no means common in England or in our country. Watson declares that many physicians have never seen a case. But three cases have occurred in the practice of the author in a quarter of a century.

Ninety per cent. of cases are contracted by the bite of a dog, but not every case bitten by a dog turns out to be hydrophobia. A bite from an animal which is simply enraged, maniacal, or epileptic cannot, of course, convey the disease. The mere fact that a dog has fits excludes the diagnosis of hydrophobia, which never shows convulsions, though most dogs thus affected are accused, persecuted, and killed. Hence the proverb, "Give a dog a bad name," etc. The majority of individuals bitten by a dog actually mad escape the disease. This is especially the case in lesions of covered surfaces. The clothing wipes off the infection with the saliva. For the same reason most animals escape, the infectious matter being retained in the hide or wool. Hence bites on the face, neck, or hands are much more dangerous. The bites of wolves are worse than those of dogs, because wolves fly at the face. It is estimated that 47 per cent. of persons bitten by mad dogs suffer hydrophobia. A difference has been noticed even among dogs. As an instance, a dog was bitten by thirty different mad dogs without once contracting the disease.

According to Youatt, two-thirds of the dogs bitten by rabid dogs are affected with the disease. This is certainly a large percentage. Hertwig found that but 6 of 137 dogs bitten and kept under his observation died from hydrophobia, while all the rest escaped infection. This was certainly a small percentage. In these experiments certain dogs resisted infection altogether. Hertwig was unable to produce the disease in 19 of 30 dogs either by subjecting them to bites or by direct inoculation. Renault found that he could infect but 67 per cent. of his dogs. The rest remained free for the hundred days in which they were kept under observation. Hertwig had one dog which continually resisted the disease, though it was inoculated nine times in the course of three years. Other dogs meanwhile inoculated were attacked. Certain animals resist two, three, or even four inoculations, and then finally succumb to a last trial.

Escape is sometimes due to accident. A bite after a recent bite is less dangerous, because saliva may have been wiped off in the first bite. The danger is illustrated by the part of the body bitten. In some American statistics quoted by Watson, of 75 cases the wound was in the hand 40 times, on the

face 15, in the leg 11, and the arm 9 times. Of 495 cases collected by Bollinger, 53 per cent. were bitten on the upper extremities, 22 per cent. on the head and face, 22 per cent. on the feet, and 3 per cent. on the body and scrotum. The cures of the charlatans by so-called mad stones, etc., often of great virtue in psychical cases, get their reputation from use in cases which have escaped infection.

The period of *incubation* of hydrophobia covers a point of the most intense and anxious interest. How long after a bite may an individual be considered safe? This is the point in which hydrophobia and lepra differ from other diseases, in that the period of incubation is so indefinite. In the majority of cases it is unusually long. Thus, in 60 per cent. the period of incubation varies from eighteen to sixty days, but in 34 per cent.—that is, in a little more than one-third of all the cases—the period is longer than two months. Abundant cases are upon record of outbreak of the disease only after the lapse of three to six months, and there are cases upon authentic record where the only exposure which could have accounted for the disease occurred one year, or even more than two years, before the attack. In a very small ratio of cases (6 to 18 per cent.) the period of incubation is short, from three to eighteen days. Sometimes these alleged long periods, as well as cases without apparent cause, find explanation in a more recent infection which has been overlooked or forgotten. Thus, Youatt traced one case in a man to an attempt to untie with the teeth a knot in a cord which had been used to confine a mad dog; another in a woman to the use of her teeth to press down the seam in mending a tear in her dress where it was caught by a rabid animal. When it is remembered that the whole period of incubation in dogs is infectious, even though the animal show no signs of the disease, it may be understood how frequent are the possible sources of infection.

Variation in the time of outbreak has been distinctly observed in dogs. On one occasion six dogs bitten by one rabid animal showed signs of the disease respectively in twenty-three, fifty-six, sixty-seven, eighty-eight, one hundred and fifty-five, and one hundred and eighty-three days.

In civilized countries the disease, as stated, is nearly always propagated by dogs. In semi-civilized countries, as in Russia, Galatia, etc., it may be caused more frequently by wolves; in the East Indies, by jackals. Taking the statistics from France, Wurtemberg, and Milan of 796 human beings, 715 were bitten by dogs, 30 by cats, 31 by wolves, 19 by foxes, and 1 by a cow.

The **diagnosis** of the disease is much more difficult in the dog than in man. The change of disposition, the desire to wander, the peculiar howl, are cardinal points. In a doubtful case the diagnosis may be made to rest largely upon the condition of the stomach found after the death of the animal, for it is declared that if the stomach and small intestine appear healthy and contain normal food, the animal may be considered free of hydrophobia. If, however, the stomach be found full of indigestible foreign matter, its mucous membrane spotted with hæmorrhages, the larynx and pharynx hyperæmic or inflamed, there is strong evidence of hydrophobia. Batt insists, however, that this con-

dition of the stomach is often found in or after other diseases, and that a diagnosis may not be declared from post-mortem evidence alone.

From almost the first recognition of the disease in man attempts have been made from time to time to deny its existence altogether, and to consider hydrophobia a fright and form of hysteria or of tetanus. The fact, however, to say nothing of inoculation experiments in animals, that so many children under the age of five—9 per cent. of all cases in France—and so many idiots and imbeciles, in whom the imagination could play no rôle, have succumbed to the disease, sufficiently disproves this view. The symptoms, as will be seen, distinctly differ from tetanus, and the most that may be said of the hysterical origin is the fact that hysteria may simulate hydrophobia or any other disease.

Notwithstanding the searching investigations at the hands of the best observers, especially in connection with the study of prophylaxis, the cause of hydrophobia remains unknown. The analyses of chemistry have failed to disclose it. No specific micro-organism has been detected in the saliva or other fluid, and no distinct toxine has been eliminated from any of the secretions or tissues of the body. The poison is in all cases fixed, never volatile. It is produced only within the body, never outside of it. It acts in every respect like a chemical poison which is evolved from micro-organisms, but differs from all the known poisons by the length of time in which it may remain innocuous in the body. Other secretions than the saliva, as well as the flesh of animals, as a rule, fail to convey the disease. Though the poison is in the cord, the cerebro-spinal fluid is not infectious (Wyssokowicz); the aqueous humor is certainly not infectious (Cardelli); the gastric juice destroys all virus (Wyrskowski), as is shown by the fact that a fox ate without damage the cords of several affected foxes and dogs (Nocard).

The poison of hydrophobia (rabies) is certainly fixed in the nervous system in the large nerves, and especially in the medulla, and eminently in the salivary glands. Introduction of matter from these tissues directly into the brain (dura) develops the disease in from two to seven days; introduction into other parts of the body develops the disease only after a long interval—one to six months—as after bites of rabid animals. Whether the blood be infectious is a question upon which authorities are nearly evenly divided. It is probable that the blood is infectious only for a short time, and that it then secretes the poison in the nervous tissue. The injection of large quantities of a concentrated virus directly into the blood not only does not infect large animals (sheep and goats), but actually protects them against inoculation even after trephining. Roux, Nocard, and Protopopow confirm this fact and propose to utilize it in prophylaxis. Helmann found that the introduction of concentrated virus into the subcutaneous tissue not only did not infect, but absolutely gave immunity to, dogs, monkeys, and even rabbits. Thereupon, Ferran ventured to inject as much as 40 ccm. of the "virus fixe" into the subcutaneous tissue of man in the treatment of hydrophobia. Pasteur found such injections sometimes fatal to dogs, and Celli succeeded in producing rabies in ten to

twenty days after the introduction of the cord of rabid animals into the peritoneal sac of rabbits.

Various theories have been proposed to account for the long latency of the disease—to wit: First, the virus inoculated remains latent at the wound until it may accumulate to sufficient extent to inundate the blood and the body. This view would seem to find support in the prevention of the disease by the exsection or destruction of the wound; but the fact that the disease may be conveyed at any time during the period of incubation is a sufficient refutation of it. Second, that the poison is not taken up by the lymphatics about the blood-vessels, but travels slowly along the course of nerves until it finally, in the course of weeks or months, reaches the central nervous system. This mode of invasion has been more frequently considered in tetanus. Einhorn went so far as to declare that he had been able to trace up a line of inflammation along the course of the ulnar nerve in a case of hydrophobia. The nerves on the bitten side contain more virus than those on the sound side (Roux). Third, the poison lies latent at the wound, and from it chemical products are gradually introduced into the blood, but are neutralized from time to time by the serum of the healthy blood, by the so-called protective proteids which act as antitoxines or antidotes, until finally they fail to permit intoxication. This view has now the best support. It accounts for the escape of so many cases, with the simultaneous infection of others. It furnishes an explanation of the fact that the bite of a dog in the stage of incubation may be, but is not always, infectious. It accounts also for the favorable influence, even to the prevention of the disease, of the destruction of it at its origin. It allies it with other poisons, as in a case of septicæmia, where the removal of a local dépôt may put a stop to a long train of septic signs. This view is, however, only a theory as yet. It is claimed by the Pasteur school that exsection and cauterization will not prevent infection, any more than such treatment will prevent vaccination. Escape from infection in this doctrine means failure of inoculation.

Morbid Anatomy.—Notwithstanding the tempestuous and terrible signs of the disease, little or no lesion may be discovered upon autopsy. The symptoms are explained by the action of a virulent chemical poison which does its work, disappears, and often leaves no trace. The negative evidence thus encountered is testimony of great value. Some signs of catarrhal inflammation are usually to be seen in the throat, more especially in the larynx. The lungs show both hyperæmia and œdema. Spots of ecchymosis are sometimes found in the pericardium and heart. The kidneys are deeply injected; the epithelial lining of the tubules is more or less opaque, and sometimes shows molecular degeneration. The blood is black and thick.

The only really important changes are encountered in the brain, and very frequently they are entirely lacking. On removal of the calvarium the brain is found wet. The longitudinal sinus is filled to distension with fluid, black blood. Sometimes there is evidence everywhere of extensive hyperæmia. The only changes which can be said to be at all characteristic are microscopic, and they are, with the rest, sometimes entirely absent. The small vessels are

dilated, and invested upon their exterior with leucocytes which invade also the circumjacent tissues. These changes are most marked in the medulla and the upper part of the spinal cord, as well as in the cerebral cortex, whence the symptoms of hydrophobia arise. (See Figs. 32, 33.) Gowers observed this condition in 7 of 9 cases. Emigration or accumulation of leucocytes is at times so great as to fill up the whole space within the lymphatic sheath.

FIG. 32.

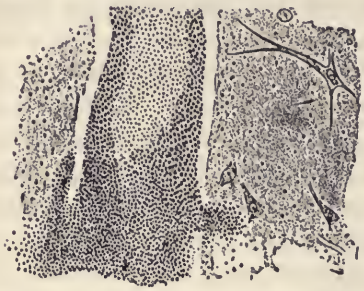


FIG. 33.

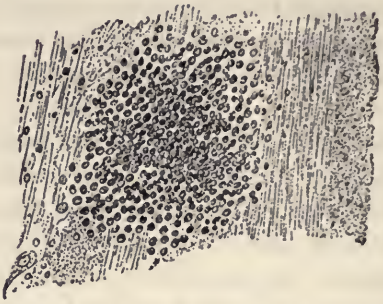


FIG. 32.—Hypoglossal Nucleus: leucocytes around a vessel and extending into the adjacent tissue (Gowers).

FIG. 33.—Accumulation of Corpuscles ("miliary abscess") in the Fibres of Origin of the Hypoglossal Nerve (Gowers).

These escaped and accumulated cells constitute what may be called miliary abscesses. In association with them are observed at times small hæmorrhages, seldom large enough to be visible to the naked eye. This perivascular accumulation of leucocytes, especially in connection with the vessels of the medulla and cortex, constitutes the most constant and characteristic lesion of hydrophobia. Unfortunately, as stated, this sign, with all the rest, is sometimes entirely absent.

Symptomatology.—Hydrophobia is divided into three stages—the melancholic, spasmodic, and paralytic. The disease is usually announced by changes at the seat of the wound, which, as a rule, has long since healed. There is, it may be said, nothing in the nature or course of the healing process in an infected different from that in a simple wound, The bite of a mad dog heals as quickly and kindly as that of a healthy dog. The wound may show no change from the beginning to the end of the disease. Sometimes no trace of it can be discovered, but not infrequently, as stated, inflammatory changes set in at the cicatrix, which may become reddened or swollen. The wound may open anew or become the seat of pain, itching, numbness, or other paræsthesia. Sometimes pain radiates from it in various directions. Sometimes the first feeling is in the nose or throat, a sneezing, a dryness, or a rawness which is considered a "cold."

A peculiar state of depression or irritability soon sets in, sometimes suddenly, with headache, anorexia, insomnia, anxiety. Mental symptoms assume prominence according to the temperament of the individual. A man may deny the fact that he ever was bitten by a dog, while he is unable to divert

his mind from the actual occurrence and the terrible consequences which are liable to ensue. The inquiry or suggestion of a thoughtless, meddlesome, or inquisitive neighbor will plunge the strongest man into melancholy or mania. The mental distress is, however, always an exaggeration of a state of apprehension, of a sense of impending danger or imminent death, and, though a man may show under the stress of this suffering signs of insanity, there is no time when he may not be recalled to himself by a right address. A patient affected with the first stage of hydrophobia is a pitiful picture. He sits quietly, apparently listlessly, his whole mind intensely concentrated upon the one thought, from which no appeal or address may really divert him. It is only in the very first hours of the attack that he may find relief in walking about or in change of scene. He soon becomes exhausted, and sits with an expression of intense anxiety, to which he makes total surrender. At the same time, the special senses are keenly alert, so that a flare of light, a draught of air, a noise, may produce intense excitement. The very first day shows the characteristic sign of the disease, the fear of water. The patient suffers with thirst, but is unable to allay it. He may make the attempt, may succeed at first in swallowing a mouthful or two, but soon abandons it, either on account of the intense suffering which ensues or from the fear of its certain following. An unmistakable sign of the disease is the occurrence of burning, more especially a sense of tightness or constriction, of the larynx. The fear of water is the fear of exciting spasm of the larynx, and the reflex excitability of the larynx becomes so intense that spasm is later precipitated by the sight, the sound, or the mere thought of water or the mere sight or touch of a smooth or cold surface. A coachman under Watson's observation had to desist from sponging himself, according to his habit, with cold water, though he said he "could not think how he could be so silly." Frequent sighing is a common sign at the inception of the disease.

The first stage usually lasts about twenty-four hours, when the second stage, the spasmodic or true hydrophobic stage, sets in. This stage is characterized by an exaggeration or an intensification of the spasmodic contraction of the larynx. Every attempt to swallow is attended with frightful anxiety. The contraction is so powerful as to lead to dyspnœa, with maniacal excitement. The patient may strike about in every direction, roll his head from side to side, while the mouth opens and closes convulsively, sometimes with snapping sounds, whereby wounds are occasionally inflicted upon ministering hands, and the disease has actually been conveyed in this way. These convulsive seizures gave rise to the stories that hydrophobic patients bite and snap like dogs, and led, through the fear which they excited, to the cowardly assassination of patients by shooting them down—a practice still in vogue on the confines of Austria—or by smothering them between feather beds. The paroxysms seem all the more dreadful because they are attended with the escape of glutinous, foaming saliva, which is sometimes ejected with great force in every direction. Inspiration is also attended with gaping and sighing and various sounds, sometimes simulating the bark and howl of dogs. These

symptoms occur in paroxysms, in the intervals between which the mind is clear, though sometimes, in those of highly nervous temperament, it may be excited to show more persistent hallucination. The pulse is quickened and rendered irregular, but with all the struggle there is, as a rule, but little elevation of temperature.

The inspiratory spasms and convulsive attacks may cease entirely. The patient may become able to swallow with perfect ease. The recovery is illusory, as the case usually suddenly succumbs to heart failure.

The second stage is thus characterized by the severity and intensity of symptoms. It is easy to be seen that the convulsions are in all cases of reflex origin, and the disease is characterized by extreme hyperæsthesia of the medulla, whence the convulsive manifestations emanate on the very slightest outside irritation. Another distinguishing feature is the mental anguish, the unspeakable terror, which is depicted upon the face. During the paroxysms the patient may lose his self-control. He may foam at the mouth, make snapping movements in convulsions, whereby he may even accidentally or apparently purposely inflict wounds upon attendants, but so soon as the paroxysm is over he recovers consciousness, and usually apologizes pitiably for his excess. He may even warn his attendants to subject him to greater restraint in protection of themselves. The employment of any forcible measures, however, as a rule, aggravates the explosion.

The second stage usually lasts from one to three days, rarely as long as four days. The patient now becomes gradually exhausted. Paroxysms occur, but they are less intense. The extreme anxiety of mind is diminished; there are intervals of nearly complete tranquillity. It is plain to see, however, that while the breathing is easier and the explosions less severe, and while there may be even ability to swallow, the patient becomes more and more prostrated and reduced. The strong man is broken. The heart's action is weak, the pulse flutters, the surface is covered with a cold sweat. The movements of the body are so much enfeebled as to present the appearance of paralysis; hence this second stage has been characterized as the "stadium paralyticum." Death, which may occur suddenly in a convulsion or from asphyxia, usually comes on quietly from failure of the heart.

Hydrophobia is, as stated, an exquisitely acute infection. However long the period of incubation, the whole duration of the disease proper is measured in a few days. Of all cases, 82 per cent. perish in from two to four days. Individual cases may succumb in two, or may last as long as five or six, days.

The **diagnosis** is generally easy, and rests chiefly upon the heightened reflex of the medulla as manifested by spasm in the muscles of deglutition and respiration.

The disease is differentiated from tetanus by its much longer period of incubation. Tetanus occurs in from three to ten days after the wound or injury. Tetanus usually begins with trismus, and is often attended with opisthotonos. It lacks the laryngeal symptoms and spasms of hydrophobia. It lacks also

the psychical exaltation and mental anguish of hydrophobia. Tetanus may also be distinguished by its special micro-organism.

The disease is often distinguished with great difficulty from the imaginary condition known as lyssophobia, or fear of hydrophobia. These cases have a common origin, though in the one case the wound comes from a non-affected animal. It might be imagined that lyssophobia occurred more frequently in nervous subjects or in women. This is not the case. The strongest men have suffered, and not infrequently actually succumbed to fright or fear of hydrophobia. Some of these cases have been rescued by knowledge of the fact that the animal was not rabid; hence the advisibility, when possible, of secluding the animal, that the existence or course of its disease may be observed. The fact that the animal recovers at all almost necessarily excludes hydrophobia. Abundant cases are recorded where information of the recovery of the animal or the sight of the animal itself has allayed the most intense nervous symptoms.

The **prognosis** is fatal. It is commonly said that the physician who cures is death. Bollinger goes so far as to say that the cases of alleged recovery may be invariably found to be due to some other disease or to the fact that the animal was not rabid. Yet it must be admitted that dogs have recovered from the disease. Law mentions eight such cases, two of which were attested by successful inoculation of other animals. The possibility of spontaneous recovery may therefore be entertained in man. Wounds on the face are, as stated, always the most serious. Bouley declares that 90 per cent. of these cases are followed by hydrophobia, whereas the mortality from wounds of the hands is 63 per cent., of the lower extremities 28, and of the upper extremities 20 per cent. Bites in the vicinity of motor nerves are the most dangerous; the wilder the animal and the longer the teeth, the shorter is the incubation and the more grave the prognosis (Babes). Many cases are certainly rescued by prompt treatment. Bollinger quotes in proof of this the following statistics in France: Of 200 human beings bitten by rabid animals, 134 were cauterized. Of these, 92—that is, 69 per cent.—remained healthy, while 42—that is, 31 per cent.—died of hydrophobia. Of those non-cauterized, 83 per cent. succumbed to the disease. In one case 16 persons and 1 ass were bitten by the same animal. The human beings were cauterized and rescued without exception. The ass, which received no treatment, died of the disease.

The only true **prophylaxis** is through the enforced use of the muzzle, which renders all other prophylaxis superfluous; but for various reasons, including a kind of sentimentality, the process of muzzling has never been rigidly enforced outside of a military country like Prussia. The disease, which was formerly common in Prussia, was actually extinguished, as stated, for nine years by the rigid enforcement of universal muzzling. Holland secured the same exemption in the same way. The number of dogs may be limited by imposition of higher taxation. Every dog should have a known master. Suspected dogs must be carefully confined for as long a period as six months. Dogs imported from countries of lax laws in this regard should be quarantined for six months. Actually rabid dogs or other animals that need not be

preserved to determine the condition of human beings or other animals attacked should be killed at once. Filing the teeth or attachment of blocks of wood about the neck, confinement by chains, attempted prophylaxis by injection of virus, are all means too unreliable for practice.

Treatment consists in the destruction or elimination of the poison in the wound. Absorption should be first prevented where practicable, as on the extremities, by a ligature above the wound. A piece of cord or handkerchief should be firmly twisted about the limb with a piece of wood. Where it may be done the patient should withdraw the poison from the wound by suction. With proper precautions this act may be substituted by another person. The act of suction is, however, dangerous in cases of carious teeth or wounds in the gum, cheek, or other parts of the mouth. The operation may be performed, nevertheless, if the individual take the precaution to rinse the mouth thoroughly after every suction with carbolic acid. Hertwig found that the virus of hydrophobia applied to the mucous membrane of the mouth and digestive tract was entirely innocuous. This process, which has been resorted to from the most ancient times, has never yet proven infectious. In the first decades of the present century in Lyons certain women, *hundsäugnerinnen*, pursued this business as an avocation. They received ten francs for the first, and five for each succeeding act. On the surface of the trunk and some parts of the face the poison may be exhausted by cups. Immediately after suction the wound should be cauterized. Youatt relied entirely upon such a superficial caustic as the nitrate of silver. As he was himself bitten seven times and operated on 400 persons, only 1 of whom died—and that one, as he declared, from fright—this caustic may be considered sufficiently strong if applied immediately. Caustic potash burns deeper. The actual cautery, as from a poker, a nail, the galvano-cautery brought to a white heat, would certainly destroy the poison more effectually. Where wounds are very extensive or numerous, the effect may be best accomplished with stronger solutions, 1 : 500 or 1 : 1000, of corrosive sublimate. Extensive laceration of extremities may require amputation.

Psychical treatment is of supreme importance. Romberg first advised the necessity on the part of the attendant and friends "to preserve a calm demeanor, to avoid all allusion to the previous injury, and to appear cheerful." To secure diversion without effort or remark is an essential factor in the relief of suffering at least. The intense reflex excitability of the medulla is best met by seclusion in a quiet and rather dark room. The exhibition of cases as curiosities or as objects of morbid sympathy is a cruelty, if not a crime. Frequent warm baths where at all permissible, as at the very start, tend to allay excitability and spasm. Very soon, however, resort must be had to anodynes and anæsthetics. Violent cases may require the use of chloroform. The same object may be at first obtained with chloral. The various remedies recommended as specifics—curare, Calabar bean, pilocarpine—have proven useless except in allaying spasms. The use of animal poisons has proven equally futile. Watson speaks of cases treated with the virus of snake-bites. One

man was bitten by nine vipers without effect. Opium is the best shield. Sooner or later resort must be had to morphine, in the later course of the disease preferably subcutaneously, with a view to at least secure euthanasia. With this history hitherto it may be appreciated with what acclamation was hailed the claim by Pasteur of the discovery of a means of preventing the disease by the use of attenuated virus. It had been always known that the disease expends its main force upon the medulla. Whatever lesions are encountered in the disease are seen here.

As soon as Pasteur had determined that the virus of hydrophobia comes to be located in the central nervous system, especially in the spinal cord, he began his experiments with this substance to secure attenuated matter. He found that a continued inoculation of the virus from rabbit to rabbit increased its virulence to such degree that after about twenty-five generations he got a virus which showed its effect after an incubation of but eight days. In twenty-five generations further the period of incubation was limited to seven days. This virus was taken as a so-called *virus fixe* as a basis substance for protective inoculation. Pasteur discovered that desiccation of the medulla from such an animal in sterilized glass vessels in which had been put pieces of caustic potash brought about a gradual reduction of virulence. The medulla became less and less poisonous. The drying process was continued, until after two weeks' desiccation it was entirely innocuous. Injections were now made with an emulsion of the non-virulent medulla, and were followed by emulsions of medullæ of increasing virulence, up to those which had been dried but one or two days. Dogs so treated were immune to infection with fresh hydrophobic matter.

In the treatment of the hydrophobia of man Pasteur began with weaker preparations—to wit, with the medulla of the rabbit after fourteen days' desiccation, and increased the following days up to that of the fifth day, whereby immunity or protection was secured. The attempt to use stronger preparations in a shorter time in protection against the more dangerous and extensive laceration of wolf-bites had to be discontinued. This treatment has been used now in thousands of cases, and, while it cannot be said to have furnished perfect results, as a number of cases thus treated have nevertheless succumbed to the disease, it must be admitted that the majority of cases have been rescued from the horrors of hydrophobia.

A better method is promised in the conclusions of Centanni, who utilized the principle of antirabic vaccination (inoculation), first devised by an old Italian physician, Eusebio Valli, in the production of an innocent virus obtained by the action of gastric juice upon the cords of infected rabbits. The cord, emulsified in peptones, gradually parts with its virulence, and loses it altogether in twenty hours. The essential substance is a flocculent deposit, which may be preserved for weeks in glycerin or dried with sulphuric acid. Rabbits can be thoroughly immunified with this material.

The protective substance is prepared as follows: Four grammes of spinal cord are emulsified with artificial gastric juice (solution of English peptones)

for nineteen hours. At the end of this time a few drops are aspirated from the mixture and injected into the sheath of the sciatic nerve of two rabbits. The rest of the emulsion is neutralized with bicarbonate of sodium and filtered. The essential substance which remains on the filter is repeatedly washed for several hours with distilled water, and then dried. The nearly dry matter is divided into three equal parts, two of which (each with 5 ccm. of neutral glycerin) are put into tubes; hydrogen is introduced into one, the air is exhausted from the other, and the two tubes are united by fusion. The third part is dried by sulphuric acid.

The glycerin emulsion in the tube suffices, in five subcutaneous inoculations, to render rabbits absolutely immune in six days after infection with the ordinary virus (strassengift), while one of the two non-protected control animals died in seventeen, the other in eighteen, days.

In subsequent experiments made by Tizzoni and Centanni it was ascertained that this matter not only protected against, but actually cured, the developed disease (guarire negli animali la rabbia sviluppata). Five rabbits infected by injection into the sheath of the sciatic nerve with virus, which killed control animals in fifteen to seventeen days, were inoculated with the protective matter, 11–26 ccm. in doses of 3–5 ccm., twice on the seventh, once on the eighth, once on the eleventh, and once on the fourteenth day after infection. The injections were intravenous, subcutaneous, and intraperitoneal. In all five cases the symptoms of rabies had more or less fully developed. All five remained without a sign of subsequent infection, and the effect was the same in all three, regardless of the method of application of the serum.

This discovery, which promises results of inestimable value, has, up to the period of the present writing, not yet been utilized in the treatment of hydrophobia of man.

TRICHINOSIS.

By JAMES T. WHITTAKER.

Trichinosis is an acute infection caused by the ingestion of the trichina (θρίξ, τριχὸς, hair) spiralis in raw or underdone pork,—characterized by gastritis and enteritis, followed, in consequence of migration of the parasite, in the course of a week by pains in the muscles and contraction of the joints, with œdema of the eyelids and face, prostration, insomnia, profuse sweats, and exhaustion.

The history of the trichina is wholly modern, and is all included in the present century. Calcified capsules were seen in the muscles as early as 1821, when Hilton first described them as minute white masses, which he regarded as cysticerci. Peacock made a preparation for the museum of Guy's Hospital in 1828. Paget, at that time a student at St. Bartholomew's, distinctly noticed them in the muscles of man. He took a specimen to Owen in 1835, who gave the parasite the very appropriate name it bears. Leidy in 1847 discovered in a piece of ham upon his plate the same immature nematoid, but neither he nor his predecessors appreciated the significance of its presence. Herbst (1851) bred muscle trichinæ in dogs by feeding them with the infected flesh of a badger. Leuckart (1855) first saw the escape of intestinal trichinæ from their capsules in the body of mice. In the same year Küchenmeister maintained that the trichina was the larval state of the trichocephalus dispar. This erroneous view was more widely disseminated by Leuckart, who declared that he had bred trichocephali in myriads by feeding trichinous flesh to hogs. Virchow was unable to confirm this conclusion, as he observed the immature trichinæ become in all cases mature in the intestine of the dog. Cases of muscle trichinæ continued to be occasionally reported in England and America, as well as Germany, from this time on, but the capsules were still looked upon as dissecting-room curiosities, sprinkling muscles like the deposit of eggs of insects and preventing good preparations. Even the discovery of young trichinæ in the capsules in the breeding experiments of Leuckart and Virchow contributed in no way to dissipate the belief that these worms were innocent, wandering nematoids.

The new era of definite knowledge began with the year 1860, and with the study by Zenker of an individual case, that of a servant-girl in the hospital at Dresden. This famous first patient was attacked about Christmas, 1859, with anorexia, insomnia, lassitude, depression, and fever. The case was regarded as typhoid fever, a diagnosis which had to be changed with the supervention of a train of symptoms which could not be classed with the

signs of this disease. By the end of a week the patient complained of severe pain all over the body, more particularly in the muscles, and more especially in the extremities. Any movement of the body increased the pain to such a degree that the patient was compelled to lie with the limbs flexed upon the body. Pneumonia subsequently set in, and the patient died at the end of a month after the first signs of disease. A post-mortem examination revealed trichinæ in myriads, all alive and in all stages of development, lying coiled and curved and straight in the sheaths of the muscle-fibres and in all the striped muscles, including the heart itself. It was subsequently ascertained that this domestic had showed signs of illness shortly after the ingestion of a meal of ham and sausage. Zenker was fortunate enough to find some of the same meat and to discover in it encysted trichinæ. It turned out also that other domestics had suffered, and that the butcher, who was in turn sought out by the indefatigable observer, had been ill with rheumatism, "as if paralyzed," ever since he had killed the pig. He was at the time of observation unable to move his arm, legs, or neck. He had never in his life been ill before, and attributed his attack to a bad cold contracted on the day of the slaughter.

Zenker forged the last link in the chain of his investigations by sending a piece of the muscle of the girl to Virchow, who fed a rabbit with some of it, developed in the animal general muscular paralysis, and recovered young trichinæ from its muscles. With this flesh other rabbits were fed with the same result. Virchow, Leuckart, and Zenker then demonstrated the migration from the intestine to the muscles, and two years later (1852) Friedreich made the first diagnosis of the disease in life by the detection of the parasite in a piece of exsected muscle.

Thus in less than a year the clinical history of the disease was established by a chain of evidence which was complete in every link. It was now soon recognized that many cases, even epidemics, which had hitherto been considered as irregular or anomalous typhoid or typhus fever, more especially malignant rheumatism, occasional cases of poisoning, English sweat, and black death, were due to this cause. Thus attacks and epidemics at Wurtemberg, Breslau, Magdeburg, Hamburg, various districts in France and Belgium, are recognized to have been cases of unsuspected and unknown trichinosis. On one occasion nearly the entire crew of a merchant-vessel sailing from Valparaiso to Hamburg was killed by the ingestion of pork from a pig killed on the sea.

Fact is stranger than fiction. At a festival at Heltstadt, Prussia, of which 103 persons partook, more than 80 persons were attacked with the disease, and more than 20 died within a month. The cause was subsequently discovered in some sausage-meat taken from a sick pig killed by mistake. Three or four days after a church celebration in 1846 seven of eight people were seized with diarrhœa, pain in the back of the neck, and swelling of the face. Four of the seven died. The remaining three continued ill for a long time. The innkeeper was suspected of having poisoned his guests with white wine, and, though the charge could not be proven, the odium was so great as

to compel him to close out his business and leave his native land. Eighteen years afterward Langenbeck of Berlin, in exsecting a tumor from the neck, discovered in the platysma myoides innumerable dead trichinæ enclosed in calcified capsules in the body of one of the survivors, and thereby the history of the "poisoning" was made plain.

Trichinosis was diagnosticated for the first time in the United States by Schnetter of New York (1864), and in the same year by Voss in four cases on board one of the Bremen steamers in the harbor of New York. The disease was first recognized in England in Northumberland in 1871.

The trichnia spiralis is present in man in both the developed and undeveloped states—developed, mature in the intestinal canal; undeveloped, immature in the muscles. The trichina also infects the hog, rat, cat, fox, rabbit, and guinea-pig. It is a pure parasite, the sole example among the entozoa, having lost all relation with the external world.

The hog is usually infected from itself. It was formerly maintained that the hog got the disease from the rat, which was once considered the original host of the parasite. Leisering went so far as to declare that "to exterminate the rat is to exterminate trichinæ." Zenker showed, however, that rats get their infection from the hog, and Gerlach established the fact that, whenever trichina is found in the rat, trichinous hogs or other carnivora are within reach of the rat. The hog is therefore the peculiar and original bearer of trichina. In it the whole course of the evolution of the trichina takes place (Heller).

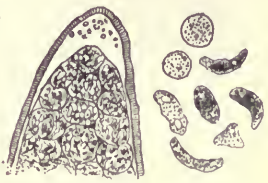
In the inspection of meat care must be taken to avoid considering certain objects found in the muscle as encapsulated trichina. First, the so-called Miescherian sacs, sausage-shaped psoro-sperms which may usually be distinguished by the fact that they are traversed with transverse lines and discharge upon incision minute kidney-shaped objects (see Figs 34 and 35) which may undergo change of form; second, certain crystalline objects found in the muscles, described by Virchow as the guanine gout; they may be distinguished by the fact that they dissolve on the addition of hydrochloric acid, to leave the muscle-fibres unaffected.

Fig. 34.

Miescher's Sac, × 100 (Leuckart).

About 2 per cent. of swine are found to be affected, and trichinæ are found in the muscles in about 2 per cent. of cases in the dissecting-room.

Fig. 35.



End of Sac, with kidney-shaped bodies free and enclosed (Leuckart).

Anatomy, etc.—The mature intestinal trichina (see Fig. 36) is round, elongated, white, and, as its name implies, extremely filiform, on which account it is barely visible to the naked eye as a fine wool hair or silvery thread. The head, formerly regarded as the tail, is drawn out almost to

a line, while the caudal extremity is somewhat rounded off and is not much thinner than the body. The alimentary canal begins with a muscular mouth,

Fig. 37.

Fig. 36.

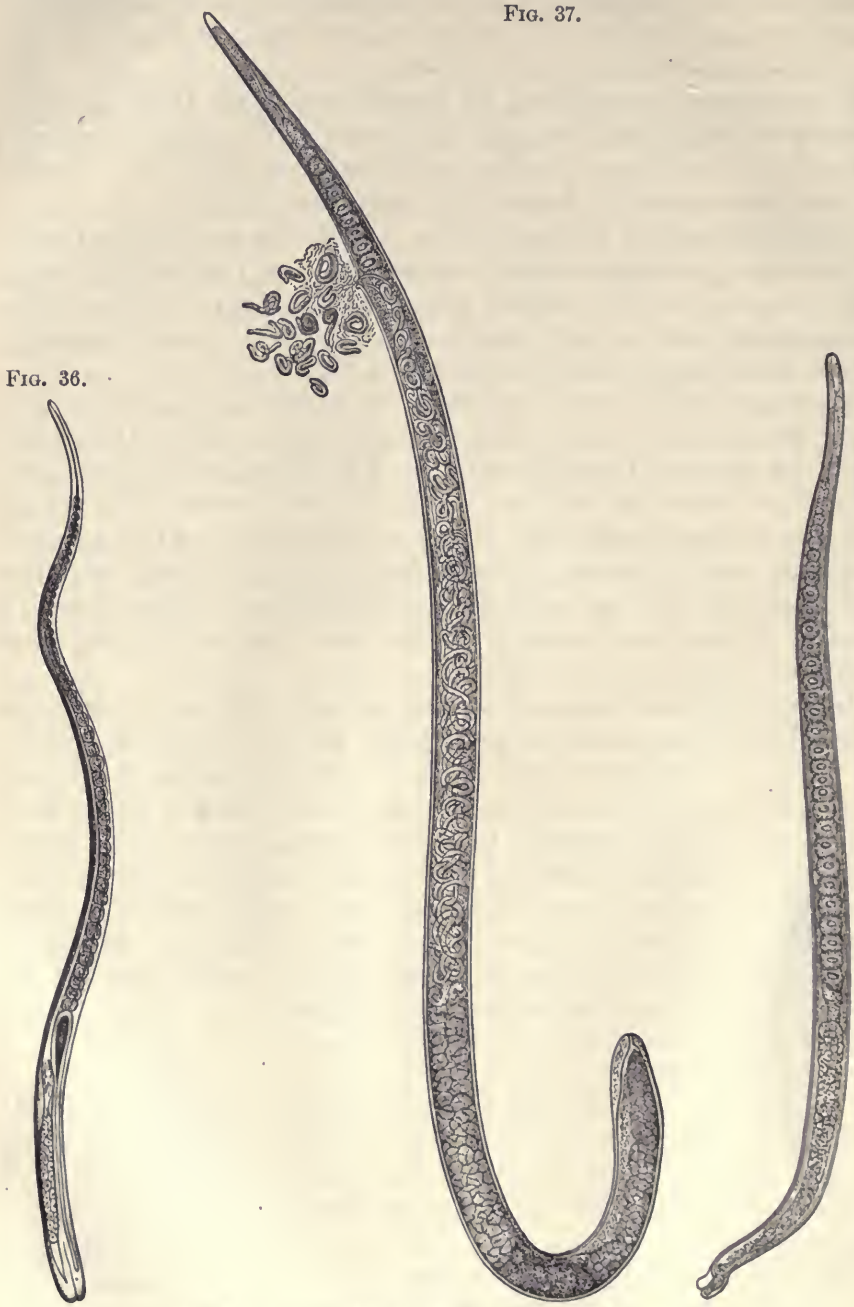
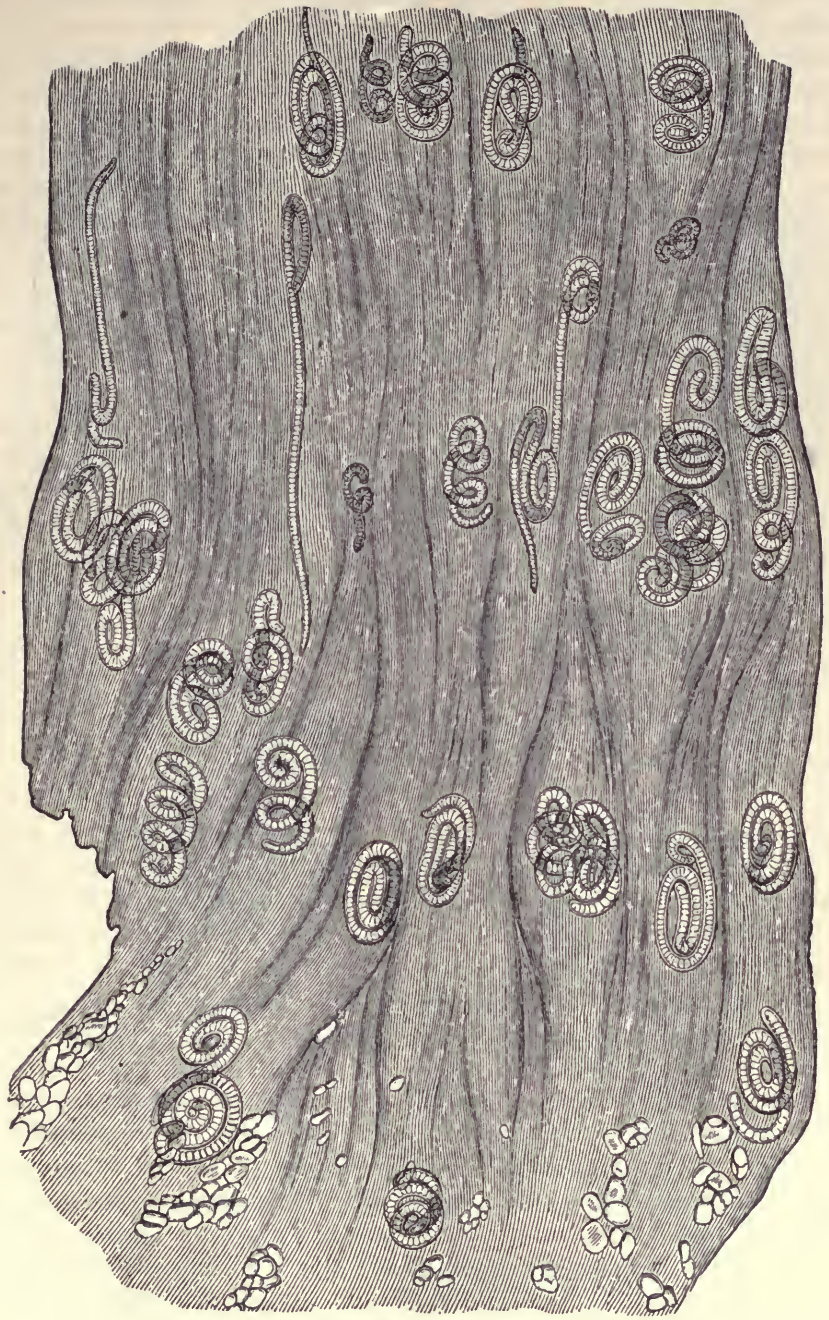


Fig. 36.—Unimpregnated Female Trichina (Leuckart).
Fig. 37.—Male and Female Trichinæ, female discharging young (Heller).

is continued into an elongated œsophagus, expanding into a flask-shaped stomach, to be again continued into an intestinal canal, which at its extremity

Fig. 38.



Tendinous Insertion of Muscle, showing Trichinæ (Heller).

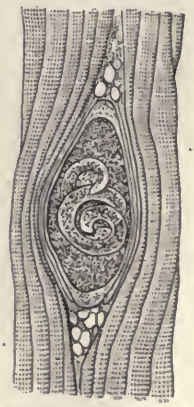
receives in the male the opening of the seminal duct, arising from a single testicle, a thick cul-de-sac which runs along the side of the body. The female, $\frac{1}{8}$ inch in length (see Fig. 37), is twice as long as the male to accommodate the ova with which it is stuffed, and which are hatched within the body and born alive. The orifice of the vagina is situated at the junction of the first and second quarters of the body. Each female may give birth, in the course of a month, to over a thousand young. The parent trichinæ are short-lived. They are probably, for the most part, digested and absorbed after reproduction, as they disappear from the intestine in from five to eight weeks, and are, unfortunately, not often to be found in the stools.

The newborn, immature trichinæ, $\frac{1}{200}$ of a line in length, having escaped from the body of the parent, penetrate the intestinal wall, probably by means of chemical irritation, to migrate, chiefly along the meshes of the connective tissues, to contiguous muscles, more especially to the diaphragm, abdominal, intercostal, laryngeal, cervical, ocular, and proximal muscles of the extremities, in which latter region they are crowded, as if arrested, at the tendinous extremities. (See Fig. 38.) Here they continue to grow for fourteen days to the length of half a line, when they coil up to assume the peculiar spiral form (see Figs. 39 and 40), disintegrating the muscular tissue, expanding and thickening the

FIG. 39.

Living Embryos (Heller).

FIG. 40.



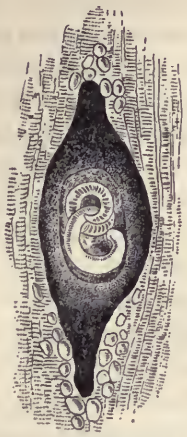
Encapsulated Trichina (Leuckart).

sarcolemma, and, as a result of the inflammatory process thus produced, leading to the formation of a lemon-shaped capsule $\frac{1}{5}$ of a line in length, in which they lie for the most part singly, or more rarely in groups of two, three, or even four. Thus they remain encysted alive for a year or more, exceptionally as long as twenty-five years, or become subsequently calcified after calcification of the containing capsule—a process which begins at the poles of the cyst, but is not of necessity fatal to its contents even when complete. (See Figs. 41 and 42.)

Muscular tissues thus infested when taken as food (one ounce containing at times fifty to one hundred thousand parasites) is dissolved in the process of digestion, liberating from their capsules, in from three hours to three days,

the muscle trichinæ, which attain sexual maturity in the intestinal canal in five days, and then reproduce their species with the rapidity described. Trichinæ have also been found in the blood, mesenteric glands, and peritoneal cavity.

Fig. 41.



Calcified Trichina (Leuckart).

Thus the trichina spiralis, which was formerly regarded as an accidental and innocent inhabitant of the muscular tissue, has been unmasked since the first observation of Zenker in 1860 as one of the most widely disseminated and deadly of all known parasites.

Fig. 42.

Calcified Trichinæ, natural size (Heller.)

Symptoms.—The symptoms of trichinosis vary according to the quantity of trichinæ ingested and the irritation produced. Small numbers produce no symptoms, calcified remnants having often been found in autopsies with a history of absence of any symptoms in life. A certain stage of development and capsulation is also requisite to infection. Too young or insufficiently protected trichinæ are killed in the stomach. Calcified capsules may not liberate their contents. The irritation, with the consequent rapid increase of peristalsis in childhood, often causes the expulsion of trichinæ unliberated from their capsules in the stools. The ingestion of alcohol in large quantities with the meal may destroy them as rapidly as they are liberated.

The stage of invasion, which shows itself in from three hours to three days or longer as successive quantities may be ingested, is characterized by irritation on the part of the stomach and intestines—*i. e.* by anorexia, nausea, vomiting, tenderness to pressure, pain in the bowels, and diarrhœa. These symptoms may be absent altogether or may vary greatly in intensity, to assume at times such severity as to be mistaken for cholera, as in the epidemic of Hedersleben, where three victims died on the sixth day. Animals fed with trichinous flesh often succumb on the fourth day. The fever, thirst, headache, and general prostration, which may accompany the local signs, belong equally to other causes of intestinal irritation, and are not peculiar to trichinosis. Sensations of distress, nausea, or vomiting may occur within a few hours, and may repeat themselves for several days. It is curious that the appetite may be at one time keen and at another entirely absent. Diarrhœa is much more common and persistent. Gastric distress is often entirely absent. Affection of the intestine is present in the majority of cases.

Characteristic symptoms announce the advent of the stage of migration and colonization in the muscles, which begins, as a rule, on the seventh day with œdema, functional disturbance, and pain in the muscles. Œdema shows itself first or is noticed first, as a rule, in the eyelids, disappearing in a few

days, and returning later in the course of the disease. This œdema is often coincident with pain, tension, and restriction of movement in the muscles of the eyes as evidence of early invasion of these muscles, though the presence of œdema here as elsewhere, in the absence of muscular signs, has also been ascribed to the action of some toxic principle acting upon the vaso-motor system. Œdema of the face is often, that of the hands and feet more rarely, associated with that of the eyelids. Pronounced œdema of the skin over the affected muscles occurs even more constantly than about the face—is absent, in fact, in only 10 per cent. of cases. This cutaneous œdema also disappears for a few days, to return later. It is distinguished from the œdema of heart and kidney disease by its association with the muscular signs, as well as by the fact that it spares the genital organs, the scrotum, and labia majora.

Muscle-symptoms appear on the ninth or tenth day as a rule, delayed at times to the fourteenth, varying in every grade of intensity from lassitude, stiffness, or tension to board-like indurations and most atrocious pains. The flexors of the extremities, the biceps and muscles of the calf especially, become swollen, tense, and tender, the extremities being held in semi-flexion to simulate the postures of acute articular rheumatism. Invasion of the diaphragm, abdominal, and intercostal muscles gives rise to dyspnœa; invasion of the masseters, which may excite trismus, renders mastication painful or impossible; while invasion of the tongue and pharyngeal muscles may restrict or prevent deglutition, accounting thus for the rapid emaciation. Invasion of the larynx is shown by hoarseness of voice or aphonia in 20 per cent. of cases, and of the ocular muscles by fixation of the eyeball, chemosis, and occasionally by mydriasis and nystagmus. Impairment of hearing follows invasion of the stapedius muscle.

Difficulty of deglutition, alteration of the voice, especially hoarseness, even aphonia, indicate attack of the muscles of the neck. The most serious results ensue in consequence of invasion of the diaphragm and intercostal muscles. Most cases of trichinosis suffer occasional attacks of dyspnœa. In fact, difficulty of breathing may come on early under toxic influence, as in the case of affection of the muscles of the extremities. There is at this early period also, in the majority of cases, and probably for the same reason, more or less bronchial catarrh; and when this condition persists into the period of immigration catarrhal pneumonia may result from insufficient expansion and defective expectoration. The development of pneumonia is difficult to recognize on account of the decubitus of the patient. Patients occasionally succumb to hypostatic pneumonia, unsuspected for the same reason.

None and Hoeffner observed in a certain number of cases lack of the triceps and patellar reflex. It was absent for some time, and then gradually returned. There was no case in which any increase was observed, though the skin reflex was unchanged. Both the direct and indirect excitability of muscles under the galvanic as well as under the faradic current was markedly reduced.

Among the disturbances of sensation may be noticed at times paræsthesia,

more particularly pruritus. Anæsthesia is much more rare. Kratz called attention to the violent neuralgic attacks in the abdomen, the so-called cœliac neuralgias, which occur more especially in the second week, and most frequently at night. They are marked by an intense griping pain at the pit of the stomach, radiating to the back, attended with pallor, prostration, coldness of the extremities, and collapse.

Sweating is another common symptom of trichinosis. It occurs early, always in connection with the muscular pains, and is profuse and distressing in correspondence with the severity of the latter. It is often attended with miliaria, occasionally with herpes. Pustular eruptions—Friedreich once found a free trichina in a pustule—acne, furunculosis, may follow the disappearing œdema of the face.

Still another quite common as well as obstinate symptom is insomnia, which often rapidly exhausts the patient. With this exception the cerebrum shows no symptoms. Though most cases are characterized by apathy or depression, the brain remains clear up to the last stages of the severe attacks, when somnolence, stupor, or delirium may for a short time precede the end.

Fever does not belong of necessity to trichinosis. Average cases show slight elevations of temperature, which at times present the course of remittent, or more frequently of typhoid, curves. Fever may be entirely absent throughout the history of the disease. Severe forms may be accompanied by a temperature of 104° F. When present it is irregular, and not infrequently assumes, as stated, a typhoid form. The confusion of trichinosis with typhoid fever was formerly common, and the mistake has occurred not infrequently even since the days of accurate thermometric observation. The irregularities of the second and third week somewhat simulate the amphibolic stage of typhoid fever. Defervescence is never so regular or classical even when it occurs at the time of the fall in typhoid fever.

Trichinosis develops no special symptoms in the genito-urinary apparatus. There is, as a rule, no interference with the course of pregnancy or of the puerperium. Trichinæ have never yet been found in the fœtus.

Morbid Anatomy.—The most obtrusive condition is œdema, which shows itself about the face and extremities of infected patients at any time after the fourth week of the disease. The contractions which existed before death are continued after it, especially in the arms. The blood is usually found fluid, and there are free effusions in the various serous sacs. Hyperæmia may be still present in the mucous membrane of the small intestines. Ecchymoses are not uncommon. Trichinæ may be sometimes discovered in the intestines even as late as the eighth week of the disease. The spleen is not enlarged. The liver is usually fatty. The heart is often flabby. The lungs show signs of bronchial catarrh and hypostasis.

The most characteristic changes are encountered in the muscles. They are at first simply somewhat pale; later they become cloudy, and still later streaked and shrunken. The trichinæ are found most abundantly in the diaphragm, the intercostal muscles, and the muscles of the neck. In the extremities the

body of the muscle is frequently free, and the trichinæ are crowded, as stated, about the tendons. Under the microscope it is seen that the muscular tissue has lost its striæ. Nuclei increase in number and size. Spindle-shaped connective-tissue cells develop in the intermuscular connective tissue. The lesion affects the muscular structure itself, the protoplasm. The sarcolemma thickens about the trichinæ to constitute the capsule. The muscular tissue subsequently suffers waxy degeneration.

Post-mortem examination generally shows hyperæmia of the brain and its membranes, enlargement of the heart, hypertrophy of the walls of the left ventricle, fresh pleuritic effusions, hyperæmia of the mucous membrane of the bronchi and of the whole alimentary canal, migrating trichinæ in all the muscles.

Duration.—The disease lasts from two weeks in the lightest cases to eight weeks in pronounced cases, and with sequelæ for the greater part of a year in the severest forms. Kunze heard complaints of rheumatic pains in bad weather four years after the Hedersleben epidemic, and Kratz found weakness of the muscles in one case eight years after the attack.

It is a recognized fact that tolerance to trichinæ varies in different individuals. Children, as stated, void them readily, on account of the increased peristalsis of the alimentary canal in childhood. Old people are less readily affected. Certain numbers may be tolerated with impunity in adults, but the continued ingestion of infected meat may introduce amounts in excess of this tolerance, when gastro-intestinal signs may more or less suddenly supervene. Where small quantities have been introduced, or where the mass of the trichinæ has been voided, gastro-intestinal signs may be entirely absent and the subsequent stage of migration may be but little marked. The existence of the parasite may then be recognized or suspected to account for long-continued, persistent muscular pains in the remoter history of the individual. The exact duration of the disease may therefore not be determined. Usually the lightest cases terminate in a few weeks, while the gravest extend over many months; but the very lightest so-called ambulatory cases may suffer pains for months or years, and the cases which show the sharpest signs of invasion may escape with the mildest signs connected with colonization. It may be said that the average duration of the disease ranges from three weeks to three months.

The **diagnosis** is illuminated often by the fact that others are simultaneously affected and by the inspection of suspected pork, possibly by the detection of mature or encapsulated trichinæ, more especially after a brisk cathartic, in the mucus, but never in the fluid contents of the voided stools; positively by the discovery of immature trichinæ in the muscles extracted, preferably after linear incision under antisepsis, from the deltoid or lower part of the biceps muscles—for the most part an unnecessary procedure. A history of gastro-intestinal irritation, followed by constipation, œdema of the face on the eighth day, and muscle-signs by the tenth day, with sweating, insomnia, headache, thirst, and fever, sufficiently characterizes the disease.

The diagnosis is assisted, as stated, by the fact that the trichinosis attacks not one, but a number of individuals: at the same time it may be helped by the known habits of individuals with regard to the character of food and its preparation. The distress of the stomach, more especially the diarrhœa which follows the ingestion of pork in the course of a few hours or a few days, next the tenderness of so many muscles, especially at the extremities, then the œdema of the eyelids and face, later the pain in the muscles and contraction of joints, serve to distinguish the disease.

The most valuable of all the early signs of trichinosis is œdema, because it is so rarely absent or, as Heller says, so insignificant and transitory as to be overlooked. Usually it shows itself first, as stated, in the eyelids and face as early as the seventh day of the disease. In the extremities it occurs later, not before the ninth day. With that of the eyelids it disappears to return later, more pronounced than ever. It cannot be attributed to occlusion of vessels, but is probably due, as Friedreich claimed, to the action of a toxine on the vaso-motor nervous system.

Œdema about the eyelids and face, the result of local disease, as from thrombus of the orbital veins, compression of the cavernous sinus, is very rare, and is of course unpreceded by gastro-intestinal irritation and unattended with pains in the muscles, sweatings, etc. The absence of any disease of the internal organs sufficient to account for the dyspnœa, hoarseness, insomnia, œdema, sweats, and pains finally leads to the recognition of the disease even when its nature is entirely unsuspected at first.

Cholera is eliminated by the profuse sweat and œdema which belong to trichinosis. The cramps of cholera may, like trichinosis, attack the muscles of the abdomen and extremities, but they do not attack the diaphragm or intercostal muscles and muscles of the neck.

Articular rheumatism, which has pain and sweating in common with trichinosis, is distinguished by the affection of the joints proper, more especially of the smaller joints, by the absence of gastro-intestinal irritation, dyspnœa, insomnia, and affection of the muscles of the jaws and eyes.

Muscular rheumatism selects by preference other muscles than those affected in trichinosis, and is unattended with gastro-intestinal irritation, œdema, fever, and sweats. Yet Grawitz, Virchow's assistant, declares that trichinæ were found, on autopsy, in one-third of the cases of so-called muscular rheumatism.

Typhoid fever is differentiated by its mental disturbance, characteristic temperature curve, diarrhœa generally throughout the whole disease, meteorism, and is not attended with œdema, asthma, and muscular signs.

Meningitis shows herpes as a rule, hyperæsthesia, opisthotonos, a contracted abdomen, and has a different history.

Finally, polymyositis, which shows pain in the muscles, tension, deformity, prostration, œdema, sweats, and insomnia—in short, most of the signs of trichinosis—is distinguished by isolated attacks—*i. e.* of individuals—by the absence of history and gastro-intestinal signs, preference of the extensor

muscles, and exemption of the diaphragm, larynx, tongue, and pharynx. Excised portions of muscle show hyaline or waxy degeneration, but no trichinæ.

Trichinosis is always a serious disease, and the **prognosis** in apparently the mildest cases must be stated with reserve; for the mildest invasions are sometimes followed by the gravest symptoms in the later course of the disease. The mortality rests largely upon the amount of meat ingested, but depends also upon the degree of heat to which it has been subjected. In this regard there is a great difference between the outside and inside of a large piece of meat. Most cases of infection occur, however, after the ingestion of raw or underdone sausage and ham. Sausage too quickly taken from the fire, and uncooked in its interior, is actually the most common mode of conveyance of the disease. The fact that the infected may be often mixed with sound meat explains the difference in intensity of symptoms in different individuals partaking of the same quantities of meat at the same meal. Children almost never succumb, because most of the trichinæ are ejected by diarrhœa. Patients who survive the eighth week generally recover. Severe myositis or dyspnœa, profound prostration, and nervous symptoms aggravate the prognosis. Recovery is, as a rule, much more tedious and protracted than after other acute infections of corresponding severity. The mortality ranges from 1 to 70, averaging 30, per cent. Death occurs usually from exhaustion or blood-poisoning in from four to six weeks—exceptionally earlier from gastro-intestinal irritation, and later from hypostatic pneumonia and marasmus.

In a recent epidemic in Saxony, 235 cases in thirteen places showed a mortality of 14½ per cent., while the general mortality in that country for the previous five years was but 1.06 per cent. In this latest reported epidemic the first symptoms appeared from the sixth to the tenth day after the ingestion of insufficiently smoked sausages. The symptoms were anorexia and nausea, pain in the stomach, severe diarrhœa persisting for several days; thereupon contractions and pains in the joints, pains in the muscles, swelling of the legs, more rarely of the arms, hands, and neck; œdema of the eyelids was always present. There was little bronchial catarrh and less pneumonia. The heart was irregular from the start. Death followed in from the second to the tenth week, most frequently at the third and fourth week, from dyspnœa and heart failure; in the more protracted cases from chronic marasmus.

Prophylaxis.—Naked-eye inspection of meat does not disclose the trichina spiralis except in cases of calcification, and calcification is not necessarily fatal to the trichinæ. Putrefaction does not destroy them. Copious libations of alcohol with meals is a preventive as unreliable as unadvisable. Smoking and pickling, as ordinarily practised, kill only the surface trichinæ. A temperature of 160° F. is fatal to the trichina, so that thorough cooking of meat offers a sure prevention of infection. A long subjection to high temperature is requisite to secure penetration to the interior of a large mass of meat of the necessary grade of heat.

Treatment.—Successful therapy depends upon an early diagnosis, which is

often unattainable. A brisk cathartic, calomel, gr x–xx, castor-oil f℥j, or infusion of senna, followed by irrigation of the colon, offers a hope of discharging many of the worms before they have been liberated from their capsules; and, inasmuch as Kratz and Cohnheim found trichinæ in the stools as late as the twelfth week, it may be said that it is never too early or too late, for purposes either of diagnosis or of therapy, to give this method trial. Recently liberated trichinæ may be benumbed and more readily discharged by the administration of thymol, ʒj–iss, divided into two or three doses (capsules), or extract of male fern, ʒj–iv. After colonization in the muscles the treatment becomes purely symptomatic. The hope of radical extermination by rapidly diffusible agents, picric acid and benzine, or water-extracting agents, glycerin and alcohol, has proven illusory. Applications of hot water, salicylic acid, gr. vij, salicin or salol, gr. x, more especially phenacetin, gr. x, or antipyrine, gr. v, every hour, may be tried in relief of pain not so great as to indicate morphine, which becomes a necessity in severer cases. Sodium bromide, gr. xl, phenacetin, gr. x, chloral, gr. xv, may suffice to secure sleep, which is, however, in bad cases forced only by morphine. As the safety of the patient depends upon speedy encystment of the trichinæ, a process which is hindered by motion of every kind, repose and quiet as absolute as possible should be enjoined and secured. The strength is to be sustained by alcohol and food until the force of the disease is spent.

GLANDERS.

By JAMES T. WHITTAKER.

Glanders (from Lat., *glans*, gland); Farcy (from *farcio*, to stuff); Greek, *μᾶλις*; Lat., Malleus, Maliasmus; Ger., Rotz, Wurm; Fr., Morve,—an infection, acute and chronic, of the horse and allied solipeds, ass and bastards; communicable by inoculation to many domesticated animals (but not to cattle) and to man; produced by a specific bacillus; characterized by the formation of nodules (granulomata) and ulcers in the mucous membrane of the nose, with discharge of fœtid pus as from glands, whence glanders, and also by deposits in the skin and subcutaneous lymph-structures, whence farcy; and subsequent general infection.

Apsyrtus, a veterinary surgeon in the army of Constantine the Great, is credited with having made the first mention of glanders under the name *malis*, a term which included, however, many other maladies. Vegetius also spoke of it, and Aristotle described it in asses. The disease had in former times a much more intense interest in that to it at various periods was credited the origin of syphilis, tuberculosis, scrofula, diphtheria, and pyæmia. The chief interest of glanders at the present day is in connection with diseases of the horse.

The first case of glanders in man was recognized by Lorin, a French military surgeon, in 1812. The case was that of a veterinary surgeon who had performed an operation upon a diseased horse, and had become affected with the disease in the form of tumors on the fingers of both hands. The tumors in this case were extirpated and the man recovered in fourteen days. Schilling of Berlin and Muscroft of England recorded accurately-studied cases in 1821. Rayer published the first monograph in 1837. Virchow contributed exhaustively to the pathology of the disease in 1855–63.

The question as to the possibility of spontaneous origin was definitely denied with the discovery by Löffler and Schütz (1882) of a specific bacillus, the bacillus mallei, which these observers isolated, cultivated, and inoculated to reproduce the disease in the horse.

The bacilli of glanders much resemble in form and size those of tuberculosis and leprosy, though shorter and more slender than either. They are immobile, maintain their virulence when desiccated for three months, and are readily colored with alkaline aniline dyes. They form a characteristic colony on the surface of the potato as a delicate yellowish transparent coat, like a thin layer of honey, as early as the second day. Acting upon the methods of Koch with

tuberculin, Kalning (Dorpat, 1891) succeeded in extracting from cultures a product which he proposed to use in prevention and treatment. Unfortunately, Kalning fell a victim to inoculation with the disease, but his studies were immediately taken up by Preusse (Danzig) and Pearson (Berlin), who also succeeded in extracting a dark-yellow, rather opaque, oily fluid of peculiar odor and neutral reaction, which they called mallein (glanders lymph), and with which they obtained characteristic reactions in horses affected with the disease.

The original seat of the disease in the majority of cases is the nasal mucous membrane, whence it may be disseminated through the body, to show itself more especially in the skin. The disease may be always recognized unmistakably by the examination of tissue exsected from the masses in the nose or in the skin. The bacilli are not readily recognized in fluid secretions, as they are easily destroyed by other bacteria. Field-mice cannot be used for the physiological test, as they are so exquisitely susceptible to the bacteria of septicæmia. The guinea-pig is to be preferred, as offering a much more exclusive soil.

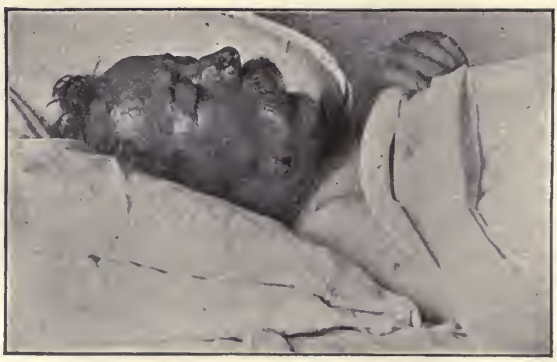
The disease is disseminated through the lymph-vessels and also through the blood-vessels, and is communicated to man either through a broken integument, especially in the nose, during the process of currying or feeding, or through other contact with diseased horses, as in slaughtering, skinning, and tanning. It is sometimes, but much more rarely, conveyed by the ingestion of infected meat, a mode of infection more common in animals fed upon horseflesh, as in menageries. The most unsuspected and unavoidable infection (fortunately, of most rare occurrence) is that which occurs in inhaling into the nose or open mouth the discharges from a horse's nose or mouth, as after the act of sneezing, snorting, coughing, etc. Exceptional cases have been recorded from drinking from the same pail used in watering horses or from the common use of a handkerchief. The bacillus may also be lifted into the air and disseminated in the vicinity of the animal, especially in close apartments, stables, etc., whence it may be inhaled into the respiratory tract of man. The disease has attacked and exterminated an entire family, man, wife, and four children, from the use of the same dish. Glanders occurs in the great majority of cases among hostlers, coachmen, drivers, stock-farmers, veterinary surgeons, butchers —that is, individuals who come in closest contact with the horse—and is of course much more common in the male sex. Bollinger found but 6 females in 120 cases, and then in the case of women compelled to substitute men in the care of horses. For the same reason children are almost exempt from the disease.

Symptomatology.—Man is much less susceptible to glanders than the soliped. The period of incubation after inoculation or inspiration varies from three to five days; it may extend to three weeks. The disease manifests itself at the point of inoculation with redness, swelling, and pain, with speedy affection of the neighboring lymphatics. Constitutional signs occur in the course of a few days. They may even precede apparent changes in the wound. Chilly sensations with fever are attended with headache and prostration. Vague rheumatic pains, more especially in the neighborhood

of the joints, with local symptoms in the skin, may more distinctly announce the infection. Where or while the local symptoms or the cutaneous signs are still absent the disease simulates typhoid fever, for which it has often been mistaken. The character of the disease is, however, soon made manifest by the appearance of hard red nodules, varying in size from a pea to a walnut, much resembling the eruption of small-pox. These nodules soon show softening of the centre, and become converted into pustules which burst, to give vent to thick, fœtid pus. The nodules may increase to such magnitude as to form tumors, the so-called farcy-buds, or in the process of suppuration constitute abscesses, the rupture of which leaves ulcers. These ulcers may destroy tissue to such depth as to expose the tendons and bones. The process may extend rapidly in twenty-four to forty-eight hours, or more slowly to persist for three or four weeks.

It is a fact, to be explained perhaps by the rôle of the nose in respiration, that while the manifestations in the skin are much less frequent and severe in the horse, symptoms on the part of the nose assuming in this animal so much greater prominence, the converse is true of man. Glanders in the nose is less frequent and severe in man than in the horse. Hauff declares that in more than half the cases in man the nose is not at all affected. Occurring in man, it shows the same symptoms as in the horse. The secretions, which may come only from the affected side, soon become changed, and the discharge from the nose shows the same thick, purulent, fœtid matter as in the case of the horse. There may be usually seen at a glance on inspection such swelling and redness of the nose and face as at times to simulate erysipelas. Sometimes tubercles may be discovered upon the alæ of the nose. (See Fig. 43). As in the horse,

Fig. 43.



Human Glanders (Pepper).

the affection of the nose may show itself later in the course of the disease, often in the second or third week. The mucous membrane of the eyes, mouth, fauces, and of the whole respiratory tract may subsequently become involved. The appearance of the membrane, with the tendency to hæmorrhage, fœtor oris, and dysphagia, may much resemble scurvy. There may be always observed

in these cases the same involvement of the glands. The submaxillary and sublingual glands may suppurate to discharge externally.

Affection of the bronchial mucous membrane is evidenced by harassing cough, with the profuse expectoration of the same fœtid matter and the subsequent development of dyspnœa. Fever may be entirely absent, or may, in an individual case, assume prominence, with a temperature of 106° F., and a feeble, irregular pulse, like that of pyæmia.

The chronic distinguishes itself from the acute form by its less intense manifestations and more protracted course. The affection of the nose, when present, does not vary in any essential from that already described. It is, however, less frequently present in man than is the acute form of the disease. There is the same purulent discharge with its excessive fœtor, the same swelling of the whole structure, while the nares are blocked with offensive crusts. Peculiar repulsiveness is added to individual cases by gangrenous changes which may occur at the root of the nose.

The manifestations in the skin are much more common, and upon these the diagnosis is for the most part established. Nodular masses may form anywhere over the body, more especially upon the extremities, to discharge sanguineous serum and pus. Sometimes the affection is more superficial, and shows itself in the form of blebs, which may, as stated, closely simulate small-pox, chicken-pox, or pemphigus. These blebs or bullæ later show, however, purulent contents or break to leave sluggish, indolent ulcers and erysipelatous appearances, which are liable to occur in the course of the disease, not only on the surface of the body, but also about the face. Lymphangitis and lymphadenitis develop as in the case of acute glanders. The whole disease runs a much more sluggish and less intense course. The fever is even more irregular than in cases of acute glanders. It is sometimes absent for a certain period, but shows itself sooner or later, if only in consequence of the extensive suppurative process, as a pyæmia. Profuse sweats with colliquative diarrhœa, as a rule, soon exhaust the patient. It may be said that the picture of glanders, like that of anthrax, varies according as the disease shows itself in local signs at its point of entry, or constitutionally as the result of absorption and dissemination in the various tissues and organs. In the first case the disease shows itself in the skin or the mucous membrane in the form, as stated, of nodules, which undergo suppuration with lymphadenitis, erysipelatous and phlegmonous inflammation. The discharge from the nose, with its characteristic hæmorrhagic appearance and fœtid odor, is often the first sign to excite suspicions of the nature of the disease. Violent pain in the frontal region indicates extension to the frontal sinuses. Chills and fever announce absorption of the bacilli into the blood. The profound prostration, more especially the depression of the sensorium, leads often to a diagnosis of typhoid fever, small-pox, or pyæmia, but the localizations in the skin, the abscesses, and ulcerative processes in the mucosæ declare the character of the disease. The various complications of pyæmia may subsequently ensue: arthritis, serous or suppurative inflammations of the various serous membranes, with exudations,

suppurating nodules, and masses in the muscles and bones, followed by extensive destruction of muscle and necrosis of bone, with deep erosions in the mucosæ and subcutaneous tissues, are common phenomena of marked cases.

These various complications may follow each other rapidly in acute cases. The blood is quickly poisoned, and the patient succumbs in the course of a week, in the more subacute cases in two to four weeks, with delirium and coma. The disease is much more protracted in chronic cases. It may last for several weeks, months, even years, and finally cause death by marasmus. There is during all this time constant liability to the development of the acute form with its more rapidly fatal consequences.

Morbid Anatomy.—The surface of the body presents the appearance of a case of pyæmia in that various eruptions, pustules, abscesses, and ulcers show themselves upon the surface, especially on the face and extremities. The predominance of blood in the contents of the pustules or nodules distinguishes the lesions of glanders from those of a simple pyæmia. The appearance of the face, the condition of the nasal and frontal bones, may at once reveal the nature of the disease. Extensive erosions, the result of masses of cicatricial tissue in the nasal mucous membrane, with necrosis of bone, are further signs of local lesion. Sometimes the septum nasi, vomer, the bones of the palate are broken down and disintegrated as in the case of the horse. Nodules may also be found in the respiratory tract, in the lungs, and in almost any of the internal organs, the brain, liver, spleen, and kidneys. The skin shows the farcy-buds, the pustules, and abscesses. Lymph-vessels and glands in the vicinity of these nodules show signs of infection. Erysipelatous and phlegmonous inflammations may be seen upon the surface and in the various membranes. Serous and purulent effusions may be found in the joints and serous cavities, where also bloody effusions are not uncommon.

The **diagnosis** is made to rest upon the nature of the avocation and the possibility of exposure. It is further determined by the two signs which have given names to the disease—to wit, the glanders, which finds its analogue in man in the term ozæna. It is to be remembered, however, that ozæna applies also to fœtid discharges from the nose from various other causes, notably from syphilis. The second factor is the farcy, the nodular eruptions, abscesses, and ulcers found in the skin. The disease is recognized in its constitutional form by the signs of pyæmia—that is, by the chills, fever, and sweats, hebetude, delirium, and coma, together with the various metastatic dépôts.

Syphilis may be separated in a doubtful case, *ex juvantibus*, as iodine and mercury have no effect upon glanders.

Tuberculosis shows, as a rule, predominating signs on the part of the lungs, and while it may affect the bones, as in a case of glanders, tuberculosis distinguishes itself by sparing the nose and skin, organs of selection in glanders. Small-pox is more uniform in its eruption. The pustules of glanders appear in successive crops and rapidly ulcerate (Liveing). Pyæmia usually results from a single centre or dépôt, which may be recognized or discovered. Cryp-

togenetic cases may be distinguished at times only by the discovery of the specific micro-organism of glanders.

The diagnosis of glanders really rests absolutely upon the recognition of the bacillus mallei. Travers, long before the discovery of the specific micro-organism, established the diagnosis in doubtful cases by inoculation of goats and rabbits with matter discharged from some of the ulcers. Bollinger recognized the disease in the same way by the inoculation of a horse. The inoculated animals showed the special lesions and succumbed in the course of two or three months. Cornil succeeded in inoculating two of fifteen guinea-pigs by rubbing cultures into the intact skin. Washbourne and Schwartznecker established a diagnosis of human glanders by the isolation of the micro-organism, its cultivation, and the inoculation of animals. Jackowski called attention to the affection of the testicle that occurs in these cases, and Strauss adopted the method of intraperitoneal injection as the quickest means of absolutely identifying the disease by implication of this organ. He was led to adopt this method on account of the difficulties attending the inoculation of animals with the products of the disease. Subcutaneous injections in dogs do not always give definite results, and the inoculation of less susceptible animals—*e. g.* guinea-pigs—is unsatisfactory because of the length of time before death, twenty-five to thirty days. Field-mice and marmots succumb in two to five days, but these animals are often difficult of access.

After the intraperitoneal injection of the discharges of glanders into the bodies of male guinea-pigs there is observed first, as a prominent lesion, affection of the testicle as early as the second to the third day. The scrotum becomes tense, red, and shining; the epidermis desquamates. Suppuration speedily occurs to perforate the integument, and in the pus is to be found the bacillus mallei. The animal succumbs at some time between the fourteenth and fifteenth days. The complication results also under subcutaneous injection, but much later, ten to twelve days. Löffler showed that it was not only the tunica vaginalis, but also the parenchyma itself, which showed nodules of the disease. The tunica vaginalis is covered with granulations, and by the third to the fourth day its layers are agglomerated by an exudation of pus rich with bacilli.

A means of diagnosis is also offered with the injection of malleïn (Preusse), which, as in the case of tuberculin in tuberculosis, produces a peculiar reaction in glanders.

The **prognosis** in a case of acute glanders is absolutely unfavorable. The only possible rescue may result from the speedy destruction and thorough annihilation of the first infection. Nearly all of the acute and more than half of the chronic cases succumb to the disease.

Prophylaxis.—Animals affected with glanders are to be isolated and killed. According to the records of the Berlin Health Office (1890), there were reported as affected with glanders 1337 horses; 80 died, 93 were killed at the request of their owners. There were destroyed by the police 1598

animals. In all, 1771 horses perished. For those killed by the police there was paid by the state 459,834.08 marks indemnity.

The cadaver is to be cremated or buried deep. Litter and fodder are to be likewise burned, and stables thoroughly disinfected. All persons who have come in contact with infected horses should be warned of danger.

Treatment.—Local dépôts are to be treated thoroughly and promptly by the application of the actual cautery, strong carbolic acid, mineral acids, and corrosive sublimate. Chronic cases are to be supported with quinine, arsenic (Gamgee), and alcohol.

FOOT-AND-MOUTH DISEASE.

By JAMES T. WHITTAKER.

Synonyms.—Lat. Aphthæ, from Greek ἄφθαι (Galen), Epizooticæ; Ger. Maulklauenseuche, Klauenseuche; Fr. Stomatitie aphtheuse; It. Febbre aftosa.

Definition.—A mild, acute infection of the lower animals, especially of cattle, sheep, pigs, less frequently of the goat, horse, much more rarely of fowls, dogs, and cats; evidently caused by a peculiar micro-organism not yet exactly defined; characterized by the formation of vesicles and ulcers in the mucous membrane of the mouth, with the development of eruptions and ulcers in crevices about the feet, sometimes about the udder, communicable to man for the most part through the milk of diseased animals, to appear, with malaise and light fever, as vesicles and ulcers in the mouth, of benign course and short duration.

The disease was recognized in animals in antiquity, but was in the early history of veterinary medicine evidently confounded with anthrax and actinomycosis. Hierocles, a Greek veterinary surgeon, seems to have been familiar with it. Livy certainly described it. Fracastorius (1513) speaks of the vesicles in the mouth and cleft of the hoof as they occurred in animals in an epidemic in Italy and France. Sagar (1764) first noticed the disease in man as caused by the ingestion of the milk of cows. It was attended with a sense of heat and dryness in the mouth and throat and difficulty of swallowing, due to an inflammation and aphthæ which were to be observed in the mouth. Brosche (1820) first saw eruptions upon the fingers and toes in the case of two young girls who had to do with diseased cows. Bollinger makes mention of an epidemic which prevailed in Bohemia in 1827, affecting both man and mule. Hertwig (1834) established the contagiousness of the disease by experimenting upon himself and two other medical men. They drank daily for four days a quart of fresh milk from diseased cows. Symptoms of fever, headache, dryness and heat in the mouth, and itching in the hands and fingers began in two and lasted for five days, at the end of which time vesicles appeared in the mouth. The disease has now, therefore, a recognized place in human pathology. Though benign in its manifestations and course, it is nevertheless a serious affection, from the fact that so many young animals, sucklings, succumb on account of degradation of the milk. It is stated that in some epizootics as many as 75 per cent. of sucking calves perished. The disease, once developed, is exceedingly persistent; stables remain infectious for a long

time. It is then gradually transported along the lines of travel, hence along the courses of rivers, and with a general tendency westward, to assume at times very wide range. Thus in the year 1871, 700,000 animals were attacked in England alone, entailing in the same year in France a loss of 30,000,000 francs. In 1869 the disease ranged over nearly all Europe. Switzerland alone loses by it about 10,000 francs per year. It makes up for its mildness by its range, and costs a country more than the malignant diseases, anthrax, glanders, and rinderpest.

The infectious principle, evidently a micro-organism, has not yet been distinctly isolated. It is certainly distinctly communicable by inoculation. Nesswitzky (1891) conveyed it with the contents of vesicles and secretion of ulcers as well as with milk. The period of incubation is variously stated at one to twelve days. Some of the animals were attacked in twenty-four hours after inoculation, some not until four or five days, some not until five to seven days. Inoculation failed in the experiments of the Berlin Health Office in 30.3 per cent. of cases. Klein (1886) in his studies of the disease in sheep eliminated a streptococcus which he believed to be the cause of the disease. It had much resemblance to the streptococcus pyogenes. Pure cultures of it injected subcutaneously developed in sheep no symptoms of the disease. On the other hand, sheep fed with these pure cultures showed the typical symptoms of foot-and-mouth disease. The curious observation was made with these studies that animals previously treated to subcutaneous injections remained exempt after feeding experiments. Klein hence concluded that inoculation conferred immunity. Some doubt pertains to these conclusions, because of the lack of control experiments and the means of excluding spontaneous infection. The immunity conferred by inoculation is a discord in the records of artificial immunity. The infectious matter exists in the contents of the vesicles, in the saliva, in nearly all the secretions, and certainly in the blood and milk.

Siegel found in an epidemic of stomatitis in man a very delicate bacterium, ovoid in shape, an elongated coccus or a very short bacillus, which developed in agar or gelatin without fluidifying the soil and without being colored in the usual way. He believed it to be derived from cattle affected with foot-and-mouth disease.

Schottelius in his studies discovered a peculiar streptococcus, some examples of which were rounded, while others had a peculiar elongation or protuberance like that which shows in the prolongations of white blood-corpuscles. He called these bodies streptocytes. They differed in many particulars from the ordinary streptococcus, but gave only negative results in inoculations of various animals. Schottelius was never able to observe the micro-organisms described by Siegel in cases of foot-and-mouth disease.

The disease shows itself in the lower animals as a mild fever attended with a catarrhal inflammation of the mucosa of the mouth. There soon develops on the inner surface of the lips and along the edge of the jaw, where the teeth are absent, at the tip and borders of the tongue, yellowish-white vesicles, which

show later purulent contents, and rupture in the course of one to two days, to leave superficial erosions and ulcers. The ulcers heal in the course of three to six days.

The affection of the feet may show itself at the same time or later than that of the mouth. In the clefts and at the crown of the hoofs there is to be observed the development of the same vesicles, which rupture to discharge purulent contents, which in turn inspissate to form crusts and leave more or less extensive ulcers. The affection of the feet renders the animal unable to stand or walk, so that at the height of the disease it must maintain the recumbent posture. Similar appearances are to be observed also about the udder, especially at the orifices of the milk-ducts. Thus, vesicles, pustules, crusts, and, in consequence of their detachment, more or less extensive ulcers, show themselves about the bag. The milk of the affected animal is altered in quantity and quality. It is reduced often as much as one-half in the human, assumes a yellowish colostrum-like appearance, and coagulates prematurely. It has a bitter, nauseating taste and develops a dark-yellow sediment. The disease terminates usually in twelve to fourteen days.

Man is usually affected through diseased milk, which retains its infection even when added to coffee or when diluted with normal milk in the proportion of 1 to 10. Boiling absolutely destroys the poison in the milk and renders it perfectly harmless. It is doubtful if the disease can be conveyed by the meat of diseased animals, but instances of infection have been reported from the ingestion of butter and cheese made from the milk of diseased cows. Infection by direct inoculation, as in milking, is not uncommon in those who have the care of diseased animals.

The chief interest in connection with foot-and-mouth disease occurs in relation to aphtha, which is declared to be the expression of the disease in man. It has been observed that aphtha prevails coincidently with outbreaks of the foot-and-mouth disease in cattle. What lends also especial support to this view is the fact that the appearance of the disease is much the same in man as in animals. The question is not yet settled.

The period of incubation in man ranges from three to five days. The disease may begin with chills or chilly sensations, followed by fever, anorexia, and malaise. Vesicles now appear upon the inner surface of the lips and tongue along with a sense of heat and dryness; there is difficulty in speaking, chewing, and swallowing. The mucous membrane is very much reddened and swollen, and saliva flows abundantly. There is also often noticed at this time a vesicular eruption on the fingers and hands, sometimes in association with intestinal disturbance. The vesicles upon the fingers are at first small and transparent. They soon increase in size, and change in color to show purulent contents, and sometimes closely simulate the eruptions of small-pox. Cases have been reported where the eruption was so extensive as to cover the entire body (Biercher). Holm saw vesicles on the nipple of a woman who drank daily large quantities of milk from cows affected with the disease.

The catarrhal inflammation may assume such proportion as to constitute

an extensive stomatitis. Briscoe saw a case in which the tongue was so much swollen as to project more than an inch from the mouth.

Prophylaxis includes proper care of the animal regarding pasturage and stables. Man is best protected by the ingestion of milk from healthy cows, or, if that be impossible, by the thorough boiling of milk from diseased cows.

The **diagnosis** is usually easy. It may be known that the disease exists at the time in animals. The peculiar coincidence of eruption in the mouth and extremities, sparing the rest of the body, is unlike any other eruptive disease. Thus, the mycoses of the mouth are unattended with affection of the feet, and eczematous and other eruptions of the feet are unassociated with eruptions in the mouth.

The **prognosis** is favorable. The disease runs a mild course, and terminates, as a rule, in from five to eight days. Extensive affection of the hands, with the difficulty of proper protection, may extend the disease to several weeks. Fatal cases have been reported in very delicate children.

Treatment.—Stomatitis is best treated with weak solutions of borax as mouth-washes. Erosions and ulcers should be cauterized with the nitrate of silver, which not only protects an abraded surface from irritating contact, but also by its antimycotic properties directly addresses itself to the cause of the disease. The superficial lesions of the extremities may be best treated by lead washes, diachylon ointment, light bandages, etc. The fever and general distress of infection may call for mild or repeated doses of phenacetin, chloral, or Dover's powder.

GENERAL SYMPTOMATOLOGY OF DISEASES OF THE NERVOUS SYSTEM.

By HORATIO C. WOOD.

THE symptoms of disease of the nervous system are due to disturbance of the functions either of the nerve-centres or of the peripheral nerves, and are therefore best studied in outline under the headings of Motion, voluntary and reflex; Co-ordination; Sensation; Vaso-motor and Trophic Alterations; and Disturbance of Intellection, including memory, speech, and emotion.

MOTION.

PARALYSIS, or true loss of motor power, must be distinguished from the loss of motion due to local disease and to arrest of function of the muscles or of the joints by pain on movement. This pseudo-paralysis can usually be recognized by the fact that passive motion and local pressure give pain. It must be remembered, however, that when contractures exist or when peripheral nerves are diseased true paralysis may exist, although passive movements are painful.

Paralysis may be complete or incomplete. When it affects the whole body below the head it is spoken of as a *General Paralysis.* A general paralysis can never be absolutely complete, since the subject must die from loss of power in the respiratory muscles before such condition is reached. *Hemiplegia,* strictly speaking, is a paralysis of one lateral half of the body, but the term is universally used not only when one-half of the face, arm, and leg are paralyzed, but also when only the arm and leg of one side are affected. It is indeed very rare for the trunkal muscles to participate in a hemiplegia. A spinal hemiplegia is conceivable, but in fact hemiplegia is almost universally of brain origin.

Paraplegia is paralysis of the lower transverse half of the body: with the rarest exceptions it is spinal. *Monoplegia.* paralysis of one part, may be *facial, brachial,* or *crural.* It may be due to lesion of the brain, of the spinal cord, or of the nerve-trunk. A cerebral lesion causing monoplegia is almost always cortical, and a spinal lesion is almost invariably situated in the ganglionic cells in the anterior cornua.

A *Local Paralysis—i. e.* a palsy of a single muscle or muscle-group—is produced by lesions situated like those of monoplegia, but less extensive. A *Multiple Paralysis* is a paralysis of more or less scattered groups of muscles not directly connected either functionally or anatomically with one another, and may be looked upon as an association of local palsies. It is usually due to

disease of various groups of spinal ganglion-cells, but may be peripheral, and in rare cases is the outcome of multiple cortical brain lesions. In paralysis of the face the mouth is always drawn toward the opposite side, unless contractures in the paralyzed muscles have taken place, when the mouth may be drawn toward the paralyzed side. In paralysis of half of the tongue the tip in motion turns toward the paralyzed side. In the examination of the paralytic it is customary to note the exact power of grasp by means of the dynamometer: a pulley and weight apparatus may be used for the upper arm and leg, but in practice a sufficiently accurate judgment may be made by noting the extent of forced movements, the endurance in walking or in standing on one leg, the ability to get out of a chair, etc.

Convulsions.—Three types of convulsions are recognized: the *epileptiform* or cerebral, in which consciousness is completely lost; the *hysterical*, in which consciousness is disturbed; and the *tetanic* or spinal, in which consciousness is normal and reflex activity grossly exaggerated. In nature these varieties of convulsions grade imperceptibly one into the other. A detailed discussion of convulsions will be found in various articles, especially in those on epilepsy and hysteria.

Automatic Movements.—The condition sometimes seen in epilepsy and in various abnormal states, in which a series of seemingly voluntary acts are performed without clear consciousness, is spoken of as automatism. An automatic act often involves an elaborate series of movements, such as those that occur in bowing, getting out of a chair, and the like. The *chorea major* of some German writers represents a form of automatism, and has no relation with true chorea or choreic movements.[1]

Reflexes.—For the performance of a reflex action an arc composed of afferent nerve, motor ganglion-cell, efferent nerve, and muscle must be complete. Disturbances of reflex activity must be due to disturbances of this arc, in which are of course included such portions of the nerve-fibres as are in the nerve-roots and spinal cord itself.

The *superficial reflexes* are excited by irritations of the skin and mucous membrane, either by tickling, pricking, pinching, or gently scratching the surface, or by means of a dry electric brush. As the superficial reflexes are not constant in the normal individual, the absence of a skin reflex is of uncertain diagnostic import, whilst the presence of the reflex shows the integrity of the nerve-arc implicated. The most important of the superficial reflexes are: the *plantar reflex*, contraction of leg, evoked by tickling the sole of the foot—reflex arc involving the lower end of the cord; the *gluteal reflex*, consisting of contractures of the gluteal muscles produced by stimulating the skin of the buttocks—arc, through the fourth and fifth lumbar nerves; the *cremaster reflex*, causing the drawing up of the testicle when the skin of the inner side of the thigh is stimulated—arc, the first and second pair of lumbar nerves and their spinal centres; the *abdominal reflex*, causing contractions of the abdominal

[1] The confusion is made still worse by the fact that some Continental writers speak of very bad cases of St. Vitus's dance as *chorea magna*.

muscles, chiefly the rectus, when the skin of the sides of the abdomen is stroked from the ribs downward—arc, from the eighth to the twelfth dorsal nerves; the *epigastric reflex*, causing a dimpling of the epigastrium on the stimulation of the same side of the chest in the sixth and fifth intercostal spaces, and sometimes even in the fourth—arc, from the fourth to the seventh pair of dorsal nerves; the *erector-spinal reflex*, causing contraction of the erector-spinæ muscles when the skin along their edges is stimulated—arc, in the dorsal region of the spinal cord; the *scapular reflex*, causing contraction of some or nearly all of the scapular muscles on superficial irritation of the scapular region—arc, the upper two or three dorsal and lower two or three cervical nerves; the *palmar reflex*, producing contraction of the flexors of the fingers on tickling the palm of the hand—arc, through the cervical enlargement of the cord; *cranial reflexes*, such as contractions of the palatal muscles by irritation of the fauces, sneezing by irritation of the nasal mucous membrane, cough by irritation of the laryngeal mucous membrane, closing of the eyes by irritation of the conjunctiva, movements of the iris by light.

Of the *deep reflexes*—that is, of those connected with such deep-seated tissues as tendons and bones—the most important are elicited by striking the patella tendon (*Westphal's symptom, patella reflex, knee-jerk*) or by flexing the foot forcibly, so as to stretch the Achilles tendon (*ankle clonus*). In some cases tapping of the biceps or flexor tendons in the arm will produce contractions of the muscles; a jaw or *chin reflex* is obtained by allowing the jaw to hang passively or by gently supporting it with one hand whilst with the other the blow is struck on the chin with a hammer in a downward direction. Ankle clonus[1] is never (elbow-, wrist-, and jaw-jerks rarely) present in normal individuals. In testing the knee-jerk the bared leg is so supported that the foot swings free from the floor, and the tendon above or below the patella is struck with the edge of the hand, with the fingers, or with a small hammer having an elastic steel handle and an India-rubber head. Any voluntary movement, such as clinching the hands at the time of the delivery of the blow, increases (technically, "reinforces") the contraction. The knee-jerk is probably absent in about 2 per cent. of normal individuals.

Paradoxical Contractions are contractions which in certain diseased conditions are produced by suddenly relaxing the muscle, as may happen to the anterior muscles of the leg when the foot is forcibly flexed.

Spasms—*i. e.* involuntary, not permanent, contractions of muscles—may be clonic, that is, of brief duration with intervals of relaxation; may be tonic, that is, prolonged without intervals of relaxation. The permanent shortening or contraction of the muscles is spoken of as a *contracture*. This contracture may be due to disease of the muscle itself or the nerve-centres to which it is tributary, or may be the outcome of a lack of power in the antagonistic muscles.

A **Tremor** is a to-and-fro, vibratile movement which is produced by more

[1] A clonus is a to-and-fro vibratory movement, and in cases with highly exaggerated reflexes can sometimes be produced in other joints than the ankle.

or less rhythmical, successive contractions of antagonistic muscles. It does not in any way simulate voluntary movements. Tremors are of two kinds: tremors which occur whether the part be at rest or in motion; and tremors (*intention tremors*) which occur only upon movement of the part affected or of some other portion of the nervous system. Intention tremors are almost invariably the outcome of a multiple cerebro-spinal sclerosis. Persistent tremors may be due to old age, to alcohol, tobacco, or other poisons, to general paralysis, or to paralysis agitans. In some cases, especially in mercurial poisoning, the toxic tremor may simulate an intention tremor. Senile tremors may also sometimes cease during absolute repose.

Choreic Movements may be defined to be irregular movements produced by independent contractions of single or associated groups of muscles, not vibratory in character, and more or less simulating purposive movements, but never forming a complicated series of apparently purposive actions. They may vary in intensity from the slightest irregular movements of the fingers or toes, or even a mere condition of excessive muscular activity resembling restlessness, up to the most severe and violent motions. They may be confined to a single group or to associated groups of muscles (*local chorea*), or may affect the entire muscular system (*general chorea*). When the whole body is affected the muscular contractions do not take place regularly or consentaneously, but momentarily, here and there. In some cases they are under the control of the will for a short period of time, but always assert themselves in a few minutes. The choreic movement is usually irregular, but it may be rhythmical. *Rhythmical choreas* more or less closely resemble tremors, differing chiefly in that the movements are much slower and more extensive.

The term "chorea" has been frequently used in literature as synonymous with choreic movements, and has also been applied to many diseases of an entirely diverse nature. Lesions of any of the ganglionic cells connected with the cerebral or pyramidal tract—that is, of the tract commencing in the brain cortex and ending in the motor cells of the anterior cornua of the spinal cord —may produce choreic movements, so that the choreic movement is not more uniform in its significance than is paralysis.

Generalized choreic movements (chorea of many authors) may be due to St. Vitus's dance; to reflex irritation; to organic disease of the nerve-centres, including in this cases of chorea in the insane; to pregnancy; to Huntingdon's disease; to changes in the nervous centres occurring in old age; to hysteria.

Local choreas have little or no relation with general chorea. Some local choreas are probably reflex, as in those cases in which violent local chorea develops briskly in the course of an acute internal inflammation, such as pleurisy or pneumonia. In other local choreas (*habit choreas*) the spasmodic movements have their origin during childhood in a frequently repeated purposive act which has grown in a neurotic temperament into a fixed habit of the nervous system, no longer under the control of the person. A brow may be lifted at intervals, a shoulder shrugged, an eye winked, a jaw dragged forward, a trick of gesture incessantly repeated, even a cough or a sniffle perpetually indulged in.

The habit chorea has a tendency not only to become more and more uncontrollable with years, but also to increase in its range. It is probable also that, in the beginning, some of these protracted habit choreas are not purely voluntary movements, but are, at least in part, due to a functional disturbance of the affected nerve-centres.

Co-ordination.

The function of co-ordination—*i. e.* the mutual action or reaction of muscles that completed movements may be performed—may be disturbed in the whole organism or in the arms or legs separately. When loss of co-ordination is slight some care is necessary to detect it. If a normal individual be placed in a strictly erect position, with the heels and toes of the two feet closely approximated, a certain amount of swaying of the body occurs, especially if the eyes be shut. If, however, there be loss of co-ordinating power, this swaying is greatly augmented. This so-called "position test" becomes more severe and difficult if the patient be required to stand on one foot. In a doubtful case the patient should be required to stand alternately on each foot, to walk backward, and to attempt to turn suddenly. Any marked awkwardness in these actions should give rise to suspicion.

In making the various tests the practitioner must beware of mistaking for true loss of co-ordination the inability that arises from muscular weakness, from muscular stiffness, or from the vertigo of cerebral disease. Especially is titubation (see Brain Diseases) not to be confounded with the loss of co-ordination.

The co-ordination in the arms is tested by the power of executing delicate movements. Thus, the patient may be told to close the eyes, clench the hands with extension of the index finger, extend the arms widely, and rapidly bring the index fingers together. If co-ordination be imperfect, the points of the fingers will not come in contact.

Sensation.

In the study of sensation it is necessary to distinguish algesia, or the power of feeling pain, from sensibility. Sensibility itself must also be redivided into the sense of touch, or the power of recognizing contacts; electrical sensibility, or the power of recognizing electrical currents; thermic sensibility, or the power of recognizing the temperature of bodies; pressure sense, or the power of recognizing weights; and the muscular sense, or the power of estimating muscular movements. In all testing of sensibility the doctor has to depend upon the statements of the patients, and care is sometimes necessary to avoid being misled. When great accuracy is required the sense of touch may be tested by means of the æsthesiometer. This consists of a pair of ordinary compasses with blunted points, furnished with a graduated scale, or of a pair of points, one of which slides upon a bar so that the distance between the points when separated is known. On the surface of the body these points are felt as two points or as a single point according as they are more or less widely

separated and as the skin is more or less sensitive. The sensibility varies greatly in different parts of the skin and also on the same portion of the skin in different individuals. Any wide deviation from the following scale may, however, be regarded as pathological: the top of the tongue, 1.18 mm.; the end of the fingers, 2.25 mm.; the side of the first phalanx, 16 mm.; the back of the hand, 3.1 mm.; the upper arm and thigh, 3.7 mm. The smallest required distance is oftener less in the transverse than in the longitudinal direction of the limbs. In practice it will usually be found better to compare the affected part with the opposite side of the body, rather than with any theoretic formula. Care should always be taken to apply the points simultaneously and with equal force.

The sense of pressure is tested by laying the hand, foot, etc. upon a firm, hard surface, like that of a table, and placing graduated weights upon it. Several forms of apparatus have been devised. A very convenient method is to more or less partially fill a series of ordinary shot-gun cartridge-shells with shot, so as to form a regular series of weights which resemble one another exactly to the eye.

The muscular sense may be tested in the arms by testing the power of the patient for recognizing the amounts of various weights when lifted.

Thermic sensibility is tested by the alternate application of hot and cooler bodies. More or less complicated instruments have been constructed under the name of *thermo-œsthesiometers*, but vials of water of different temperatures are sufficient for practical purposes. The temperature-range of most accurate sensation lies between 27° and 30° C., then between 33° and 39° C., and lastly between 14° and 27° C. The variations above or below these limits produce simply sensations of pain. According to the experiments of Nothnagel, the smallest perceptible differences of temperature are the following: on the breast, 0.4° C.; on the back, 0.9° C.; on the back of the hand, 0.3° C.; palm of the hand, 0.4° C.; arm, 0.2° C.; back of the foot, 0.4° C.; lower extremities, from 0.5° C. to 0.6° C.; the cheek, 0.4° C. to 0.2° C.; the temples, 0.4° C. to 0.3° C. In practice few normal individuals will recognize, I believe, differences of temperature so small as those here mentioned.

The results of vaso-motor and trophic alterations are so evident to the senses that no discussion of them is required here; whilst the difficulties that surround the apprehension of symptoms due to disturbance of intellection are so great as to require elaborate discussion in the article upon Mental Diseases.

MENTAL DISEASES.

By HORATIO C. WOOD.

General Considerations.

At least in the United States, alienists have long been so set apart from other physicians that they seem hardly to form an integral part of the profession, whilst to a large proportion of the practitioners of medicine the subject of insanity is, as it were, a closed book, unopened in the medical schools, unstudied in the after-years. Nevertheless, in the great majority of cases the general practitioner alone has opportunities to study the beginning of mental aberration, and too often his failure to apprehend works ruin to the patient. Within the limits assigned in the present volume it is practically impossible to write a treatise on insanity which shall meet the needs of the specialist, but it does seem to me possible to make such a statement of the general principles and the important clinical facts of alienism as shall serve to the student or practitioner of medicine as a general guide, and as a foundation upon which can be built, if wanted, more detailed knowledge. More than this, I believe that the great danger of all specialism is lack of breadth of view; and possibly the fact that the writer of this article has worked for fifteen years in general clinical medicine in the wards of large hospitals, and has had large experience as a general medical practitioner, may give a flavor to the writing different from that of the work of the pure specialist. Under the circumstances it has seemed essential to devote proportionately more space to the general consideration of the subject than would be allotted in a treatise on insanity.

For the purpose of studying the symptoms of mental disorder the human intellectual faculties may be separated into the will, the intellectual faculties proper, such as reason, imagination, etc., and the emotions, such as fear, anger, etc.

Disorder of one mental faculty is almost invariably accompanied by a greater or less degree of disturbance of the other mental faculties, but, *a priori*, there seems to be no reason why one faculty of the mind should not suffer alone, and cases are said to occur in practice in which a single faculty appears to be under the influence of disease when no other evidences of mental disorder can be detected.

The human will acts chiefly upon the lower intellectual and emotional brain-functions as a repressive force. It inhibits or puts aside this thought or that distraction or this emotion, rather than brings forward another thought or emotion. We cannot will ourselves into a passion, though we can by a direct effort of the will inhibit or repress a rising anger. If we desire to produce a fit of anger, we do it by bringing before the mind thoughts which act as stimu-

lants to the desired emotion: the almost unconscious recognition of this fact has led to the expression "working one's self into a passion." As is usually the case in disorders of inhibitory nerve-function, affections of the will are most plainly and frequently manifested by weakness or failure of power.

It is true that the excessive obstinacy and self-assertion so often seen in insanity at first sight appear to indicate abnormal exaltation of the will, but these extravagances of thought and action may be due to the overpowering influence of some emotion or some idea which so dominates the will as to govern entirely the actions of the individual. The obstinacy and self-assertion are, under these circumstances, really the outcomes of a weakened will rather than of an overpowering egoism, the person being obstinate or aggressive because his will is enslaved by a lower intellectual or emotional nerve-centre. Thus, in melancholia inflexible obstinacy may result from the absolute despotism of an overwhelming sorrow. In hysteria the will is probably always abnormally feeble, but the persistence and apparent wilfulness of hysterical subjects are proverbial.

Weakness of the will is produced by various organic brain-diseases which lower the nutritive tone of the cerebral cortex. It is caused very frequently by chronic poisonings, being one of the most pronounced symptoms of alcoholism and of opiumism. Under these circumstances the subject may show an extraordinary determination and persistency when dominated by his appetite, and yet he is really most infirm of purpose, entirely unable to decide upon a course of action in regard to ordinary matters or to carry out his decision when reached. He is liable to be inordinately influenced by his associates and by his environs, cannot resist entreaty and temptation, and so becomes more and more the sport of his desires and of external influences.

Acute illness, starvation, hardships, age, chronic diseases, any influence which lowers the nutrition of the higher nerve-centres, may produce weakness of the will. So varied are the causes of *abulia* (or abnormal weakness of the will) that the symptoms have no further diagnostic import than to show a serious functional or structural alteration of the cerebral cortex.

Exaggeration of the will-power is known as *hyperbulia*, and reveals itself in some forms of mania and cerebral cortical excitement.

The emotional nature may be by disease depressed, exalted, or perverted: the alteration often affects persistently a single emotion or a single class of emotions, or it may attack successively, at shorter or longer intervals, emotions that are antagonistic. Thus, a subject may be in a continual state of joy or of emotional depression, or he may rapidly or slowly pass from one state of emotional excitement to another, now carried away by anger, now prostrated by fear, now soaring with joy, now overwhelmed by sadness.

In advanced stages of cerebral disease a condition of true emotional enfeeblement or lethargy may be present, so that external circumstances which naturally affect most vividly this or that emotion fail to produce any response. This mental condition ought logically to be known as emotional depression. It is to be clearly distinguished from excitement or over-activity of the depres-

sive emotions, such as sorrow, and their congeners. Viewed in this way, the melancholic person is not in a condition of emotional depression, but in one of emotional excitement—*i. e.* of excitement of the depressive emotions. Melancholia is, it is true, frequently associated with depression of the nervous system, but this is not always the case, and the victim of melancholia agitata may be in a condition of general nervous erythrism as pronounced as that which affects the maniac with widely-expansive delusions. On the other hand, high hopes and abundant joy are in advanced general paralysis closely linked with the most profound evidences of failing nerve-power. If melancholia is to be considered a state of lowered emotional activity, whilst joy and anger are the outcomes of emotional excitement, it logically follows that the antagonistic emotions are different manifestations of one cerebral function, joy being the result of excessive stimulation, sorrow of excessive depression, of the same brain-cells—a conclusion which I think few persons would be ready to accept as correct.

The relations between the diverse emotions of which I have just spoken are of some importance as explaining the fact that in various mental affections mania and melancholia, or opposite emotional states, may follow each other, and even appear to be produced by the same brain lesion. Thus, in paretic dementia the persistent hyperæmia of the brain-cortex may cause throughout the attack intense sadness, or an emotional depression may suddenly replace the expansive happiness usual to the affection. To account for such a change it is only necessary to suppose that there is a shifting of the hyperæmia and the excitement from one portion of the brain to another.

The lethargy due to absolute loss of mental power spoken of on page 552 really is closely associated in its origin and nature with stupor, differing, however, from that condition in that consciousness is not lost: both stupor and emotional lethargy may in the insane be closely simulated. An insane patient may lie in bed absolutely still and inert, with closed eyes, giving no response to the loudest questioning and making only a feeble and slow resistance to personal violence; or when, with head bent forward, joints flexed, and face frozen into an immobile apathy, he sits motionless in his chair, he may seem to be lost in unconsciousness, but none the less may he have knowledge of his surroundings and of his sorrows. The pseudo-lethargy may be the direct result of an intense emotion or of delusion, and not be consciously assumed; but not rarely it is put on for a definite end, and maintained with a tenacity of purpose which defies detection even during the intoxication caused by ether or by alcohol. The occasional revelations made by patients after they recover their reason show that a delusion may act very directly in the production of an assumed stupor. A man believes that he has received commands from the Almighty to isolate himself from all communion with his fellows, and in maintaining the assumed stupor battles for his eternal salvation; or the lunatic conceives that his attendants are conspiring against him, and will do him great evil if once they are assured he is alive. In some cases the pseudo-lethargy is the result of an overwhelming emotion produced by the delusion.

The man about to be devoured by foul beasts or by the flames of hell is dumb through fear, or, as the German alienists say, is thunderstruck. Occasionally the insane sleeper is convinced that he is dead, and by this delusion his will is so far paralyzed that it is unable to act, and the man really cannot move, although the lower nervo-muscular apparatus is intact.

The intellectual functions proper may suffer from actual exaltation, giving rise to increase of power; from an exaltation which is so unbalanced as to produce a derangement of action; from a real depression or loss of power.

Absolute increase of mental power is a rare condition, and is never present in any *advanced stage* of disease. The subject of a pronounced mental exaltation has a passion for intellectual labor, accompanied by a corresponding power of accomplishment. It is no longer an effort to fix the attention upon an intricate subject for successive hours. The sense of fatigue is lost, and the brain works on without pain, the quality as well as the quantity of the result being beyond that which the individual in his normal condition can produce. This state of mental exhilaration sometimes comes on during protracted mental labor. It is probably always associated with hyperæmia of the brain-cortex, and is usually accompanied by pronounced insomnia. It is a very dangerous condition, and should be the signal for immediate cessation of mental effort and for medical treatment. It is sometimes developed without obvious cause as a prodrome of severe mental disease. Thus, I have seen it precede a fatal outbreak of acute phrenitis, and it may usher in paretic dementia.

If one or more of the mental functions are excited entirely beyond the control of the will and judgment becomes impossible, a mental condition is produced which in its most severe acute form is sometimes spoken of as delirium, and in its milder or more chronic forms as insanity.

Failure of the mental powers is a very common result of functional and organic brain disease. When complete it constitutes the condition known as dementia.

It is often of vital importance to recognize the dawnings of mental failure. The failure usually manifests itself first in loss of memory. This will be sufficiently discussed later. (See page 666.) Next to memory in the order of implication, and sometimes even preceding it, is the power of fixing the attention. The mind of man naturally wanders from subject to subject. A continuous thoughtful application depends upon the exertion of the inhibitive power of the will in repressing distracting thoughts and shutting out new perceptions. The power of persistent attention to one subject is to a great extent acquired by training. Its exercise is a large feature in all severe intellectual work. Consequently, when the brain is exhausted not only do the reasoning faculties labor with difficulty, but increased effort is required from the weakened will to maintain the necessary fixity of attention. Mental toil becomes, therefore, most irksome, as is recognized by the common expression of sufferers that "work is becoming more and more of an effort." Failure of memory and failure of the power of fixing the attention have no particular diagnostic

import. When they coexist and are associated with any other evidences of mental derangement they indicate a serious disease of the brain itself. The loss of the power of fixing the attention, however, when it exists alone, usually depends upon simple cerebral asthenia—a condition in which there may also be some loss of memory.

A symptom which may depend upon either mental excitement or loss of mental power is *incoherence*. An incoherence due to a heightened but irregular cerebral activity results from the excessive rapidity of the intellectual acts, as well as from their lack of connected sequences. Before one idea is fully translated into words another rushes into expression, and a hopeless confusion of talk results. The ideas tumble out as it were over one another. Incoherence from lack of mental power, on the other hand, arises either from the inability to complete the mental act or from the lack of the power of translating it into suitable words. In typical cases there is little difficulty in distinguishing between these varieties, which it is allowable to call respectively *active* and *passive* incoherence. The rapid utterances of the raving maniac usually show most plainly that his mind is pouring out broken hints of an infinite series of jostling ideas; whilst the slow, confused, disconnected, hesitating words of the dement no less unmistakably portray his inability fully to conceive an idea and embody it in words. There are, however, many cases of disease in which mental excitement coexists with failing power, and in which, therefore, the incoherence is of mixed type.

Besides the symptoms of mental aberration connected with the intellectual and emotional faculties already spoken of, there are certain specific manifestations—chiefly, although not altogether, dependent upon disorder of the purely intellectual faculties—which need careful consideration. Closely connected—indeed, in large part due to disorder of the perceptive faculties—are hallucinations and illusions.

An *hallucination* is the perception by any of the senses of an object which has no existence. It is the conscious recognition of a sensation of sight, hearing, feeling, taste, or smell which is not due to any impulse received by the perceptive apparatus from without, but arises within the perceptive apparatus itself: in other words, an hallucination is a subjective sensation, which assumes the definite attributes of an objective sensation. It is commonly simple—*i. e.* connected with a single sense. Thus, the vision is usually seen, not seen and felt. The false voice is heard, the mysterious presence is felt, but the presence and the voice usually do not coexist. In the order of their frequency of implication the senses may be enumerated as follows: sight, hearing, touch, smell, taste. The particular characters of the perceived object vary indefinitely, and involve the whole range of perceptions. Every variety of color and form, of sound and odor, of feeling and taste, may be perceived.

In some cases, as in mirage, a *false perception* may amount almost to an hallucination; that is, an impulse from without may give rise to such a distorted, misleading conscious perception that the person really sees or feels or hears that which has no existence. A distorted sensation—or, in other words,

the perception of an object in characters which it does not possess—is frequently spoken of as an *illusion*. In nature there is no sharp line between illusions and slight distortions of the perception of objects, or between illusions and hallucinations. An hallucination may be caused by an external stimulus so slight that it cannot be discovered, but it may arise entirely from within the nervous system.

An hallucination has no definite diagnostic import. It may come from exhaustion of the nervous system, especially when there is at the same time an intense desire. Thus, the wife, worn out with long watching and grief, sees in obedience to her yearnings the living form of her dead husband. The monk, exhausted by long prayer and fasting, if consumed by ardent devotion, is comforted by saints or angels, or, if he be tormented by suppressed sexual desires, is haunted by troops of tempting devils or voluptuous sirens. The person perishing with thirst sees or hears cool springs, babbling brooks, or plashing fountains; gorgeous feasts float before the vision of the starving; and the shipwrecked mariner is tantalized by rescuing barks.

Hallucinations may be the result of the immediate action of a poison, as in the beatific visions of the hasheesh-eater, or may be the outcome of the peculiar nervous state which follows the abuse of narcotic stimuli, as in delirium tremens. Conditions of the nervous centres at present inexplicable may call hallucinations into being, as in hysteria. More rarely the hallucination is the result of an organic brain disease, when its nature is almost invariably pointed out by coexisting symptoms, such as epileptic paroxysms or local palsy. The structural alteration in such cases is commonly in the nerve-tract especially connected with the affected sense.

An hallucination does not necessarily depend upon or prove the existence of intellectual unsoundness. It is, however, very apt to be associated with such unsoundness, because the condition of the sensory brain tract which produces it is apt to accompany a similar condition of the higher or intellectual centres. Moreover, it often affords us a means of testing the condition of the brain-centres. If the judgment fails to correct the testimony of the disordered sense by that derived from other senses, the subject is of unsound mind. When, for example, the individual believes that the vision that he sees or the voice that he hears really exists, then is his judgment dethroned. It will be readily seen that in such a case it is not the seeing of the vision, but the loss of the power of weighing evidence, that is the proof of the intellectual degradation. As will become very apparent during the discussion of delusions, the hallucination in the case just imagined has given rise to a delusion.

An hallucination which is not very vivid is sometimes spoken of as a *pseudo-hallucination*. Certain authorities attempt to make a sharp separation between pseudo and true hallucinations. It is affirmed that the pseudo-hallucination always remains to the individual who has it a subjective phenomenon, whilst the genuine hallucination appears to the individual as reality itself. It is clear that the pseudo-hallucination of some writers is simply an hallucination which is recognized by the intellect as a subjective phenomenon, and therefore does

not give rise to an insane delusion. The true hallucination of such writers is a combination of an hallucination with an insane delusion. In nature there seems to be every possible gradation between the faintest delusion or hallucination and the illusion or hallucination which is completely believed in by the individual and most completely dominates him.

The word *delusion* may be defined to be a false belief, but as it is used by alienists the term means something more than this. By Spitzka the insane delusion is said to be "a faulty belief out of which the subject cannot be reasoned by adequate methods for the time being." The objection to this definition is that there are many faulty or false beliefs held by perfectly sane persons out of which such persons cannot be reasoned, but which are not insane delusions. Thus, either the Christian or the Mussulman, under such definition, is the victim of an insane delusion. To meet the necessities of the case the definition should be modified so as to read, "A faulty belief concerning a subject capable of physical demonstration, out of which the person cannot be reasoned by adequate methods for the time being."

The parallelism between a delusion and an hallucination is very close. A delusion is a false belief; an hallucination is a false perception. The delusion becomes an insane one only when the false belief cannot be dissipated by absolute proof of its incorrectness. The hallucination becomes an insane one only when the false perception cannot be corrected by the judgment through the other senses. In either case the essence of the insane mental state is loss of power to receive and weigh adequate evidence.

Thus, John Smith hears voices where there are none: he is insane only when he is unable to correct the evidence received through the sense of hearing by that received through the senses of sight and feeling. If he persistently believes that persons speak to him, although he cannot see or touch them, his judgment is in abeyance. On the other hand, John Jones believes that a certain barn exists upon a certain field where there is no barn. Under these circumstances he has a delusion, a belief which has grown up in his mind from some cause unknown. Now, if, when taken to the field, he is incapable of receiving the evidence of his senses and persists in his belief that the barn is there, he is insane; but if he receives the evidence of his senses and perceives that the barn does not exist, he is not insane. In case of insane hallucinations or delusions the truth or falsity of the vision or of the belief is not essential. The essential thing is the condition of the mind of the individual —a condition which prevents it from receiving evidence. Hence an insane belief may be true although insanely held.

In the supposititious case given above assuredly the mental state of the individual is in no wise dependent upon the absence of the barn, although such absence renders a test of the subject's mental condition possible. The distinction just drawn may seem unimportant and so trite as to be unworthy of discussion, but the failure to understand it has been one cause, in my experience, of the inability on the part of learned lawyers to comprehend the subject of insanity.

Not long ago, after due process of law, an insane man by the name of Taylor was hung in Philadelphia for the unprovoked murder of a prison-warden. It was in evidence that the man believed that all the attendants of the prison were Catholics, and were "down on" him because he was a Protestant, and were destroying him. The prosecuting attorney asked, "Supposing it were proved that the prison attendants were Catholics, would it not have to be acknowledged that the man's belief was correct, and that he was not insane?" Apparently neither lawyer nor judge could be made to understand that the falsity or the truth of the prisoner's belief in the Catholicism of the attendants had little to do with the question of his insanity. It was proved that he had other delusions of persecution, and his having adopted a belief in regard to the Catholicism of his attendants which was in accord with such delusions, without any evidence of their alleged Catholicism, and having reasoned insanely upon the subject and acted in accordance with conclusions so reached, showed that his action rested upon mental unsoundness. Surely the "*Because I am a Protestant*, therefore they are destroying me," ought to have made the mental condition of the prisoner clear. In the language of Spitzka, "Repeatedly does it occur in the alienist's experience that the facts of a case and the delusion *happen* to correspond." This is well illustrated in a case reported by him. An artist's model asserted that he was the finest-built man in the United States. He really had a magnificent figure, but his announcement was, notwithstanding, that of a paretic dement, for inquiry elicited the statement that the "girls looked at him because he had a peculiar expression in his eyes which they fancied," and he revealed other unmistakable evidence of general paralysis.

An insane belief or delusion may rest upon an hallucination, may be built upon a foundation of disordered sensation, may spring from the most trivial circumstances, or may, so far as can be judged, be self-engendered in the mind. Thus, the voice that is heard as an hallucination gives rise to the delusion of an ever-present persecutor; a persistent distress in the abdomen to a delusion of pregnancy or that the bowels are dropping out, etc. The following case from my notebook illustrates very forcibly the curious way in which a delusion develops in the mind without the slightest foundation in verity: A man after a malarial fever began to have suspicions in regard to the chastity of his wife. For a time he kept these to himself, but finally he accused her of infidelity. After this had continued for some weeks he presented himself with his wife at my clinic, saying to me, "I think my wife goes with other men: she thinks I am crazy. I am uncertain whether she or I am right." On being questioned, he stated that he first noticed her looking behind her, as though she were looking for some one, when they walked together; that he afterward saw a handkerchief lying on the bureau in her room, just as she would have left it if she had been flirting with some one out of the window, and that when he saw a chair by the window of her room and a man at the corner of the street he was convinced that his suspicions were correct: in this he was corroborated by finding three dollars in a trunk, which he believed his

wife had received "for evil courses," although she had declared that he himself had given it to her. He further stated that he watched her eyes. In a very eager, tremulous manner he said, "I got a lamp, and when I found her eyes were dark beneath, I told her there was something wrong with her, and then she began to think there was something wrong with me. I firmly believed she was going with other men." The man had an inherited tendency toward insanity, and had lost much sleep. When his whole case was thoroughly explained to him, he said that he "now understood it, and was glad to hear it, and that it gave him power to brace himself against the notion," ending with the assertion that he believed that "he had a good woman." In reply to a question, he said, "I do not think there is danger of my hurting my wife, but these things come on me so that I cannot control myself at times, and I am willing to go to an asylum if it is thought to be right."

The relation between the emotional state of an insane man and his delusions is very close. Expansive or happy delusions accompany emotional exaltation, while horrible or sorrowful delusions go hand in hand with depressive emotions. Thus, the melancholic woman is oppressed with the belief that she is hopelessly damned, that her husband is unfaithful, or that she is pregnant with devils; whilst the maniac, overflowing with animal spirits, is a prophet sent of God, is owner of uncounted millions, or mayhap is about to become the mother of the Messiah. The emotional state and the delusions constantly react upon one another. Some alienists believe that the character of the delusion is directly dependent upon the dominant emotion.

The nature of delusions varies so indefinitely as to render any attempt at a thorough classification futile. There are, however, certain classes of delusions which are so frequently met with and so characteristic as to require especial study. The most important of these are—1. Expansive Delusions; 2. Hypochondriacal Delusions; 3. Delusions of Persecution.

Expansive Delusions usually concern the personality of the individual who has them, either as to his prowess, his mental or physical attainments, his possessions, or his future prospects. The patient boasts that he is the strongest man in the world, asserts that his mental powers are immense, or that he is a king or other notability, or more commonly talks of his millions of money, his gold-mines, his farms of unlimited extent, his vast stables full of unnumbered horses of the choicest breeds, his far-reaching and gigantic business schemes, etc. This condition constitutes the *délire de grandeur*, and, whilst in the majority of cases it depends upon the existence of general paralysis, it may be present in many forms of mental disease. I have seen it very pronounced in cerebral syphilis, and have watched the millions of dollars possessed by the subject shrink to thousands, and the thousands to hundreds, as the brain lesions grew less under the administration of mercury. Then even the hundreds disappeared, and his own poverty was confessed; but the assertion still remained that "his uncle was worth a million," until at last this too vanished in the recognition of the desolate truth.

Hypochondriacal Delusions relate to disease of the person of the patient,

and are usually, but not always, associated with a depressive emotional state. They sometimes rest upon a substratum of ill-feeling, or even of actual disease, in the part alleged to be hopelessly affected. They are often obviously absurd, as that the legs are made of glass. Of all forms of delusion, this is the one in which the gradations between the sane and the insane belief are most subtile. Every step can be found between the slightest exaggeration of symptoms and the hypochondriacal foundationless belief. Unless a hypochondriacal delusion is upon its face absurd, the physician must be very careful in basing upon it an opinion that the subject of it is irresponsible, since many invalids are hypochondriacs and have exaggerated beliefs bordering closely upon delusions, but are, nevertheless, of sufficiently sound mind for the performance of the ordinary duties of life.

Delusions of Persecution are not always associated with a pronounced depressive emotional condition. They are always the source of great annoyance and distress to the subject, and are usually associated with hallucinations which I think are most apt to be connected with the sense of hearing. Very commonly obscene, reproachful, or threatening voices are heard at all times and in all places. Usually the delusion of persecution does not attach itself in the mind of its victim to one person, but to classes of people or to unseen spirits. Sometimes, however, the delusion does affix itself to one individual, as in a recent case in which a woman travelled across the continent of America to kill a doctor who she believed was placing a spell upon her. Of all the quiet classes of the insane, those who have delusions of persecution are the most dangerous. They are impelled by motives of revenge and of fear to kill those who are persecuting them. This is especially the case when the delusion attaches itself to one individual; but even voices in the air may lead to sudden violent assaults upon bystanders who are for the moment thought to be the source of the words. Moreover, the lunatic may at any time fix in his mind upon any acquaintance or notable person as the origin of his persecution and make his plans in accordance.

A very important division of delusions is into systematized and unsystematized. A *systematized delusion* is one concerning which the subject reasons, and which he defends more or less logically. Any character of delusion may be systematized. If a lunatic asserts that he is worth a million of dollars, and simply sticks to his belief when it is denied, he has an unsystematized delusion of grandeur; but if he should attempt to defend his delusion by describing how he had inherited his wealth or how he had acquired it through investments or business ventures, his delusion would be systematized. Again, a person suffering from melancholia believes that his soul is lost. If, when opposed, he simply reavows his belief and assigns no reasons for it, his delusion is unsystematized; but if he says he is lost because he has committed the unpardonable sin, quotes Scripture to show that such a sin warrants his doom, and perhaps tells why and when he sinned, his delusion is systematized.

Great diagnostic value has been attached by some recent writers to the distinction between systematized and unsystematized delusions, and much has been

predicated upon it in the classification of insanities. According to my experience, however, in nature every gradation is to be found between the most thoroughly systematized delusion and that which is most completely isolated. I have seen various cases in which it was doubtful whether the delusion should be classed as systematized or unsystematized; and, whilst I acknowledge that in typical paranoiacs the delusions are systematized and in typical general insanities they are unsystematized, I am of the opinion that in this character, as in others, the two groups of general and partial insanities pass in nature insensibly into each other.

There are certain conceptions or general ideas which may arise in the brain of a person, and to a greater or less degree dominate his actions, although the reason may not be unsettled and the falsity of the conception may be recognized by the individual whom it controls. Such a phenomenon is known as an *Imperative Conception*, and differs from a delusion in that its falsity is recognized, although the individual is powerless to withstand its influence. Closely allied to the imperative conception is the *Morbid Impulse*. Some alienists, indeed, teach that the imperative conception gives rise to the morbid impulse. In certain cases this undoubtedly happens, as when the imperative conception of personal defilement gives origin to the impulse of escaping from that which defiles; but a morbid impulse may arise without any discoverable imperative conception. Thus, I long had under my care a man in whose family insanity was distinctly hereditary, but in whom the only symptom that I could find was an impulse to assault bystanders—an impulse apparently born of no reason, although felt with such urgency as to fill the patient with a terror of himself. Once, upon returning home, I found this man sitting in my office terribly excited, and greeting me with, "Doctor, doctor, I nearly did it! I nearly did it!" It appeared that he had spent forty-eight hours without intermission in a vortex of political excitement, and suddenly the impulse to kill had come on him with such power that only by fleeing to my office was he able to save himself. The impulse to throw one's self from a precipice, caused by standing on its brink, is a familiar instance of a mild morbid impulse without an apparent foundation of an imperative conception; whilst the reasonless dread which many persons have of a snake, toad, cockroach, or other harmless creature probably depends upon an incipient imperative conception of personal defilement.

The act which results from a morbid impulse is sometimes spoken of as an *Imperative Act*. An imperative conception is viewed by some alienists as an "undeveloped delusion." It is, however, not a proof of general mental unsoundness, but in some cases finally the reason of the patient fails to recognize the untruthfulness of the imperative conception, which conception thereby becomes converted into a delusion, precisely as an hallucination may give rise to a delusion.

A very important and common imperative conception is a morbid fear. This may take almost any form, and may be simply an exaggeration of a normal feeling or may arise *de novo*. Thus, in some persons the fear of a thunderstorm is so violent as to destroy for the time being all rationality; in

others the natural dislike for filth is increased until it dominates every action of life. On the other hand, the horror of walking in an open place, which is sometimes so overwhelming, seems scarcely to be based upon any natural feeling. To many of these morbid fears names have been given by systematic writers. The fears, however, vary so in their detail that it is not possible to express them accurately and fully by any system of nomenclature. A few of these names may be cited, as representing the more characteristic forms of morbid fear. The following list, taken from Dr. Beard, portrays very well the absurdities of nomenclature:

Astraphobia, fear of lightning; Topophobia, fear of places (a generic term, with these subdivisions: Agoraphobia, fear of open places; Claustrophobia, fear of narrow, closed places); Anthrophobia, fear of man—a generic term, including fear of society; Gynæphobia, fear of woman; Monophobia, fear of being alone; Pathophobia, fear of disease—usually called hypochondriasis; Pantaphobia, fear of everything; Phobophobia, fear of being afraid; Mysophobia, fear of contamination.

As illustrating imperative conceptions a few cases from my own experience may be cited. A very strong shoemaker, past middle life, was oppressed with the idea that he could not walk unless he had some covering over his head. On a stormy day the natural cloud-canopy sufficed, and on a clear day an umbrella carried over his head gave a measure of relief, so that he was able to command his movements. He could walk in a thick wood, but, as he himself said, if ten feet of clear sky intervened between the wood and a spring, he would die of thirst before he could cross over. No other symptom of physical or mental ailment could be detected.

A lady had a dread of personal defilement. Hundreds of times daily she washed her hands, without avail; bank-notes fresh from the press were the only money she would use; a door-knob she would never touch, but would remain in the room until some one opened the door; in putting on her clothes only the inside of each piece was touched by her fingers, and this as daintily as possible. Without entering into further details, suffice it to state that her whole life was arranged in order to avoid as much as possible contact with any person or thing. On my asking her to shake hands her embarrassment was extreme: though naturally polite and feeling under some obligation to me, she was nevertheless entirely dominated by her imperative conception. Finally she said, "Dear doctor, don't ask me: you know you touch so many people."

A gentleman entirely rational, able to manage his business affairs well and to converse on all subjects, was completely ruled by imperative conceptions and morbid impulses, the connection and the independence of which are well illustrated by his case. Thus, for many years he had an impulse continually to rub his arms against his sides, and this he did incessantly until coat after coat was rubbed into holes. No morbid conception could be found underlying this or some of the other impulses which he had. Nevertheless, he did have imperative conceptions with outgrowing secondary impulses. For many months he was markedly mysophobic. Then he had the conception that he

must lay things down straight and could not do it. Most of his waking moments were at this time spent in putting down and arranging. When he placed a book on the table, over and over and over again he would lift it up, straighten it, pick it up and relay it, etc. Often at night he would be two or three hours getting away from his coat, which he was perpetually arranging upon the chair on which he had laid it. There was no delusion, and on my asking the man why he yielded to the impulse, he said, "I can resist it for a while, but after a time the same overpowering sensation comes as when I hold by breath, and I must do it. I have found that if I say very fast, 'It is straight, it is straight,' over and over again, at the same time cracking my fingers briskly by shaking my hand, the impulse often suddenly vanishes, with immediate relief."

The end of this unfortunate victim of disordered nerve-centres was very tragic. By great care and effort he had succeeded in concealing from the general public his mental weakness, and was engaged in business enterprises of large magnitude. In the course of one of these it so happened that he became involved in a lawsuit which finally necessitated his going upon the witness-stand. The newspapers of the morning of the day upon which his testimony was to have been taken announced his sudden and unaccountable suicide. Excessively sensitive and proud, when he found himself in such a position that he must reveal to the public his extraordinary peculiarity, he preferred to such exposure death by his own hands.

The relation of imperative conceptions and morbid impulses to insanity is a matter of great theoretical and practical interest. They are undoubtedly frequent in the insane, and usually careful examination of a case in which they are present will reveal distinct symptoms of alienation. They may, however, exist in persons whose intellectual actions are in other respects entirely normal, and in whom the judgment is not dominated by the conception, although the conception may cause him to perform actions which are against his judgment. To himself the sane subject of an imperative conception seems possessed by a demon whom he must obey.

The relation of morbid conceptions and impulses to legal responsibility for acts committed involves questions of great practical difficulty. The victim of the morbid impulse cannot properly urge such impulses as excuses unless the deed in question be immediately produced by them. When the act is committed because the actor is forced to do it by a morbid impulse, the actor is, of course, morally blameless; but who can tell whether the impulse was resisted to the uttermost? Moreover, the needs of society, and the ease with which such impulses could be alleged or counterfeited, very properly cause us to pause in attempting by them to excuse a criminal act. The clearest possible proof should be required that the impulse was really morbid and irresistible.

By the use of the word "mania" as a suffix numerous names have been formed which are sometimes incorrectly used as denoting the morbid impulse, although they in fact are only correctly applicable to the mental state underlying the impulse. In *pyromania* the morbid impulse is to set fire to buildings;

in *kleptomania*, to steal; in *homicidal mania*, to kill; in *suicidal mania*, to commit suicide; in *arithromania*, to be perpetually making calculations or counting in abstract numbers, or perhaps reckoning a multitude of some supposititious concrete thing.

The so-called "manias" are not, however, distinct insanities at all: most of them are formed of reasoning insanities; but a morbid impulse may arise in almost any form of insanity. Again, what seems a morbid impulse is often the result of a logical deduction from false premises by the diseased mind. Thus, the man who, not believing in a future existence, commits suicide because he is suffering from the unutterable misery of melancholia, is logical and reasonable in his suicide, and does not kill himself through any *morbid—i. e.* unreasoning—impulse. Suicidal and homicidal maniacs are simply maniacs who have a tendency to kill themselves or others.

Morbid Desires are exaggerations or perversions of natural appetites, and are chiefly seen in regard to hunger and the sexual passion. Mere depravity and wickedness may convert man into a monster: neither cannibalism nor the lowest sexual degradation is necessarily the offspring of disease. Nevertheless, disease may affect the appetite for food or for sexual congress, as it does other functions of the nervous system.

In mania, in paretic dementia, in hysteria—indeed, in almost any form of insanity with excitement and exaltation—the sexual passion may become an all-devouring, insatiable lust. In the female this condition is known as *nymphomania;* in the male, as *satyriasis*. The victim of it talks incessantly and indecently about sexual congress, makes furious love to all persons of the opposite sex, exposes the person, etc. *Erotomania* is a very frequent condition in which there is the appearance but not the reality of sexual excitement. The subject of it conceives a strong attachment for some person of the opposite sex whom perhaps he or she has never seen, and lives in an attitude of a perpetual worship. Sometimes the object is in public life, and is followed from place to place with a pertinacity and publicity which may amount to actual persecution. Even if opportunity offer, the erotomaniac makes no effort at cohabitation. Satyriasis leads to sexual excess and to rape. Erotomania is a platonic affection, which involves the higher conceptive sphere rather than the lower nerve-centres and leads to sexual abstinence.

Human character is the result of the established balance between the will, the intellectual attributes, and the emotional forces of the individual. When any of the correlated factors are altered there must be a corresponding change in character. Character is, therefore, always seriously implicated in mental affections. Not rarely changes in the intellectual or emotional nature so subtile or hidden as not to be readily perceived register themselves with astounding distinctness on the dial-plate of character. Hence alterations of character are of the weightiest diagnostic import. They may be the first evidences of a developing pure insanity, but when sudden and severe they usually point toward dementia paralytica. A primary sudden criminal outbreak in dementia paralytica is generally sexual in its direction. Thus, in a case formerly under

my care the first marked disorderly action was an attempt to rape a servant-girl. After this it was discovered that very large and foolish purchases had been made as the beginning of a grand business scheme entirely foreign to the daily occupation of the man. An estimable citizen goes to a distant city and attempts to turn a hotel into a bawdy-house; another, whilst still performing acceptably the duties of an important public office, tries to seduce, and, this failing, to rape, his own daughter.

In dementia paralytica, as in the pure insanities, the moral degradation may, however, run in other than sexual channels. The temperate man suddenly becomes addicted to drink; the honest man all at once appropriates large sums of money, which, it may be, he spends in licentious revels; he who has always been exceptionally self-controlled becomes violently passionate; the amiable, loving husband and father changes into a household demon. Careful examination under these circumstances will usually detect other symptoms of the coming or already-present insanity.

Before entering upon the discussion of the classifications of insanity the question how much of abnormal mental action is compatible with sanity seems naturally to present itself. Its answer involves the definition of the words *sanity* and *insanity*, and, like these definitions, probably will always be unsatisfactory. Insanity is not a definite disease, but an abnormal state, varying indefinitely in its intensity, separated by no tangible line from sanity, arising from a number of diverse diseases, and terminating in most various ways.

Moreover, the manifestations of insanity are simply alterations, exaggerations, or perversions of the normal faculties, and therefore offer nothing that is absolutely new. Emotional depression deepens into the profoundest melancholia, emotional exaltation lifts itself into the highest mania, by a gradation as insensible as that by which the beach slopes into the deep ocean or the mountain rises into the air; and who shall say where the dividing-line is between the state in which the man is master of the mood and that in which the mood is master of the man? The insane impulse is but an exaggeration of that which bids a man standing on the verge of some great height to plunge headlong, or which, spreading from breast to breast, fills a mob with reckless rage or scatters it in causeless panic. Who shall say when the man could by violent effort control the impulse, and when the impulse of necessity overpowers the man? Thus it is in all forms of insanity.

For his own purposes of science, or even of treatment, the physician needs no definition of insanity, but the relations of man to man are so altered by insanity that the law must take particular notice of the subject of insanity. Even, however, for the purposes of the law insanity is not a fixed term, because it is a well-assured axiom that a man may be legally sane—*i. e.* responsible—for one class of acts, and insane—*i. e.* irresponsible—for another class of acts.

As already contended, there can be no scientific definition of insanity except that it is a state of mental aberration. Such a definition does not meet

the needs of the court-room, which demands an arbitrary although shifting line between the sane and the insane. The term insanity as used by judges and lawyers is legal rather than scientific, and the law ought clearly to define the word. It does, however, no such thing. It does not frame an authoritative definition of insanity, but through the mouths of its exponents puts forth an abundance of contradiction.

Probably as good a definition of insanity as the expert can frame to meet the clamor of lawyers is, that insanity is a condition of mental aberration sufficiently intense to overthrow the normal relations of the individual to his own thoughts and acts, so that he is no longer able to control them through the will. The difficulty of applying this definition to the individual case consists in the fact that the will does not all at once lose its grasp on the lower faculties, but that little by little these slip from under its control. Of degrees of responsibility none but the All-knowing can judge, and to say with assured correctness just when the lost control has been lost is not given to mortals. In a court of justice it becomes the expert to state as nearly as may be the exact mental condition of the prisoner, leaving to the judge the decision as to his legal responsibility—*i. e.* the relation of his mental condition to the law of the commonwealth in which the trial is held.

Insanity being a symptomatic condition, and not a disease, it is illogical to consider its different forms as distinct diseases. The best that can be done is to describe the diseases of the brain and the insanities which accompany them so far as we know such diseases, and, when our knowledge of diseases fails, to discuss forms of insanity not as diseases, but as symptom-groups.

The purposes of discussion necessitate the naming of these symptom-groups. Naming symptom-groups naturally leads to the delusion that these groups are diseases; hence melancholia, mania, etc. are constantly written about as though they were terms of equivalent force to typhoid fever or scarlatina, whereas they are simply the names of symptom-groups of the same rank as diarrhœa, paralysis, or dropsy.

This is shown by the following facts:

1st. Similar mental symptoms may be produced by various organic brain diseases; or, as Dr. Charles F. Folsom says,[1] "tumors, new growths of all kinds, exostoses, spicules or portions of depressed bone, embolisms, hæmorrhages, wounds, injuries, cysticerci, may give rise to any of the symptoms of the various psycho-neuroses and cerebro-psychoses."

2d. Almost any form of insanity may exist without demonstrable organic lesion. This is shown by the well-known fact that in a large number of autopsies upon the insane skilled observers have failed to detect alteration of brain-structure.

3d. Antagonistic forms of insanity may be produced by lesions which are, so far as we can perceive, identical, as is witnessed by the circumstance that in paretic dementia the usual expansive delusions may be replaced by a profound melancholy. Further, lesions usually accompanied by insanity may

[1] *American System of Practical Medicine*, vol. v. p. 202.

exist without mental disorder. Dr. Folsom says: "Indeed, nearly every pathological condition of the brain known in insanity—in kind, if not in extent and degree—may be found in diseased or injured brains where there has been no mental disease in consequence."

4th. The form of the insanity may change in the individual without appreciable cause and without conceivable change of disease.

5th. Almost every grade of case exists in nature, uniting by an unbroken series the various insane-symptom groups. Thus of the two most antagonistic forms of acute insanity, acute mania and acute melancholia, Bucknill and Tuke say:[1] "Between acute mania and acute melancholia no distinct line of demarcation can be drawn. The domains of the two diseases overlap so much that, in practice, cases not infrequently present themselves which may with equal propriety be referred to one or the other."

The considerations which have been brought forward show that the various forms of insanity are not entitled to be considered as distinct diseases, and that at present we cannot connect cerebral lesions and mental symptoms in their causal relations. More than this, the rapid recoveries which sometimes occur in apparently hopeless cases of insanity show that the symptoms cannot depend upon alterations of the brain-substance sufficiently gross to be detected by our present methods.

I shall narrate, as showing this, a single case, that of a lady with whom I was thrown in almost daily contact for many years: At about the age of forty-five she was taken with religious melancholia of the most pronounced character, which was accompanied by agitation, and sometimes by frenzy. This persisted for fifteen years. There had been in all this time not the slightest wavering of the mind of the woman in regard to her future life. She firmly believed that her soul was irretrievably lost. At the same time her general emotional nature had undergone a retrograde change: she had become exceedingly jealous of attentions paid to other persons, and had lost many of the peculiar traits of refinement which had been her especial characteristics. After being in an asylum for some time she recovered intellectual power sufficient to enable her to take charge nominally of her husband's house, which was really managed by her attendant, but there was no wavering in her delusion nor even any temporary abatement of her misery.

One night the attendant noticed this lady on her knees at the bedside. This was the first time in fifteen years that she had been known to kneel in prayer. The nurse, being a wise woman, did not disturb her, and there she remained all night. In the morning she joined the family, and said that she had found Christ, and that she was perfectly well and happy. Her old disposition had returned, and her peculiar jealous sensitiveness had disappeared. The woman who had been buried for fifteen years had emerged in one night without even the grave-clothes about her. This continued for one week. Then the old cloud came on her, and for days she was in the old condition; but suddenly the sunlight again broke through the clouds, and she remained well for three

[1] Phila. edition, 1874, p. 427.

or four days, to relapse, and after some hours again to regain her sanity. These attacks continued to recur at gradually lengthening intervals. Finally she had been perfectly sane for several consecutive months, when suddenly she was seized with a serous diarrhœa, causeless as far as could be ascertained, and hopeless as far as relief by remedies was concerned. In forty-eight hours she was dead. I believe that the cause of that death was the same obscure something which had so potently affected for years the emotional life: that which for so many years had dominated the nerve-centres of higher life attacked and paralyzed the lower centres of animal life, and death came speedily.

We can scarcely conceive the nature of a lesion which, after having for fifteen years held the nerve-centres in an iron grip, suddenly let go its hold. For its demonstration the microscope is useless. Our best instruments show us in human spermatozoa nothing but irregular, transparent specks of protoplasm, not to be distinguished one from the other. Yet the records of past generations are written in the little formless particles, in which also are enfolded the potentialities of future successions of men. Structure and function seem so widely independent that it is almost hopeless to expect that we shall ever understand the infinitely delicate changes which take place in the complex protoplasm of the brain, and to be able to say why waves of emotional and mental paralysis sweep over the individual. I believe that the changes are physical, but I believe that it is not within human power to recognize their nature. The microscope is a coarse, blundering tool, powerless to reveal the ultimate changes of nervous protoplasm gone mad.

I have ventured to occupy space with the above considerations, partly because they seem to me very important, and partly because, for the purposes of brevity, I shall omit the section of Pathology in the articles upon the pure insanities.

A scientific, thoroughly satisfactory classification of insanities is in the present state of our knowledge probably not possible. Holding as I do the belief that many of the so-called insanities are mere symptom-groups arbitrarily separated, the simplest arrangement seems to me the best. In accordance with this I shall adopt the following classification, which is quite similar to that of Krafft-Ebing:

Group I.—Complicating Insanities.—The outcome of a distinct organic disease of the brain, not dependent upon acquired or inherited constitutional diathesis.

Meningitis, tumors, and most other organic brain diseases may be associated with disturbance of cerebration, but usually the mental symptoms are subordinate to other evidences of organic brain disease, and most of these diseases have been discussed in the present volume under the head of Organic Diseases of the Brain. In both the acute and chronic forms of periencephalitis, however, the evidences of mental aberration so predominate over the physical disturbance that the subjects usually find their way to insane asylums, and the disease is usually treated of in text-books on insanity, and this custom is here followed.

The mental aberration and deterioration of old age are commonly supposed to be dependent upon organic change. In accordance with this view I shall consider it in the present group. *Amentia,* or imbecility from arrest of development, may also well be considered as among the organic insanities.

Group II.—Constitutional Insanities, in which the cerebral disorder is due to an acquired or inherited constitutional disease, including in the latter term diathesis, constitutional diseases, and subacute and chronic poisonings involving widespread areas of the body.

The most important of the diathetic insanities are the gouty, the epileptic, the hysterical, and the syphilitic. Numerous poisons disturb cerebration, but the only toxæmic insanity which it seems necessary to notice at this place is that due to alcohol.

Group III.—Pure Insanities, in which the mental disorder is not dependent either upon demonstrable organic brain lesion or upon a diathetic or other poison. The pure insanities seem to me very naturally divided into two subgroups, which may be known as the Functional Insanities and the Neuropathic or Constitutional Insanities.

The *Functional Insanities* are those insanities which are liable to occur in almost any person, or at least which do occur in individuals who have previously shown no mental warp, and who may recover and during later life remain free from mental aberration. *Constitutional Insanities* are the outgrowth of an original vice of nervous construction, such vice of construction not being sufficient to reveal itself by anatomical peculiarities, but showing its presence throughout life in functional aberration. The general tendency of constitutional insanity is to increase in severity as the patient grows older, and a constitutional insanity, once developed, is rarely if ever permanently recovered from. It is especially these forms of insanity which grade so insensibly into sanity. At the bottom of the series is the typical human individual; then the man who is original and strikingly independent in thought and act; then the man who is so set apart by mental peculiarities from his fellows that he is known as eccentric; then the lunatic, eccentricity grading by an unbroken series into a complete insanity, the subject of which is not to be influenced by the motives which usually dominate men, and is indeed incapable of reasoning correctly or indeed of controlling his own acts.

Functional Insanities.	Neuropathic Insanities.
Melancholia.	Constitutional affective insanity (folie raisonante).
Mania.	Moral insanity.
Confusional insanity.	Paranoia—insanity with irresistible ideas.
Terminal dementia.	Periodic insanity (folie circulaire).

GROUP I.—ORGANIC INSANITIES.

Acute Periencephalitis.

Definition.—A very acute, usually fatal, disease of the brain, attended by stupor, wild delirium, general disturbance of the psychic functions, by restlessness, convulsions, and other disturbances of the motor function, and by fever; dependent upon acute hyperæmia and subsequent inflammatory changes in the brain cortex.

Synonyms.—Acute peripheral encephalitis; Phrenitis mania gravis; Typhomania; Acute delirium; Delirium grave; Bell's disease (Luther Bell).

Pathology.—Excessive hyperæmia affecting both the cerebral cortex and its membranes is the first alteration in the present affection. This is rapidly followed by œdematous exudation, with a choking up of the lymph-spaces both of the pia and the cortex by the corpuscular elements of the blood. The periglanglion space, as well as the interstitial lymph-sheaths, becomes crammed with these bodies. I have myself seen also minute apoplectic hæmorrhages in the gray matter. In one case which I examined the ganglionic cells themselves appeared to have undergone some change.

Etiology.—Acute periencephalitis appears to occur fully as frequently in women as in men, and usually during active adult life. Abuse of alcohol, profound grief, protracted worry, especially when accompanied by great overwork, partial starvation combined with the gnawing anxiety of deep poverty, certain acute fevers, sunstroke, blows upon the head,—these are commonly assigned as the causes of the disorder, which also in some cases appears to have been the result of chronic disease of the skull or its membranes. The affection may also develop as an exacerbation of chronic periencephalitis, and I have seen it come on without apparent cause during locomotor ataxia. Recorded cases of death from alleged acute hysteria have probably been instances of this disease. The combination of overwhelming mental and physical strain is perhaps the reason of the comparative frequency of the disorder during pregnancy following seduction.

The **symptoms** may come on with extreme suddenness or may be preceded by prodromic evidences of cerebral disturbance. These prodromes in rare cases take the form of increase of mental power, in others of brief nocturnal attacks of wandering, delirious restlessness; or there may be short periods of impaired consciousness, especially upon waking in the morning, or, as in one of my cases, even an epileptiform convulsion. The fully-developed disorder naturally divides itself into two stages—first, that of acute maniacal delirium; and second, that of apathy and collapse, with coma.

The delirium is always of an excited type, accompanied by violent incoherent speech, and usually by a fury of fighting and of destructiveness. Hallucinations and half-formed delusions are present, and often bear a close relation to the cause of the attack. The abandoned mistress will in her ravings recount her past shame and present agony. The business-man will be perpetually occupied with an incoherent jumble of business transactions.

Almost invariably along with the delirium there is great physical restlessness, which grows more intense until it causes the patient to leap from his bed and to attempt to run away. Very commonly violent assaults are made upon the attendants. Convulsions are rare. The delirium may at first be not continuous, occurring only at night, or at least be interrupted by brief intervals of comparative rationality during the daytime. Finally, however, there is persistent intense mania. In one of my cases the patient during the day told his wife that she must protect herself from him—that he loved her most fondly, but that he was going into a condition of insanity in which he would certainly kill her. From this time until his death he was furiously maniacal during the night, although for several days he would recognize his friends during the daytime, and for a moment or two talk rationally. There is usually absolute insomnia. The pulse is rapid, and, if in the beginning it possesses a show of force, it is really soft and compressible. There is no desire for food, and generally an absolute refusal to take it. There is also distinct fever, the temperature rising sometimes to 106° F. According to my observation, the temperature varies with a stormy irregularity which is almost characteristic, rising and falling many degrees many times during the twenty-four hours. Its variations are connected with the mental and physical excitement of the patient, maniacal outbursts producing an immediate rise of the temperature. In advanced stages the temperature may fall much below the normal. The pupils may be contracted, dilated, or normal. In the course of a few hours to several days the second stage of the disorder develops. There is now quiet, with coma or else muttering, delirious unconsciousness, failing pulse, cool skin, and general evidences of collapse. In the early part of this stage, when aroused, the patient may respond incoherently or perhaps give some slight evidences of comprehending what is said to him, but rapidly sinks lower and lower until he dies from exhaustion. Early in the disorder the skin becomes very harsh, and finally cyanotic; in the later stages irregular desquamation, or even ulceration, may occur. In a case quoted by Spitzka the anæsthesia was so complete that the patient gnawed off a portion of one of his fingers. Pemphigus-like vesicles, phlegmons, decubitus, gangrenous patches of skin, or gangrenous extremities not rarely appear, but are frequently absent, and are not characteristic.

Diagnosis.—Diseases having no connection with the brain may sometimes simulate an acute periencephalitis. This is especially true of the abrupt maniacal outbreaks which sometimes occur in a latent overlooked pneumonia. The fact that acute delirium is a disease of middle life, whilst the so-called cerebral pneumonia occurs almost exclusively in young children or in persons broken down by age, excesses, or privations, should put the practitioner on his guard, and a physical examination would detect a pulmonic disease. Typhomania is distinguished from acute meningitis by the absence of general hyperæsthesia, stiffness of the muscles of the back or extremities, and of pronounced headache. In acute mania the bodily temperature is usually normal or subnormal, and, according to Krafft-Ebing, the rise of the temperature in such a case to 100.5° F. indicates strongly delirium acutum.

Prognosis.—The prognosis is highly unfavorable: about two-thirds of the cases end fatally, and when recovery occurs the mind is almost universally left more or less affected. Alcoholic cases are especially dangerous: the more violent the delirium, the insomnia, the motor disturbance, or the fever, the worse the outlook.

Treatment.—In the early stages of delirium acutum general or local bloodletting by means of leeches, irritating purgatives, the local application of cold to the head, seem to be strongly indicated, whilst hypodermic injections of morphine and of hyoscine, with the administration of chloral by the mouth, serve to allay the excitement. Much better results are obtained by repeating the remedies at short intervals in comparatively small doses than by giving large doses at long intervals. The Italian physician Solivetti has claimed extraordinary results from hypodermic injections, every eight hours, of 1 gramme of ergotin. Certainly the use of ergot would seem to be indicated, and the severity of the disorder thoroughly justifies the risk of any local trouble from hypodermics. A filtered solution of the officinal extract of ergot in freshly-boiled water should be used. In the later stages of the disorder alcoholic and cardiac stimulants may be employed *pro re nata*. Throughout the disease every effort should be made to obtain absolute rest, with freedom from the causes of excitement, whilst milk, eggs, and similar nourishing, non-irritating foods should be administered as freely as the patient will take them.

Chronic Periencephalitis.

Definition.—A chronic disease, dependent upon a peculiar inflammatory degeneration of the cerebral cortex, which gives rise to change of character; progressive mental deterioration, with delusions of grandeur, emotional exaltation or emotional depression; occasional maniacal outbreaks and epileptic attacks; progressive physical deterioration, as shown by irregularity of the pupils, disorder of speech, loss of control over the movements of the hands and legs,—all symptoms finally being swallowed up in a complete paralysis of intellection and of voluntary motion.

Synonyms.—Paretic dementia; General paralysis of the insane; Paresis; Dementia paralytica; Periencephalo-meningitis.

Etiology.—Heredity plays a very unimportant rôle in the production of general paralysis, a positive taint being present only in about 15 per cent. of the cases. The disease is very uncommon in females, and exceedingly rare in females of the upper classes, whilst it is remarkably frequent in officers and other military officials, in whom, according to Mickle, it also occurs at an earlier age than in other persons. Thus in civil life the affection is most frequent between forty and fifty, and extremely rare under thirty or over sixty, whilst in sailors and soldiers it is affirmed by Mickle that the average age is about thirty-three. These peculiarities are, however, probably simply dependent upon differences of exposure to the three great causes of the disorder—namely, alcoholic and venereal excesses, syphilis, and habitual long-continued over-exertion, accompanied by the strain of excessive ambition or of worry.

The connection between the disease and syphilis is distinct. Mendel affirms that in general paralysis 75 per cent. of the victims offer a distinct history of syphilis, whilst only 18 per cent. of the victims of other insanities investigated by him were syphilitic. The relations between the two diseases are evidently precisely those which exist between syphilis and locomotor ataxia: dementia paralytica, indeed, occurs not rarely as a complication of tabes dorsalis, whilst spinal scleroses are not rare in dementia paralytica. Sunstroke and blows upon the head are also set down by authorities as among the exciting causes of periencephalitis.

Pathology.—At autopsies upon old cases of chronic periencephalitis are usually found hyperostosis and exostosis of the skull; pachymeningitis in some form (often absent); arachnitis (with consolidation of the arachnoid with the brain); atrophy of the convolutions, especially of the frontal lobes; and internal hydrocephalus.

Two distinct views prevail as to the nature of the disease-process: one, that it is a diffused interstitial cortical encephalitis, in which the connective tissue is primarily affected; the second, that it is a diffused parenchymatous inflammation, which commences in the nerve-elements proper and involves secondarily the neurogliar tissue.

On section the brain-cortex is usually found discolored, sometimes less, sometimes more firm, than normal, often containing minute cysts or cavities varying in size from a pin's point to a millet-seed. Microscopic examination reveals degeneration or perhaps complete disappearance of the ganglionic cells and a peculiar alteration of the white fibres, which renders them much more apparent than in the healthy brain, besides pronounced degeneration of the neuroglia and large numbers of peculiar many-processed connective-tissue cells (Deiter's or spider-shaped cells). The blood-vessels are usually injected, altered in character, with distension of the adventitial lymph-spaces. The spinal cord is very frequently degenerated. Changes in the sympathetic ganglia have also been noted by recent investigators.

Symptomatology.—The symptoms of general paralysis vary so greatly that it is exceedingly difficult to reduce them to order. Four stages of the disease are recognized by some writers, but the individual case usually passes by such imperceptible degrees from bad to worse that these divisions must be looked upon as arbitrary. Moreover, in various cases the time relations of these stages vary, and some of the stages are often altogether absent or pass unobserved. Nevertheless, for the purposes of discussion I shall speak briefly of these four stages, and then take up the consideration of the individual symptoms of the disorder.

The first or prodromic stage often passes without recognition. The symptoms may resemble those of an ordinary cerebral neurasthenia—loss of power of fixing the attention, apathy, inability for mental exertion, and some emotional departure from health. In some cases vaso-motor phenomena are pronounced, showing themselves in facial congestion, headache, vertigo, tinnitus aurium, hemianopsia, and even peculiar disturbances of vision, simulating an

acute glaucoma. At the same time a slight alteration of character is evident to the close observer, the patient being in some way not himself. Krafft-Ebing gives as almost characteristic the peculiar alteration of the relations of the patient to time and space, which render him exceedingly unpunctual or cause him at times confusedly to lose himself in well-known streets. Although this stage is so often overlooked, yet after the disease has declared itself the books and correspondence of the business-man or the office histories and records of the professional laborer will, in their loss of accuracy and dignity and in their general evidences of failing power, afford a history of a slowly-progressive mental degeneration.

The second stage of the disease is that in which the mental aberration is pronounced and distinct. The disordered cerebration may be accompanied by distinct disturbance of the motor faculties, but I have seen it persist for more than a year without the slightest failure of the general physical powers.

The third stage of the disease is that in which motor symptoms become marked, as shown in inequality of the pupils, flabbiness and loss of expression of the face, disorders of articulation, general loss of endurance, and mayhap distinct paresis of the extremities.

The fourth stage of the disease is that in which the dementia is complete, and the general widespread paralysis and loss of power profound, the patient being reduced to a mere living automaton.

For the purposes of discussing the mental phenomena of paretic dementia the cases may be divided into four groups, it being remembered that in nature every grade of case exists between these groups, whilst the march of the mental malady is sometimes so irregular that in one portion of its career the case would be properly assigned to one group, whilst at another period it would represent another variety of the disorder.

In the first form of paretic dementia are included those cases in which progressive failure of power constitutes almost the whole mental disturbance, the mental faculties consentaneously growing less and less until the patient becomes childish, and at last completely demented, without emotional disturbance or delusions having been present. (It is these cases especially that are popularly spoken of as *softening of the brain.*)

The second variety of paretic dementia is that in which delusions of grandeur or expansive delirium are present. The character of these delusions has already been sufficiently pointed out. (See page 537.) It is essential to remember that these delusions may exist in so mild a degree that they may be very readily overlooked. Further, in many cases they are replaced by a *bien-être* which may be looked upon as a condition of undeveloped delusion. Thus the man sunk in the deepest poverty will be excessively happy and jolly, misfortunes having no power to depress him, although he makes no assertion of the possession of great power or wealth. In all cases of the present variety of general paralysis there is progressive mental failure, and it is therefore evident that the cases in which a simple *bien-être* exists may be looked upon as midway between the first and the second variety of the disease.

Maniacal outbursts may occur in any variety of general paralysis, but they are more common and more frequent when there are delusions of grandeur.

The third form of general paralysis is that in which there is emotional depression, and even pronounced melancholia, with depressive delusions. Not rarely the depressive delusion relates to the person of the patient, who believes himself ill, deformed, or wanting in some member or function. In this way arises the so-called hypochondriacal variety of general paralysis.

The fourth form of general paralysis is that described by Dr. Fabre, in which excitement and depression alternate so as to make a periodic or circular insanity. The existence of this variety has been confirmed by Dr. W. Julius Mickle,[1] who further says that when there are only two phases these succeed each other suddenly, but that in some cases there are three periods—(1) excitement, (2) calm, (3) depression, in this differing, therefore, from non-paralytic circular insanity, in which the usual order is (1) excitement, (2) depression, (3) quietude or lucidity.

The motor symptoms of paretic dementia consist of epileptiform convulsions and paralysis. The paralysis is characterized by its incompleteness and its connection with tremors and disorders of co-ordination. In the earliest stages of the disorder the loss of control over complicated muscular movements is first manifested in the hands, and may be very pronounced at a time when the general muscular power is but little weakened. Thus, a man may be able to lift many pounds, although he cannot write his own name. The acute development of such a loss of muscular control, occurring in a man of middle age, without obvious cause, is a serious symptom, and probably, in the majority of cases, is prodromic of general paralysis. It is especially to be noticed very early in engravers and other persons whose daily vocation requires great technical skill.

A varying inequality of the pupils may occur very early, although more constant in the later stages of the disease. It may be associated with excessive dilatation or contraction. When there is no affection of the eye or its nerves, no focal brain lesion, and no disease of the neck or of the cervical spinal cord, this symptom is very characteristic.

The departure of the speech from the norm in general paralysis is partially of mental and partially of physical origin. As a consequence of the loss by the lips and tongue of their delicacy of movement there is a difficulty of pronunciation, which is especially manifested with lingual and labial consonants and in the syllables of long words. This causes a peculiar stuttering or hesitation, with some thickness of speech and an occasional elision of syllables, so that the speech somewhat resembles that of intoxication. In advanced stages of the disease the uncertainty of the movements of the lips and tongue is plainly visible to the eye, and is associated with tremor, or, more correctly, with tremulousness. In general paralysis the mind thinks slowly and imperfectly: it fails not only in the formation of ideas, but also in the quick association of these ideas with suitable words. There is, consequently, slowness

[1] *General Paralysis*, London, 1880.

as well as hesitation of speech. In some cases the mental actions seem to be performed in a rhythmical manner, giving rise to a peculiar utterance which somewhat resembles that used by the school-boy in scanning Latin poetry, and hence often spoken of as the "scanning speech." There is also in many cases a use of improper words. Not rarely the paralytic talker drops a word from his sentence or repeats a word; mayhap he elides or repeats a whole clause. Movements of the jaws similar to mastication may take place, and even cause grinding of the teeth or champing of the jaws.

The loss of adroitness and exactitude of movement may first appear in the hands. The handwriting becomes shaky and irregular, and the letters are ill formed, even widely separated from one another, sometimes resembling hieroglyphs rather than members of the Roman alphabet. Very frequently the finely-graded strokes of correct writing disappear in a common, thick, uncertain line. The writing not only shows the physical degradation, but has the same mental characteristics as the speech. The ideas are often incongruous and devoid of proper association and the words incorrectly used. Letters are dropped out, syllables omitted or repeated, and words or even clauses elided or interjected.

The gait may be early affected. It becomes awkward and uncertain; the steps may be long and slightly irregular; and the patient's lack of control over his movements comes out sharply when he attempts suddenly to turn or to alter his position. As the disease progresses the gait becomes slow, heavy, and unsteady, whilst the widely-separated feet readily trip over an inequality or unexpected obstacle. In the advanced stages the posture of the patient resembles that of old age, the body being bent awkwardly forward or to one side. With difficulty he walks with a slow, unsafe, swerving gait, in the most advanced stages tottering forward, aided by an arm or some support, and day by day losing control over his limbs until he becomes bedridden.

Epileptic convulsions may occur in the beginning of a general palsy, and may, indeed, usher in the first distinct symptoms of the disease. Under such circumstances their significance may readily be overlooked. This is especially the case when the major attacks are replaced by or associated with petit mal, in which the only symptom of the seizure may be a sudden pallor with mental confusion or a momentary unconsciousness, or a dilatation of the pupils with drawing of the head, or a sudden fixation of the countenance with an outpouring of cold perspiration, or an automatic repetition of coherent or incoherent phrases. Such paroxysms are apt to be interpreted as syncopal, or sometimes as apoplectiform. Not rarely epilepsy in general paralysis takes upon itself the Jacksonian form, the convulsion being limited to isolated groups of muscles, or to one side of the face, one leg, or one arm, or being hemiplegic. Usually the attack begins with an aura, which is especially apt to be vertiginous. Sometimes the convulsion is preceded for several days by excessive restlessness, tinnitus aurium, and great psychical excitation. In other cases it begins with vomiting.

The epileptic attacks are apt to become more and more frequent as the dis-

ease advances, and the observation of Esquirol that a succession of epileptic fits frequently closes the scene in general has received abundant confirmation. When the true epileptic status occurs during a general paralysis the successive convulsions are often very diverse, one being complete, the next partial—in one the head being drawn to the right, in the next to the left, and so on. Frequently after the paroxysms convulsive tremblings persist in single muscles or in groups of muscles for many hours, and are followed by a more or less pronounced partial palsy. To use the words of Dr. Nichol, paralysis follows the convulsion or spasm as the shadow follows the body. During the more severe paroxysms consciousness is always lost, but, especially when the convulsive movements are more or less local, it may be perfectly maintained; occasionally it is affected as in hysteria. After severe seizures the mental condition of the patient is almost always distinctly aggravated.

It is affirmed by many authors that in general paralysis apoplectic attacks followed by a temporary hemiplegia sometimes seem to replace the epileptic paroxysms. Vaso-motor disturbances may occur among the prodromes of dementia paralytica, revealing themselves in loss of tone in the pulse; in sudden attacks of localized vaso-motor paralysis, often affecting the face and head and accompanied with vertigo ("rush of blood to the head"), or giving rise to localized superficial alterations of temperature, or even to localized sweatings. In the later stages the widespread cyanosis, superficial œdema, and coldness of the extremities, with the frequent neuro-paralytic hyperæmia of the internal organs, indicate the loss of power in the vaso-motor centres. Probably to a trophic rather than to a vaso-motor disturbance belong the frequent attacks of herpes zoster and other herpetic eruptions.

Disturbances of sensibility beyond a mere passing numbness are very rare in the early stage of dementia paralytica. Violent lancinating pains, when present, are proof of the development of tabes. In the advanced stages general sensibility is lessened and may be almost destroyed, or, what is perhaps more frequent, tactile sensation may be preserved to some extent whilst the analgesia is complete. This condition often leads to accidents which are only to be prevented by the greatest care. Thus, I have known a paretic dement scald himself to death by getting into an overheated bath, and all kinds of injuries are from time to time reported in the journals. Violent sexual excitement is often one of the earliest symptoms of general paralysis, but in the progress of the disease it gradually gives way to impotence, excessive libidinousness often persisting after the total loss of sexual power.

Disturbances of temperature, especially a tendency to an evening rise and to irregular paroxysmal alterations of temperature without apparent cause, are very frequent in general paralysis. Especially characteristic is a tendency for violent fever to be produced by every slight cause. The epileptic paroxysms are often accompanied by a marked rise of temperature, which cannot altogether be dependent upon the convulsion, since it frequently precedes by as much as eight or ten hours the development of the attack, and often lasts twenty-four hours after the fit. Moreover, violent epileptic fits may

occur without elevation of the temperature, and both Mendel and Westphal have put on record cases in which a violent, long-continuing epileptic paroxysm was accompanied and followed by a marked fall of temperature. Usually, however, a distinct drop in the temperature at the close of an attack of unconsciousness marks the occurrence of a true apoplexy. Irregularity in the temperature of the two sides of the body seems not to be rare in advanced dementia, in which condition there is also a tendency to subnormal temperature.

Diagnosis.—A positive diagnosis in the prodromic stages of general paralysis is not possible, so that the greatest care must be exercised in making statements to the friends of the patient, although sufficient certainty may exist for the purposes of treatment. Marked alterations of character occurring at this time are very significant. (See page 552.) In the more advanced stages of the disorder it is essential to separate syphilitic and focal organic disease. For diagnosis between dementia paralytica and cortical syphilis see page 730. Focal disorders are at once distinguished by the fact that in them the tendency is to localized paralysis, whereas in general paralysis the tendency is primarily to loss of co-ordination, and latterly to a widespread muscular weakness rather than a distinct localized paralysis.

In those cases in which the mental disorder precedes for a great length of time the development of physical symptoms the diagnosis must be between dementia paralytica and a pure insanity. Irregularity of the pupil under such circumstances would be an almost deciding symptom, and usually mere loss of power in the execution of fine movements, such as those of writing, buttoning and unbuttoning the clothing, dancing, etc., can be detected in the paretic dement even when the general muscular power is very good. If this cannot be done, a study of the memory becomes important. Distinct and progressive failure of memory in a patient whose mental symptoms correspond to those of a commencing general paralysis is almost positive proof that the case does not belong among the pure insanities.

Prognosis.—The prognosis of acute periencephalitis is very unfavorable if the diagnosis be positively made out: the most that can be hoped for is to obtain a remission of the disease.

Treatment.—The treatment of general paralysis must be for the most part hygienic or symptomatic. In the very beginning of the case, whilst the diagnosis is still doubtful, repeated local bloodletting by means of leeches to the temple, or the repeated use of the actual cautery or of other counter-irritants to the nape of the neck, may be of advantage. Such measures are especially effective where cortical disease follows sunstroke or blows upon the head.

It is very doubtful whether drugs have any direct power over the diseased processes, but corrosive sublimate may be given in doses of one-twentieth of a grain three times a day, continued for many weeks, or the iodide of potassium, five to ten grains a day, may be substituted. Some alienists have claimed very good results from the employment of massive doses of ergot. The solid extract should be employed, and it may be given in doses of from fifty to sev-

enty grains a day, continued for many weeks, unless distinct physiological effects are produced. For the relief of symptoms, hyoscine, morphine, sûlphonal, and other narcotics may be used in times of wakefulness or excitement, whilst tonics and laxatives are to be employed pro re nata.

The hygienic treatment consists in the protection of the patient by very warm clothing and the administration of a non-stimulating but abundant and nutritious diet; the use of massage, moderate bathing, careful outdoor exercise, etc. In all cases it is essential that physical as well as mental and emotional excitement be avoided as much as possible.

The treatment of the latter stages of the disease should be purely hygienic and symptomatic, especial care being exercised to see that the patient be protected from fæcal and urinary discharges, and that every precaution be taken to prevent decubitus. It is often necessary to keep the patient on a liquid or semi-liquid diet, as fatal pneumonia from particles of food getting into the lungs is a not very rare occurrence.

GROUP II.—CONSTITUTIONAL INSANITIES.

The constitutional insanities are not distinct forms of disease, but groups of symptoms of various and varying character which are the outcome of constitutional vice or disease. Thus there is nothing in the symptoms of a gouty insanity which would enable us to diagnose the nature of the case. The cause of the mental aberration in such a case can be recognized only by recognizing the presence of lithæmia. The importance of distinguishing an insanity of the present class lies in the fact that relief is to be obtained not by treating the insanity, but by treating the diseased condition which is the cause of the mental disorder.

The most important of the constitutional insanities are the gouty, the epileptic, the hysterical, and the toxæmic.

Gouty Insanity.—It is well known that gouty paroxysms are frequently accompanied and preceded by peculiar nervous irritability. At such times there is a depression of spirits, with an irritability so great that it can scarcely be controlled by the patient. In some cases these symptoms become so intensified as almost to amount to insanity; moreover, hallucinations, delusions, loss of mental power—indeed, almost every conceivable manifestation of mental disorder—may be directly or indirectly caused by gout. Carrol in 1859 said: "Gouty mania is occasionally seen;" and in 1875, Dr. P. Berthier[1] published a collection of 44 cases of nervous disease attributable to gout—1 of hallucinations; 1 of migraine; 4 of tetanus; 3 of chorea; 1 of hypochondria; 7 of epilepsy; 1 of paralysis; and 26 of mental affections, including in these dementia, melancholia with stupor, mania. Although in some of these cases the evidence is not at all positive that gout was the *materies morbi*, yet in others the relation seems to have been clearly made out.

In his paper read before the International Congress at London, 1881 (iii. 640), Dr. Raynor supported the following conclusions:

[1] *Des Névroses diathésiques*, Paris.

1. Protracted gouty toxæmia, when not very intense, usually results in sensory hallucinations or melancholia.

2. Sudden and intense toxæmia results in mania or epilepsy.

3. Intense and protracted toxæmia usually results in general paralysis.

4. If there be a tendency to vascular degeneration from plumbism, alcoholism, etc., varying degrees of dementia are produced.

In the discussion which followed the reading of Dr. Raynor's paper, Drs. Savage and Crichton Browne of London both expressed the belief that gout does cause insanity, the latter, however, qualifying by the statement, "only where there is hereditary predisposition to insanity."[1]

The conclusions of Dr. Raynor are borne out by a case of my own. A lady at regular intervals of four years had had a number of attacks of severe gout, associated with great depression of spirits, at times amounting almost to pronounced melancholia. Finally, at the end of four years of health, the patient was seized with symptoms of acute dementia or stuporous melancholia, associated with marked tenderness of the nerve-trunks, and, in certain portions of the body, violent neuralgia, and a urine that was loaded with uric acid and urates. Death occurred after some weeks from œdema of the lungs. At the autopsy there were found gouty kidneys and a remarkably pronounced atheromatous degeneration of the cerebral vessels, the lumina of some of the arteries at the base of the brain being almost obliterated.

Hysterical Insanity.—The peculiar mental organization (see p. 594) which underlies constitutional hysteria in its aggravated forms may amount to a distinct and characteristic psychycosis whose relations with neuropathic insanity is very evident. This psychosis has been characterized so vividly and succinctly by Dr. Folsom that I quote his words: "It is characterized by extreme and rapid mobility of the mental symptoms—amnesia, exhilaration, melancholic depression, theatrical display, suspicion, distrust, prejudice, a curious combination of truth and more or less unconscious deception, with periods of mental clearness and sound judgment which are often of greater degree than is common in their families; sleeplessness, distressing and grotesque hallucinations of sight, distortion and perversion of facts rather than definite delusions, visions, hyperæsthesias, anæsthesias, paræsthesias; exceeding sensitiveness to light, touch, and sound; morbid attachments, fanciful beliefs, an unhealthy imagination; abortive or sensational suicidal manœuvres, occasional outbursts of violence; a curious combination of unspeakable wretchedness alternating with joy, generosity, and selfishness,—of gifts and graces on the one hand and exactions on the other. The mental instability is like a vane veered by every zephyr. The most trifling causes start a mental whirlwind. There is no disease giving rise to more genuine suffering or appealing more strongly for sympathy. Yet when this is freely given it does harm. One such person in the house wears out and outlives one after another every healthy member of the family who is unwisely allowed to devote herself with conscientious zeal to the invalid."

[1] For discussion of Epileptic Insanity see the article on Epilepsy, page 617.

In nature the mildest hysteria grades without break in the series into the most severe, and the difficulty is to decide when hysteria has crossed the line that separates responsibility from irresponsibility; but certainly in some cases of hysteria the mental symptoms are sufficient to indicate restraint. Further, in some cases of hysteria an acute mania or a peculiar automatism with loss of conscious self-control may occur. (See page 594.)

Toxæmic Insanities.

The only insanities of the present class requiring notice here are those springing from the abuse of alcohol. Such mental disturbances may be divided into the subacute and chronic forms, to which the names delirium tremens and alcoholic insanity may be assigned.

Delirium Tremens.—Delirium tremens is a peculiar series of acute symptoms which are produced by excessive drinking. The affection is especially apt to develop upon the sudden cessation in the use of the stimulants, but may come on during the debauch. In their mildest form the symptoms constitute that condition known by old drunkards as "the horrors," in which the sleep is disturbed, the hand tremulous, the mind weak and confused, and the patient troubled with frightful imaginings, vague alarms, and an apparently causeless depression of spirits. When the attack is more severe, hallucinations of sight, of hearing, and, more rarely, of touch, occur. These hallucinations always have in them an element of terror or of horror. Disgusting objects, such as snakes, toads, rats, and mice, and similar unclean creatures, crawl over the bed or the person. Voices predicting evil or bringing messages of remorse or uttering threats of punishment are heard. The patient may seem violent, and may even attack his attendants, but the violence is that of terror, and not of aggression. The attack is an attempt at defence. There is great insomnia, and usually when the patient can be made to sleep the mind is clear after the awakening. This is not, however, invariably the case.

I have seen delirium tremens gradually pass through successive days of wakefulness and nights of sleeping into a chronic mania not readily to be distinguished from that arising from other causes. In the earlier attacks of delirium tremens occurring in very robust people, when all the mucous membranes are irritated, and when probably there is direct irritation of the brain and its meninges, there may be a slight febrile reaction and even a strong and excited pulse; but the disease is typically asthenic, with loss of muscular power, tremulousness, and rapid, feeble pulse, and when death occurs it is from exhaustion. Cardiac failure is in such cases always to be guarded against.

Sometimes the patient suffering from delirium tremens has sufficient rationality to receive his physician with a quiet, gentle courtesy and to answer questions without irritation. It will be noted, however, that he is evidently preoccupied, and that occasionally he turns his head or casts furtive glances from one part of the apartment to the other; and a little finesse will reveal the fact

that during the whole time he is seeing visions or hearing sounds, or is at least laboring under a profound apprehension of attack.

Diagnosis.—The diagnosis of delirium tremens is usually easy, even when the history of the case is not clear. The peculiar terror underlying all the delusions, hallucinations, and attempts at violence is characteristic, as is also the tremulousness of the hands when extended. When pneumonia occurs during a period of delirium tremens the type of the delirium may change, tremors may be lost, and the patient may become so violently aggressive as to lead to a mistaken diagnosis.

Treatment.—In the treatment of delirium tremens the first indication is for restraint to prevent injury by the patient to himself or to others. Freedom in a well-padded room may be allowed, but in the majority of cases such a room is not available, and properly-constructed straps securing the person in bed are, in a violent case, much better than restraint by means of nurses, the strap exciting less of antagonism than does an attendant, and being more steady and certain in its restraint. The second indication is for the support of the system by means of highly nutritious and stimulating food. Milk, strong soups or beef essence with eggs stirred into them just as they have ceased boiling, and similar liquids usually constitute the best articles of diet. As the digestion is in these cases often deranged, it is essential for the practitioner to remember that the food which nourishes is not that which enters the stomach, but that which is digested; so that the effort should be by frequently repeated small portions of nutriment to get as much material worked up as possible. Again, the mucous membrane, which has been accustomed to the local effects of alcohol, is often simply beneficially stimulated by amounts of red pepper and other spices which would produce gastritis in a normal stomach; hence, even though the stomach of the drunkard be inflamed, highly-seasoned food is usually of great service. The limit of the amount of food given in these cases should be the limit of possible digestion.

In the medical treatment of delirium tremens the first indication is in most cases to relieve abdominal engorgement and to remove effete materials from the system. Very frequently the best practice is to begin by the exhibition of three grains of ipecacuanha in pill form every fifteen minutes until free vomiting is produced, and even if the patient be suffering from excessive nausea and vomiting, this practice is often of service. On the other hand, when great feebleness exists the use of such an emetic may be improper. After the ipecacuanha has acted a grain of calomel may be given every hour until free purgation is induced, and not rarely an effervescent mixture containing citrate of potassium is very useful in acting as a depurant through the kidneys. Again, profuse sweating produced by pilocarpine or the hot bath may sometimes be useful, and there are robust cases which can be successfully treated by veratrum viride, in repeated doses until vomiting results.

The second indication for medical treatment is to quiet nervous excitement. For this purpose the bromides and the hydrobromate of hyoscine must be relied

upon. These drugs should be given steadily day and night at regular intervals, the hyoscine being withdrawn if it be found in any way to disagree with the patient.

The third indication is to produce sleep. For this purpose various hypnotics have been used. Sulphonal may be employed; paraldehyde has been exhibited; but the combination of chloral and sulphate of morphine far exceeds in efficiency and general applicability all other hypnotics. The chloral (fifteen to twenty grains) and morphine (grain one-quarter to one-third) may be exhibited at bedtime, and repeated in half-doses at intervals of an hour, *pro re nata*, care being exercised not to overdo the exhibition of narcotics.

The fourth indication is to support the system by means of stimulants. One of the most important questions to be decided is as to the necessity of using alcoholic drinks. In many cases of delirium tremens alcohol does harm rather than good, and in the majority of cases its use is not essential. The moral reasons against its employment are very strong, and therefore commonly it is not wise to use it. On the other hand, in feeble subjects or in old alcoholics the exhibition of alcohol in some form or other may be necessary to the saving of life. In many cases of delirium tremens strychnine is serviceable as a stimulant, but when there is any fear of cardiac failure digitalis is the most reliable of all remedies. It must be given in very large doses, and usually enormous amounts are well borne. Various clinicians have claimed very good results from the exhibition of half-ounce doses of the tincture, but it seems to me that a safer method is to give from ten to twenty minims at intervals of from two to four hours, watching closely the effect, and withdrawing the remedy as soon as any evidence of the digitalis pulse can be perceived.

Alcoholic Insanity.—The prolonged use of alcohol may lead to a gradually increasing functional disturbance of the nervous system, ending it may be in structural change. Under the continuous influence of the narcotic the brain performs its functions slowly and imperfectly and the mental movements become sluggish and weak; the memory is greatly impaired; the power of fixing the attention steadily diminishes, but the intellectual weakness is especially shown by the lessening of the power of the will, so that not only is the judgment uncertain, but its dictates are not carried out. There is also a distinct tendency to emotional depression, and often a peculiar suspiciousness which is the groundwork for delusions. A step farther, and hallucinations haunt the victim. The route to insanity and irresponsibility from this condition is short. Out of such a state is easily developed the most characteristic and frequent form of alcoholic insanity—namely, that with depressive delusions. In some cases this variety of alcoholic insanity appears suddenly with symptoms for a time not to be distinguished from delirium tremens. Indeed, I think it perfectly correct to say that a patient may pass from delirium tremens into alcoholic insanity.

It is affirmed that headache and other symptoms of sudden congestion of the brain especially usher in the attack of alcoholic insanity. When the symptoms

are active, hallucinations[1] are very numerous, constantly changing, and almost always are such as to inspire terror or disgust. In a very short time they are accompanied by delusions of persecution: voices of reproach, threatening, or remorse, mocking faces, unclean beasts, tormenting devils,—these and similar visions drive the victims into profound melancholy, and finally may lead to suicide or murder. According to Spitzka, the delusions of chronic alcoholism almost always relate to the sexual organs, to the sexual relations, or to poisoning. Underlying this variety of alcoholic mania is frequently an intense fear which may lead to violence, as when a man kills his wife because he fears that she will poison him. Not uncommonly the depressive sexual delusion leads to an outburst of uncontrollable jealousy and rage, so that wife-murder from motives of jealousy is not a rare result of alcoholic mania. There is in some of these cases a very marked relation between the presence of alcohol in the blood and the insane outburst. The maniacal drunkard may be, when not under the influence of the poison, fairly rational, but is converted by alcohol into a wild beast, although he has few or none of the ordinary symptoms of intoxication. The man may walk straight and talk rationally on general subjects, but be profoundly under the influence of a depressive or persecutive delusion which disappears when the blood is freed from alcohol.

The relation between depressive alcoholic insanity and mania a potu is, as has been already stated, very close. Insomnia, emotional excitement, especially connected with fear, hallucinations, and delusions, are common to each; but the tremors are more marked in delirium tremens, and when an attack of alcoholic insanity is acute and tremors are pronounced it may be considered to be mania a potu.

Dr. F. Lentz[2] calls attention to a form of alcoholic insanity with expansive delusions and hallucinations of sight and hearing, which, very strangely, in most instances relate to God and a future state. Visions of supernatural beings, and especially of the Deity bathed in an aureola of light, perpetually delight the patient; the ministrations of angels seem to bring relief, or mayhap the voice of God himself is heard in command or instruction.

It would appear that two forms of alcoholic insanity must be recognized—one a lypomania, or melancholia with delusions of persecution; the other a megalomania, with a strong tendency to religious hallucinations.

GROUP III.—PURE INSANITIES.

Melancholia.

Definition.—An acute or chronic pure insanity, characterized by the dominance of depressive emotions.

Melancholia usually comes on gradually, with insomnia, increasing depres-

[1] Spitzka says they are usually of vision; Dr. F. Lentz (*De l'Alcoholisme*) says that they are almost exclusively of hearing: my experience is that both forms of hallucination are frequent.

[2] *Op. cit.*, p. 491.

sion of spirits, malaise, failure of digestion, and often a whole range of minor nervous disturbances similar to those of an ordinary neurasthenia.

The basis of the fully-formed disease is a psychical anguish or depression, manifesting itself on all occasions and dominating all the life. It does not differ save in its intensity from emotional depression in health resulting from profound grief or other sufficient cause. It is only, after all, a multiplied exaggeration of a mood, but it affects all cerebration. Through it the whole outside world is hung in black, and every perception of this world is painful (*psychical dysthesia*), or all perceptions are flattened down by the absence of desire and are scarcely felt by the consciousness (*psychical anæsthesia*), or mental acts or perceptions cause intense disgust (*psychical hyperæsthesia*).

During all of this time there may be no loss of reasoning power, no true intellectual insanity. The patient fails to be interested in the life around him, not because he is incapable of understanding the problems of life, but because nothing but himself is of interest to him or occupies his thoughts, or perchance because all other things fill him with disgust, and the mere effort to drag away his attention from his own feelings gives to him great pain (*psychical hyperæsthesia*). In the lighter degrees of the affection the patient will simply say that he is horribly depressed and cares for nothing. With the apathetic total lack of energy so characteristic of the disease, the subject sits all day in a chair, quiet, perhaps with the hands folded, seemingly thinking of nothing, with an expression of perfect indifference and apathy on his countenance. There is no interest in business, in wife, or family, not because the relations are not recognized, but because the man is absorbed in his own woe, and is paralyzed by the psychical pain that attends any effort.

When the symptoms are more active and severe, instead of simple apathy there is wringing of the hands and perpetual moaning and lamentation, not for any definite reason that the patient can assign, but simply because of the depression of spirits. Under these circumstances it will be found that all his thoughts are tinctured with this emotional depression. If the man be a business-man, he sees nothing but ruin before him. If he have a conscience which is not void of offence, the memory of his past misdeeds, like a Nemesis, for ever haunts him. If his children are ill, they are going to be swept away by death. The whole landscape is covered with a black cloud, which throws everything into the darkest shadow. Nevertheless, there may be even yet no intellectual delusions. When the patient is aroused he talks well and reasons well. If you can get him to forget himself for a moment, his intellectual actions are perfect. After a time delusions make their appearance. They are in typical cases always unsystematized. They usually develop gradually, and not rarely are the outcome of some real feeling which the patient has. They may exist with or without hallucinations. Both hallucinations and delusions always take the depressive type. Hallucinations of hearing are the most frequent. The patient hears voices, but they are evil voices. Those who have committed murder have sometimes asserted that they had two voices in them, one crying, "Kill! kill!" the other voice trying to restrain them.

Men have held their hands in the fire until they were burnt black because they have heard voices telling them that it was better to enter into the next world maimed than to go with a whole hand guilty of blood or other offence.

Sometimes hallucinations of sight occur, but these are less common than hallucinations of hearing. Troops of spirits from the other world pass before the patient—never angels or spirits from heaven, but always demons of sorrow and of woe.

Delusions of touch are rare, and delusions of smell are still more uncommon. I do not recall a case in which I have seen a patient with delusions of smell. They, however, are occasionally present. The melancholic never smell pleasant odors. It is always sulphurous vapors or horridly fœtid exhalations that oppress them.

The sensory disturbances of melancholia are distinct and sometimes severe. Headache may be among the prodromes, but usually the complaint is not so much of distinct true headache as of indescribable distress, a sense of emptiness or of pressure or of some other paræsthesia in the head. In the height of the disease paræsthesia, more or less widespread, or partial anæsthesia, irregular hyperæthesia, and neuralgia, are often present. The most intense sufferings of melancholia are those produced by a peculiar horrible distress referred to the upper chest, and commonly known as "præcordial anguish." This appears to be simply an exaggeration of the cardiac distress sometimes produced in normal life by sudden and overpowering sorrow. Especially frequent in the morning hours, it may occur at any time of day, and, whilst usually paroxysmal, may continue for a length of time. The attacks usually come on suddenly, and rapidly reach their height until perchance the agony and its accompanying terror so dominate the consciousness that it is obscured or lost in the wild delirium in which, with blind disregard of himself and others, the patient convulsively attacks and destroys all within his reach—stripping himself or herself naked, breaking, mashing, cutting, tearing, even perchance disembowelling himself or twisting off the genitalia. Self-mutilation, murder, suicide, utter destruction of sensate and insensate objects,—these are the not infrequent outcomes of a despairing fury inspired by agony and unutterable terror (*raptus melancholicus*).

During a paroxysm of psychical anguish the respiration is usually rapid and superficial; the heart's action quick, irregular, with small, thready pulse; the skin cool and white: not rarely the paroxysms end abruptly with a profuse sweat—facts which have given origin to the belief that the præcordial anguish is a neurosis of the sympathetic system.

In melancholia the sexual function is usually very much depressed, often for a time being set aside. In the female, at the menstrual period, the general symptoms are apt to be worse, and hysterical nervous disturbances are not uncommon. Even early in the disorder sleep is broken, unrefreshing, disturbed by horrible dreams or veritable visions of terror, and during the height of the disease it is often almost altogether put aside. The physical health and the general nutrition are always lowered, the tongue coated, the

breath heavy or perhaps very foul, the appetite almost or completely wanting, or replaced, it may be, by an absolute loathing for food. The urine is scanty, heavily loaded with urates, oxalates, or phosphates; the muscular system relaxed, the desire for and indeed almost all power of exertion being lost. In severe cases progressive emaciation may go on rapidly to great wasting, accompanied by subnormal temperature, dry and harsh or clammy and cold skin, and cold cyanotic extremities.

Cases of melancholia seem to naturally group themselves into melancholia simplex (simple melancholia), melancholia attonita (melancholy with stupor), and melancholia agitata (melancholy with motor excitement).

The cases of simple melancholia are divided by Krafft-Ebing into—

First, *melancholia without delirium*, which includes those cases of melancholia which are not accompanied by intellectual insanity—cases which are usually associated with neurasthenia, hysteria, hypochondriasis, and similar affections, which are often called forth and called into being by untoward circumstances acting upon a neuropathic subject. Among the millions of men gathered into the American armies in the late war mild melancholia, the result of *nostalgia* or homesickness (not of cowardice), amounted in some cases to a true insanity.

Second, melancholia with præcordial anguish.

Third, melancholia with delusions and hallucinations.

Fourth, melancholia religiosa, in which the delusions lead the subject to believe that he is deserted by God, and that through eternity he shall suffer the pangs of damnation; or perhaps convince him that he is already possessed with devils (*melancholia demoniaca*).

Fifth, melancholia hypochondria, with hypochondriacal delusions. In a large proportion of the cases the delusions are connected with the sexual organs. This form of melancholia shades by insensible degrees into *hypochondriasis—i. e.* that condition in which the subject centres all his thoughts upon his own health and grossly exaggerates his own symptoms, or perhaps imagines symptoms which do not exist. Hypochondriasis by insensible degrees again grades into the natural state through a series of cases in which there are various degrees of excessive solicitude concerning health.

MELANCHOLIA ATTONITA (melancholia with stupor.)[1]—This is a very

[1] The *katatonia* of Dr. Kahlbaum, which by some authorities is believed to be a distinct insanity, is thus defined by Spitzka:

"Katatonia is a form of insanity characterized by a pathetical emotional state and verbigeration, combined with a condition of motor tension.

"The illness begins with an initial stage resembling that of an ordinary melancholia. This is followed by a period in which the patient presents an almost cyclical alternation of atony, excitement of a peculiar type, confusion, and depression, which finally merges into a state of mental weakness approaching, if not reaching, the degree of a terminal dementia. Any single one of these enumerated phases may be absent.

"The excited stage presents symptoms of a kind different from those of ordinary melancholia, and constitutes a connecting link, as it were, between the symptoms of an agitated melancholiac and those of a lunatic with fixed delusions. Some of the patients present exaggerated, others diminished, self-esteem, and not rarely does the developing delirium assume an expansive tinge.

severe form of melancholia, in which the patient passes the time in a condition of partial or even complete stupor, seemingly motionless and emotionless. In extreme cases the mental condition can scarcely be distinguished from that of primary dementia: the cerebral power seems to be completely abolished. The careful observer can, however, usually distinguish this state from that of primary dementia by noticing an occasional anxious look, a wrinkling of the forehead or other muscular contraction, which shows that the individual has still some power of thought or perception. The two states are also separated by the fact that the melancholiac after recovery has some memory of events which have happened during this condition of stupor.

In melancholia with stupor mild muscular contractures, and especially marked rigidity of the muscles, are common. In some cases there is distinct catalepsy. Sensation may be normal, but there may be either anæsthesia or hyperæsthesia. The quick, small pulse, the dry, harsh skin, the aged expression of the face, the slow superficial respiration, the subnormal temperature, the general failing secretions (including menstruation), the loss of digestive power, the tendency to emaciation,—all these are but the evidences of the general depression of the functions and of nutrition which is so marked in this form of melancholia.

Melancholia Agitata is that variety in which there is great excitement, the patient being continually on the move, rushing up and down, lamenting loudly, wringing the hands, tearing the hair, destroying his clothes, etc. The agitation may rise to the point of complete frenzy. The melancholic frenzy differs from that of mania in being founded upon a state of intense terror and fear.

Melancholia may pursue an acute, subacute, or chronic course. In even the most acute cases many weeks are required for recovery, the subacute continuing many months, the chronic many years. The recovery is usually gradual, but I have seen sudden recovery after sixteen years' illness. Remissions are common. About 60 per cent. of the cases get well, the proportion being much larger in the mild cases and much less in melancholia with stupor.

But all katatonics exhibit a peculiar pathos, either in the direction of declamatory gestures and theatrical behavior or of an ecstatic religious exaltation. Frequently the patients wander about imitating great actors or preachers, and often express a desire and take steps to become such preachers and actors."

The hallucinations of katatonia are always depressive and accompanied by a melancholic depression of spirits, which is said, however, never to be so painful as in melancholia. Severe occipital headache and cataleptoid attacks are asserted to be characteristic. The cataleptoid condition is typical and extreme, the patient remaining for long periods corpse-like and immobile. I have seen two cases which perhaps ought to be classed as katatonia. Whilst under observation there was no headache and no period of excitement, but the cataleptoid condition was very marked. For hours the patient would remain standing or sitting, perfectly immobile in whatever position he might be placed. Certain forms of melancholia attonita (melancholia agitata) resemble katatonic insanity, and the line of separation seems a very uncertain one. Masturbation is alleged to be frequent in katatonia, and was markedly present in one of my cases. It is, however, very common in all classes of mental weakness approaching dementia.

Death may take place from tuberculosis, colliquative diarrhœa, or other complications, and a small percentage of the cases end in terminal dementia.

Treatment.—In the treatment of melancholia the first indication is to obtain, as far as possible, bodily and mental rest. Arguing with the person, attempting to convince him of the wisdom of overcoming his feelings and delusions, are usually worse than useless: the attempt should be to divert his thoughts to other objects. When they can be carried out without too much friction, rest in bed, with massage, electricity, and the other concomitants of the so-called "rest-cure" are important. The second indication is the maintenance of the bodily power by means of properly regulated tonics and as nutritious a diet as can be obtained. In many cases forced feeding is essential. The third indication is the cure as rapidly as may be of any existing disease, since melancholia may originate in or be kept up by gastro-intestinal catarrh, hæmorrhoids, or other abdominal disease. The fourth indication is to subdue nervous excitement, and to procure sleep by the use of carefully-selected narcotics. If there be much excitement during the day, hydrobromate of hyoscine, in doses of the one-eightieth to the one-hundred-and-twentieth of a grain, at eight to twelve hours' interval, often acts most happily. At night chloral may be used with much certainty of action, but I think its continued use is more detrimental than that of sulphonal or even of opium. In some cases of melancholia the use of opium in the form of the extract, at regular intervals of six to eight hours, acts most happily. Prolonged warm baths, or, better still, hot packs, are from time to time of great service in quieting agitation and producing sleep. Beer or other alcoholic drinks may sometimes be given with advantage as narcotic stimulants, but the moral danger of their use must not be forgotten. In all cases of melancholia great watchfulness against sudden destructive suicidal or homicidal outbreaks is essential, as it is impossible to foretell with absolute certainty even in mild melancholia what will happen in the next few hours.

Mania.

Definition.—A mental condition in which there is great emotional exaltation dominating the individual, and accompanied with a greater or less degree of mental aberration.

Acute mania is sometimes developed with great suddenness; more often it is preceded by a prodromic stage of emotional depression. The depressive stage may last from a few days to three months. There is often a sense of lassitude, with inability or disinclination to work, a lack of the usual power of fixing the attention, depression of spirits, and a feeling of unrest which causes the subject to worry perpetually about himself; the bowels are costive, the appetite is poor, and dyspeptic symptoms are often troublesome. The resemblance of this condition to a mild melancholia is so close that it is frequently spoken of as the melancholic stage of mania. Usually, after a time, the subject gradually returns to his normal state, except perhaps that his perceptions are abnormally quick and that he is abnormally happy or even gay.

Rapidly now the emotional excitement rises, delusions and hallucinations appear, and the maniacal stage is reached. When the affection as at its height its victim raves incessantly, shouting out a perpetual stream of incoherent threatenings, revilings, obscenities, and blasphemies. With a prodigious and untiring strength he rushes about his apartment, struggles with his attendants or his mechanical restraints, tears into shreds whatever clothing he can lay his hands upon, destroys all about him that is breakable, smears his excrement over his person and surroundings, and so passes whole days and nights in unceasing fury. The insomnia is almost complete, and if for brief intervals sleep comes it is filled with dreams and is broken and fitful. The hallucinations and unsystematized delusions are constantly changing. There is usually great sexual excitement, as shown by satyriasis or nymphomania. There is often a marked blunting of sensation, so that the maniac does not feel the wounds he inflicts upon himself in his blind fury. A remarkable feature is the tirelessness: it seems as though no continuance of effort could produce exhaustion. The appetite is usually excellent, the digestion remarkably powerful, the general muscular tone is increased, co-ordination and quickness of movement are remarkable. In spite, however, of the apparent perfection of health, and in spite of the taking of enormous quantities of food, the bodily weight almost invariably falls progressively.

In most cases of acute mania periods of excitement alternate with periods of comparative calm. The usual duration of the disease is from three to six months, although recovery may occur in a few days or be postponed for over a year. Death may take place from exhaustion, or the mental aberration may pass into chronic mania or into a condition of slight mental impairment. Complete recovery takes place in about 70 per cent. of the patients.

In very many cases of acute mania the symptoms are milder, but of similar character to those just detailed. Restless, licentious, blasphemous, incoherent, obscene, the maniac lacks the fury of the previous picture, or, occupied by his own hallucinations and delusions, he may be rapt in a delirium of enjoyment. In a still milder form acute mania shows itself in incoherence, irrationality, restlessness, evidences of hallucinations and delusions, with marked insomnia, and total loss of modesty and of care for or notice of the usual relations of life.[1]

[1] The so-called "*transitory frenzy*" is defined by Spitzka as "a condition of impaired consciousness characterized by either an intense maniacal frenzy or a confused hallucinatory delirium, whose duration does not exceed the period of a day or two." In its symptoms it resembles a very violent acute mania, excepting in that its duration is but a few hours, and that afterward the patient has no remembrance of what took place during the attack. It scarcely differs symptomatically from the maniacal fury which sometimes replaces the epileptic convulsion, excepting in that it does not recur. It is well known that an epileptiform convulsion may occur only once, and yet exactly resemble the convulsion due to idiopathic epilepsy. The relation between transitory mania and epileptic mania seems, in fact, to be the same as that which exists between an isolated epileptiform convulsion and a convulsion which forms one of a true epileptic series.

The importance of this subject lies chiefly in its medico-legal relations. Thus, in a case reported by Dr. Theo. Diller (*Alienist and Neurologist*, 1892) the patient killed his own mother whilst in a paroxysm of transitory fury which developed during convalescence from influenza.

In the mildest possible form of this disease—*hypomania*—the hallucinations may be wanting, and the mania reveal itself only in a change of character, a peculiar egotistic hilarity, perpetual extravagances, restlessness, increased sexual appetite with lessened control of the will-power, leading to great sexual excesses and a tendency to brutal violence. The diagnosis in these cases is often very difficult, and can be made only by noticing the complete alteration in the life, disposition, and mental, moral, and physical habits of the individual. Indeed, I believe that precisely as does melancholia (*i. e.* depressive emotional condition) so also does mania (*i. e.* exalted emotional condition) grade insensibly by rare cases into the normal condition, and that there are states in which the will still exerts its control, although the mental and moral attributes are so altered that the man is not his natural self. Every one has his hours and sometimes days of exaltation, as well as his hours and days of depression, and exactly when or how far the mood triumphs over the individual who shall say?

Chronic Mania may develop as the result of an acute mania, or may come gradually without a preceding stage of violence. It is a condition of general mental aberration characterized by the presence of varying or non-systematized delusions and by a condition of exalted emotional excitement. In most cases the chronic maniac, although more or less disturbed intellectually all the time, suffers from irregular exacerbations, in which the condition of excitement may become extreme and the symptoms rise in severity until they resemble those of an original acute mania. During these paroxysms, and often indeed in the intermissions, there are incoherence of speech, lack of power of association of ideas, delusions, often increased activity of the perceptive faculties, with hallucinations and mental and physical excitement. The symptoms of chronic mania are similar to those of acute mania, but are less severe in type. They are also modified by the progressive failure in the intellectual power as the patient drifts toward dementia. The hallucinations and delusions are unfixed, constantly changing, are not systematized, usually are conformed to the emotional excitement of the patient, or, if they should take for the moment a depressive or disagreeable form, do not affect the mood of the individual. They may be concerning any conceivable person, thing, or place, or may take forms not reached by the most vivid imagination in its sane moments. The moral sense is completely altered or abolished: those to whom the individual had previously been attached become objects of hate; modesty there is none, the patient revelling in obscene speech and immodest gestures, and often suffering from sexual fury. Like acute mania, chronic mania varies greatly in its intensity; indeed, the mild form of mania known as hypomania is especially apt to pursue a slow course. Chronic mania not rarely changes into chronic melancholia: whether primary or secondary, it is of long duration. Occasionally recovered from, it usually terminates in from two to five years in dementia.

In this case the relations of the patient to his mother were such that no prosecution was entered upon, indeed, rather strangely, no coroner's inquest seems to have been held.

Confusional Insanity.

Definition.—An acute insanity produced by nervous shock or exhausting disease, without distinct constant emotional depression or exaltation, with marked abatement of mental power (ranging from a mild mental confusion to complete imbecility), often, but not invariably, accompanied by hallucinations and great mental excitement; with loss of physical power, usually disturbances of temperature; the whole commonly ending in complete recovery.

Synonyms.—Primary curable dementia; Stuporous insanity; Stupidität; Delusional stupor; Mania hallucinatoria; Wahnsinn; Surgical insanity; Puerperal mania; Post-febrile insanity; Mania following typhoid and other acute fevers.

Etiology.—Almost all writers on insanity recognize that a violent sudden emotional strain may produce great mental disturbance, and even complete loss of mental power. I have seen the chagrin and worry of having cashed a large forged check produce in a bank-cashier a typical confusional insanity. Various acute diseases are occasionally followed by insanity. The reasons for believing that these insanities following acute disease are of similar character are, first: Though the cases vary very much in their details, the general scope of the symptoms and the general course of the disorder are identical. There is always mental confusion, a mixture of excitement and failure of mental power; and the cases nearly always end in complete recovery if free from organic disease. Second: The belief that the insanity has a specific relation to the disease or to the surgical operation which it has followed necessitates the acknowledgment of a dozen or more acute insanities connected with specific diseases. Thus, I have seen the insanity after ovariotomy, perineorrhaphy, removal of the breast for cancer, and various other surgical operations; also after rheumatism, typhoid fever, diphtheria, epidemic influenza, and various other acute diseases. All these affections have one influence in common—*i. e.* they all tend to exhaust or impair the nutrition of the nerve-centres; and it is known that impairment of the nutrition of the centres by lack of food, combined with anxiety, is capable of causing symptoms similar to those which are present in insanities developed after disease.

Symptomatology.—In various chronic diseases attended with great bodily and mental exhaustion the brain-tissue gradually passes into a condition of perverted and exhausted nutrition similar to that of confusional insanity. Probably most of us have seen in long-drawn-out cases of consumption such a gradual impairment of the mental faculties, associated with a superactivity of the imagination, as to render the unfortunate individual a distress to himself and to his friends. As the disease progresses the mental disorder goes farther, and especially during the night the patient becomes delirious.

Almost every history of shipwreck, followed by long exposure and starvation, affords a recital of failing mental power, accompanied by increasing activity of the imagination, until desire and thought-pictures give rise to hallucinations which are at first recognized by the sufferer to be false, but at

last become to him living realities, luring him to leap overboard into what seems to him a land of plenty, but is in truth a watery abyss of death.

In times of famine or amongst the very poor of certain parts of Europe habitual privation occasionally produces a series of symptoms in which the delirium is subordinate to the complete mental failure, and that form of confusional insanity known as primary curable dementia results (*Stupidität*—Kraft-Ebing)—a condition which, it must be remembered, may also be caused by sudden shock, and which grades through a series of cases into the second variety of confusional insanity.

In extreme cases representing the so-called primary curable dementia there is almost complete paralysis of the mental function, with a loss of nerve-tone in every portion of the body, so that the patient continues in a condition of more or less profound stupor or stupidity, with shifting kaleidoscopic anomalies of motor and vaso-motor innervation, and perhaps at times of delirium or of hallucinations. The muscular strength is markedly reduced; the pulse feeble and small, usually quick, and greatly increased by voluntary effort or even by active passive movements; œdematous swellings are frequent, and shift from part to part with remarkable inconstancy, but when, as in many cases, the patient for hours at a time holds the position of standing or sitting in which he has been placed, the œdematous swelling of the lower portions of the body shows the excessive vaso-motor weakness. The pupils are usually somewhat dilated and react feebly and slowly. The cutaneous reflexes are lessened, the sensibilities diminished or perhaps almost altogether lost. The tremors of excessive weakness are sometimes present, and there is a condition of partial catalepsy. The bodily temperature is usually subnormal, and in spite of the most careful and even forced feeding the bodily weight sinks. An almost constant phenomenon is an enormous increase of the phosphates in the urine.

In the worst cases, without thought and without perception, speechless and motionless, reacting scarcely at all to any external stimuli, with even the vegetative life reduced to the lowest ebb, the unfortunate victim of the disease still continues the automatic life.

In the second form of confusional insanity (*hallucinatory insanity*) the mental symptoms may seem to be contradictory, since many of them are those which are commonly believed to be the outcome of paralysis of cerebral functions, and others are such as are sometimes thought to be evidence of excited, though perverted, cerebral activity. In the first group belongs that depression of consciousness which in the mildest forms may be shown only by a peculiar quietude and by apathy, but which in varying degrees of greater severity manifests itself by a stupor which ever grows as the disease becomes more severe in intensity, until it deepens into a complete, persistent loss of consciousness. Another outcome of cerebral weakness is the peculiar mental confusion which is the most characteristic manifestation of the disease. It may reveal itself chiefly in the inability of the patient to talk coherently and persistently—words dropping out of the sentence or being uttered imperfectly, because the mind is unable to get the right word; ideas changing in the middle of a sentence, because the power of confin-

ing the attention to one consecutive line of thought is lost—so that the attempt at conversation on the part of the patient results in a jumble of half sentences, clauses, and words hopelessly intermixed one with another. Even, however, in mild cases of disease the mental confusion usually manifests itself, not merely in the inability of the patient to hold a connected conversation, but in his want of power to appreciate persons and things about him. In the more extreme instances no objects or faces are recognized, and even in the very mild forms of the disorder the patient may recognize some of his friends, yet be unable to place himself, insisting that he is away from home, and pathetically begging to be taken to his own house. Clinical experience has shown that during the acute stage of a fever an inability to recognize familiar objects, and a confused, repeated request to be taken home, are of the most serious prognostic import: it is, however, in the disease now under discussion of no such moment.

The confusion of the patient is not altogether the outcome of pure mental weakness, but is usually in part due to the extraordinarily numerous and vivid hallucinations which affect all the senses, and compete for recognition by the consciousness with impulses which really originate in external objects. The weakened judgment is unable to distinguish between the contending claims of subjective and objective sensations, so that realities and imaginations are intermingled in a hopeless chaos; moreover, the memory is pronouncedly affected and old forms and familiar sights are forgotten, and the connection of external objects with the past is, for the time being, severed.

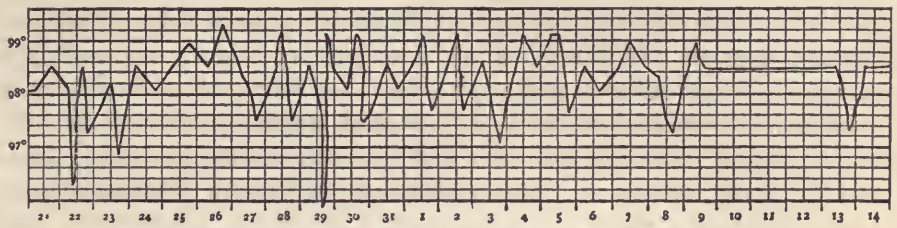
A peculiar delusion, that I have so repeatedly seen in these cases as to think it is somewhat characteristic, is that another person or persons are in bed with the patient.

The delirium is commonly mild and lacking in aggressiveness, but it may take on a very active form, or the patient may be habitually quiet, but subject to paroxysms of fury resembling those of acute mania. In these outbreaks the patient may be violently erotic; indeed, any form of mania may be counterfeited. More commonly, however, underlying even the aggressiveness and violence there is a foundation of fear which often resembles that of delirium tremens, and when with this condition of fear there is associated distinct tremulousness, the likeness to delirium tremens is very pronounced; indeed, I believe that delirium tremens should be considered a form or variety of confusional insanity.

The physical condition and symptoms in this form of confusional insanity are similar in nature to, but usually less pronounced than, those of so-called primary dementia. Little attention has been paid by writers to the bodily temperature, and unfortunately I have seen the disease almost exclusively in consultation, under circumstances which too often have rendered temperature-sheets unattainable. It is certain, however, that the temperature varies in different cases. It may be normal, but in severe cases there is usually either an habitually low temperature or a marked tendency to paroxysms of subnormal temperature. On the other hand, there may be a very distinct febrile reaction.

This I have especially seen in puerperal cases. When fever exists at all, the swing of the thermometrical mercury is remarkable for its irregularity and its extent, and a very high temperature is often followed by a sudden and marked fall below the norm. In order to show the peculiarities of temperature the following reproduction of the chart of a case of the puerperal form of the disease is appended:

Fig. 44.



The figures at the bottom represent the days of the month, beginning June 21st.

Diagnosis.—Very rarely ought there to be any trouble in recognizing the true nature of confusional insanity. The history of the attack, the knowledge that the outbreak has been preceded by an exhausting disease, traumatism, or emotion, the failure of bodily nutrition and of general nerve-force, the lack of dominant emotional excitement, the stupor, the peculiar mental confusion, the kaleidoscopic character of the hallucinations,—all these make diagnosis easy.

Prognosis.—The prognosis in confusional insanity is favorable. Krafft-Ebing states that 70 per cent. of his cases have recovered, and in my own experience, even when the mental confusion has amounted to complete and absolute imbecility, complete recovery has almost invariably occurred, provided that there have been no pre-existing organic bodily lesions, such as unsound kidneys or degenerated arteries. Death may, however, occur in complicated cases. If the mental recovery be not complete, the result is lack of mental power, but never a reasoning insanity, never a state resembling that of paranoia.

It must be remembered, however, that an emotional shock may produce an absolute, permanent overthrow of the intellectual faculties. Thus, Bucknill and Tuke record a case in which a young lady of refinement and education was assaulted and raped by a band of ruffians, and became at once a speechless idiot for life. In a second case a young lady, having by mistake fatally poisoned her father, from the time of his death "was lost to all knowledge or notice of persons and occurrences around: food she never took except when it was placed upon her tongue; the only sound which escaped her lips was a faint yes or no."

Treatment.—Usually the first question to be settled in the treatment of confusional insanity is as to whether the patient shall be sent to an asylum or kept at home. The proper answer to this depends absolutely upon the pecuniary circumstances of the sick person. If there be abundant means to provide a sufficiency of trained nurses, it is much better to keep the patient in his own

house. Nothing can be done in an asylum that cannot be as well done at home, and as the person returns to mental health the shock and depression of finding himself within the walls of an insane asylum may greatly retard or prevent convalescence. If, however, the pecuniary means be restricted, it is essential, in severe cases, to employ asylum treatment. Unless there can be an abundance of thoroughly trained nurses there is little chance that the patient will be properly fed and cared for, and the feeding is of the greatest importance. The appetite is usually *nil*, but the powers of digestion are commonly much better than they seem, so that forced feeding is well borne. Again, not rarely the patient refuses food either in part or altogether, and then artificial feeding must be resorted to, which, of course, requires skilled attendants. In all cases the administering of nutritious and even rich food at short intervals, in as large quantities as the stomach will tolerate, should form the basis of treatment. In a most severe case a patient of my own took daily for weeks sixteen raw eggs in six pints of milk, with most happy results. Usually it is necessary to give the food in liquid form.

A point to which I think sufficient attention is not given is the maintenance of the bodily warmth of the patient: by overheated rooms, by hot-water beds or bags of hot water, and by the warmest clothing obtainable every effort should be made when the temperature is subnormal to restore it. An alarming, if not fatal, fall of temperature is liable to occur suddenly and unaccountably, and to be overlooked till the patient passes into a collapse which may be hopeless. The nurses ought, therefore, to be instructed to continually watch the temperature of the patient, and in the absence of a physician to institute the proper means for heating the body whenever the bodily heat falls below 97° F.

Rest, massage, and exercise are three hygienic measures which must be carefully applied in every case. Their use is, of course, directed against the physical side of the disorder, and they are to be employed precisely as they would be in similar bodily conditions without distinct disturbance of the mental functions. It must be remembered that the underlying bodily condition is one of exhaustion, so that rest rather than exercise is needed. In bad cases the rest must at first be absolute, and it sometimes happens that the exhaustion is too extreme even for the use of massage. Later in the treatment massage and electrical stimulants are often of very great service. It sometimes happens, especially in severe cases, that after a time the bodily strength increases without a corresponding growth in mental power: under such circumstances I have found a removal from the home surroundings to the seashore, mountains, or other places of resort, combined with the encouragement of out-door exercise of a not too severe type, to be followed by the happiest results.

Drugs are to be employed with a twofold object: First, and chiefly, to increase the nutrition of the nerve-centres and general tissues of the body: for this purpose iron combined with bitter tonics, administered in small or large quantities according to the individual character of the case, strychnine given in ascending doses to the limit of physical tolerance, and phosphorus

continuously exhibited in such small doses ($\frac{1}{120}$ to $\frac{1}{100}$ of a grain) as not to disturb digestion, I have found to be of great service. Second, to obtain sleep when there is wakefulness or to quiet delirious excitement. Here the bromides naturally suggest themselves, and they are often used in very large quantities: it should be remembered, however, that the bromides are powerful depressants, not merely to the functional, but also to the nutritive, activity of the nerve-cells; and I am sure that I have seen very distinct injury done by their free use in confusional insanity. In an individual case the selection of a hypnotic or quietant is to be based chiefly upon the results of trial. In some instances opium seems to act favorably; more commonly hyoscine is of advantage. Chloral and sulphonal ought certainly to be tried on occasion. As a calmative the hot-pack is often very serviceable, and I have seen a very active delirium apparently greatly benefited by free blistering of the scalp.

Terminal Dementia.

Almost any form of active insanity may be followed by a long-continued condition in which the mind is so far lost that even the distinctive characteristics of the original insanity have more or less completely disappeared. This state is the so-called secondary or terminal dementia. The completeness of the mental ruin varies: in some cases, apathetic, mindless, without thought or emotion, the individual lives on, a mute, almost motionless, vegetating automaton; in other instances, restless, full of obtrusive or destructive activity, noisy, with incoherent talk, the dement, although overflowing with animal spirits, and perhaps also possessed by a peculiar aggressive egotism, is useless for any purpose—mayhap is almost uncontrollable and very troublesome. Sometimes the mental condition is simply that of a weak-mindedness, and the harmless imbecile seems like an overgrown child. No medical, hygienic, or moral treatment can in these cases avail anything to restore the lost mental power, and the only thing to do is to take care of the individual. Not rarely a little intellectual power remains; and if with this there be docility, the dement may be usefully employed about a farm, in the wards of a hospital, or in other situations in which he can be carefully watched over and constantly directed and taken care of.

Neuropathic Insanity.

Etymologically, the term "neuropathy" means disease of the nervous system. By modern neurologists, however, the term is commonly used in a more restricted sense, and especially as the basis of the adjective "neuropathic," to designate the condition in which abnormal symptoms are produced by an original and acquired vicious development or failure of development of the nervous centres.

Insanities, nervous diseases, drunkenness, syphilis, alcoholism, excessive poverty and lack of the necessities of life,—these and other similar active causes in the parent lead to degradation in the offspring; whilst onanism, taught perhaps even in infancy; the overcrowding, the under-feeding, and the

multitudinous ills which come to the young life born in the midst of extreme poverty; various injuries in early life; acute and chronic diseases setting in before the nervous system has unfolded itself,—these and a thousand other similar possible ills frequently cause in the unfortunate children of the human race a nervous system which for ever dooms its victim to an unhappy peculiarity amongst his fellows. Once engendered, the neuropathic constitution magnifies itself through generations, and so root-stocks appear from which spring criminals, lunatics, and a multitude of other beings concerning whom the world wonders whether they should be considered sane or insane. In very truth, there is no line, at least none that can be drawn by the finite mind. The offspring of such parentage may perform all the duties of life, but his mental organization lacks something or has suffered some twist.

Perverse, drifting almost of necessity into criminal acts, eccentric, such unfortunates are a long series of human atoms whose faulty brain-organization separates them from their more fortunate fellows. When this separation is sufficiently wide, when the mental organization is so bad that every one can perceive that the man is the victim of his own imperfectly-developed brain, he is said to be insane. But when the unfortunate individual is a little more like the normal human being, he is looked upon simply as eccentric, perverse, or wicked, and, unloved and unpitied, drifts through life, sometimes to poverty, sometimes to the hospital, sometimes to the jail, and, it may be, to the hangman's scaffold. Sanity, insanity, criminality, power over self, free will, mental attributes,—these and similar terms are household words with all of us, but no man knows whence they come or what they are, or how far the individual is master of himself or is driven by the hand of fate as represented in the physical conformation of the nerve-cells and fibres of his brain.

As has already been insisted upon, insanity is not a disease or a distinct entity. Necessity for an arbitrary line between sanity and insanity is not of scientific but of legal origin, and when the medical expert affirms that he is unable to measure out accurately the exact degree of human responsibility, he simply acknowledges that he himself is a finite being and that the problems of life baffle his utmost thought. It has been reserved for judges upon the bench and lawyers at the bar to arrogate to themselves the attribute of infinity; whilst ministers of the gospel but too often teach that the last and highest revelation of a merciful God is that this poor, broken humanity, helpless so often in the iron grip of its own perverse nature, shall be punished by flames eternal.

In obedience to some mysterious law of nature the neuropathic victim may be possessed of very high intellectual power, there being, indeed, a close relation between mental aberration and that power of original creative thought to which we give the name of genius. This has been denied by no less a physiologist than Claude Bernard, who lays stress upon the fact that genius is not heritable, whilst madness is. This is, however, no proof of the absence of relations between the two. Genius is not madness, but simply a possible offshoot from a stock of mad ancestry. Genius may exist, as in Goethe, com-

bined with the highest of reasoning powers, but perhaps more usually it is associated with mental and physical qualities which are but too plainly the outcome of defective or peculiar mental organization.

It cannot be a mere accident that so large a proportion of those whom the world speaks of as "children of genius" have had an intellectual life spent upon the border-land of insanity, or a moral history setting them apart from the normal human being and showing but too clearly the traces of their ancestry. Space is wanting to do more than call attention to the monomania of John Bunyan, whose immortal dream was no doubt to himself often more than a dream; to the overweening egotism of Byron; to the agonies of mental depression which overshadowed the life of Cowper; to the hereditary madness which led to orgies of insane cruelty in so many of the world's ablest despots, whether among the more ancient Romans or in the more recent Russian dynasties of Rurik and of the Romanoffs; to the hallucinations which caused Swedenborg to affirm that the hand of Christ "squeezed my hand hard," and Luther to declare that the devil came into his cell, stamped through his cloister, and drove him from his bed; to the glorious visions which inspired Joan d'Arc with a faith almost divine and an energy irresistible; to the direct communications with God which enabled the son of Abdallah to link together millions in a confederacy of religious belief against which Christianity has beaten as yet in vain. Edgar Poe, Heine, Munger, Baudelaire, Gerard de Nerval, Maupassant, Swift, Pascal,—these and many other examples might be cited as showing how close genius is to the mind diseased; but need of brevity forbids. I cannot, however, forbear mentioning at somewhat greater length the immortal Victor Hugo, whose uncle died insane, whose brother Charles (perhaps more talented than himself) entered for life the madhouse before twenty years of age, whose daughter is insane, and of whom the London *Medical Times* is not far wrong in affirming that some of his finest productions are those indelibly stamped with madness. A remarkable fact in the mental history of this greatest of poets was that along with his extraordinary imagination there was a shrewdness almost as great. No banker could have more carefully managed his fortune—no politician could have more tenderly nursed his popularity. He who had amassed over a million of dollars died the idol of a communistic democracy—he who had played at fast and loose with all political parties was buried amidst a tumult of universal sorrow.

Already in the mildest forms of neuropathic brain-weakness the cases arrange themselves into two groups. In the one the disordered enervation shows itself especially in the realm of intellectual action; in the other the morbid or ill-developed nervous system betrays itself in the sphere of morals. In the first group belong chiefly those who are known in the community as "harmless cranks," whilst the second group is made up chiefly of the so-called "criminal classes." Of course in the actual life every grade exists between the man whose eccentricities show themselves perhaps in the extreme piety as well as in the disordered intellection, and the man whose eccentricity is in the sphere of moral character.

In common with most alienists I believe that there are neuropathic subjects who should be considered to have passed over the boundary-line of insanity, although they have no actual delusions. I am fully persuaded that some of these subjects are actually unaccountable beings, who are the sport of their own morbid nervous organizations. Probably in all such cases both the moral and the intellectual character is affected, but in some subjects the perturbation is most obvious in the moral, in others in the intellectual, sphere; consequently, these cases of insanity are divided by some of the best alienists into reasoning insanity (*folie raisonnante*) and moral insanity.

Sometimes in *reasoning insanity* emotional exaltation exists, when the cases are spoken of as being maniacal; in other subjects the depressing emotions are dominant and the individual is melancholic. Reasoning insanity includes those cases in which, perhaps along with high intellectual endowments, there exist imperative conceptions or morbid impulses of such power as to dominate the life of the patient. (See page 539.)

As has already been shown, the morbid impulse is very closely related to, indeed, often springs from, the imperative conception, whilst the imperative conception has the closest of relations with delusions. The sequence of cases in nature from eccentricity to reasoning insanity without delusions, and from reasoning insanity without delusions to paranoia with delusions, is unbroken. A very curious form of reasoning insanity is *folie du doute*, or *doubting insanity*, in which the patient is entirely without confidence in the integrity and reliability of his own mental processes. This mental condition may be looked upon as an exaggeration of self-distrust, and when it is complete leads to insanity of conduct. Thus, a patient said to me, "Two and two make four; so my intellect tells me, but how do I know that my intellect is in its conclusion right?" "I change my baby's diaper at night; a moment later I remember doing it, but how do I know that my memory is correct or that the feeling of dryness which my hand gives me is true?" And so the unfortunate woman and the almost equally unfortunate baby spend hours in the processes of uncovering and being uncovered.

Moral lunatics are those who are not only devoid of all conscience, but actually are driven by their natures to what seem to others horrible crimes. Thus, in the case reported in the *American Journal of Insanity* the lust for blood and the sight of suffering were only satisfied with torture and murder. This man was a moral imbecile driven by a furious impulse to torture and kill: to tie up horses in the woods and gradually whittle them to death, to mutilate living cats, to torture chickens, to break the legs and tear to pieces, whilst living, small birds, were his greatest pleasure; but the very heaven of his joy was reached by assaulting, torturing, killing human beings. When twelve years old he took his toddling brother into the woods and nearly flogged him to death. He attempted to strangle a younger brother and to smother his infant sister; had stabbed various people, essayed to suffocate a harmless imbecile, and to choke another inmate of the asylum, and com-

mitted at least two criminal assaults on women, the last during an escape from the asylum.

Lack of space, however, forbids further consideration here of these strange insanities, and the curious reader is referred to larger treatises on the subject.

Amongst the moral insanities must be classed the various sexual perversions. Without doubt, vice may gradually lead a man on to all the bestialities of sodomy, but there are certainly individuals who are born with a perverted sexual sense which leads them to enjoy from the earliest puberty only the embraces of their own sex. Such men often, but not always, have feminine voices and feminine ways, mincing as they go, dressing in private in women's clothes, affecting all the airs and ways of the silly type of womanhood. I have examined in prison such men, whose statements that they did not masturbate were confirmed after careful watching and long study by an experienced prison surgeon. They affirmed that from earliest puberty women had been to them objects of aversion, and on the rare occasions when, under stress of circumstances, intercourse had taken place, no pleasurable orgasm had accompanied emission; but of man they spoke with rapture, and their eyes would glisten with lustful excitement at the mere sight of a handsome, well-formed man.

Prognosis and Treatment.—In all cases of neuropathic insanity the prognosis is very grave in regard to the mental condition, but very favorable in regard to life: the insanity being founded upon original or acquired neuropathy, remission is the most that can be hoped for.

It is also evident that there can be no specific treatment. The object of the practitioner is, by moral and physical hygiene, to give robustness to the nervous system, and by means of narcotics, very carefully and sparingly used, to subdue nervous excitement when it shall rise too high. In the majority of cases the victim of an insane neuropathy enjoys life, on the whole, better within a well-regulated asylum; and in a large proportion of cases it is only possible to protect society from the lunatic and the lunatic from himself by sequestration.

When, in the neuropathic subject, delusions appear, the case is absolutely and undisputably one of insanity, so that, whatever may be our opinion in regard to the propriety of acknowledging the forms of insanity just spoken of, all must agree that paranoia, or neuropathic insanity with a more or less uniform mental state, and periodical insanity, or neuropathic insanity with periodical alterations of the mental condition, represent true insanities.

PARANOIA.

Definition.—Insanity dependent upon original neurotic vices accompanied by more or less distinctly systematized delusions, persistent, and without cyclical changes.

Etiology.—In the great majority of cases of paranoia there is a distinct hereditary neuropathy, but in a small proportion of the cases the constitutional

neurosis can be traced to drunkenness or other vice in the parents, or to injury or disease occurring during early childhood.

Symptomatology.—Paranoia usually develops slowly, blossoming out of a character which has, from the very beginning of life, shown clearly the tendency to neuropathic madness. In rare cases the symptoms of insanity are developed suddenly in an acute maniacal or delirious attack. The delusions of paranoia are very frequently, but not always, accompanied by or even dependent upon hallucinations, which are said to be most frequent in the sphere of hearing, next in that of feeling, then in seeing, tasting, and smelling.

The course of paranoia is essentially chronic, and it is doubtful whether permanent positive cure can ever occur in a disease which is so intimately connected with an original vice of nervous development. Nevertheless, intermissions are not rare, and may continue for months or even years. They sometimes seem to be almost complete, but usually some evidences of mental aberration are discoverable. After a shorter or longer time distinct insanity recurs—not, however, in the form of a new attack so much as in that of a continuance of the old, the new attack being, as it were, knotted to the insane life which has gone before it. In judging of any individual case as to the present mental condition it must be remembered that the paranoiac not rarely hides his delusions and simulates an intermission.

Exacerbation also occurs in paranoia, accompanied it may be with great cerebral excitement, sleeplessness, and very pronounced psychical symptoms, such as ecstasy, violent hallucinatory delirium, fierce mania, stuporous dementia, etc. Again, some other form of insanity may develop during a paranoia; thus, dementia paralytica is probably more frequent in the victims of a confirmed neuropathy than in ordinary life, whilst alcoholic, hysterical, or epileptic madness are comparatively infrequent.

Paranoia very rarely, if ever, ends in complete dementia, but rather in a condition of psychical weakness and good-natured stupidity, through which may be preserved a certain show of the artistic, professional, or technical abilities originally possessed by the subject. Almost every large asylum has in it such patients, to whom the asylum has become a beloved home, and by whom much of the work of the institution is performed.

Paranoiacs may be divided into those cases in which the symptoms develop about the period of puberty and those in which the active symptoms come on later.

Early Paranoia.—The paranoia of pubescence, or *hebephrenia*, usually occurs in children whose mental life has given evidences of abnormality very early in life. According to Krafft-Ebing, in Europe such children are especially prone to believe themselves "Cinderellas for whom no fairy has awaited;" to become dissatisfied with their surroundings; to dream of higher positions in society; to perceive in themselves unlikeness to their own family and a likeness to some family of higher rank; and as the years go by to persuade themselves that they are the thing that they originally longed to be—namely, the

neglected offspring of count or prince or other social dignitary. Probably, owing to the fact that the difference between social classes in America is comparatively slight, this peculiar development of paranoia is with us rare. More common is an obtrusive self-assertion, combined with a mawkish sentimentality and sexual irregularities. Not rarely, at the time when the character of the boy should develop into that of the man, an arrest in the character seems to take place, and the silly egotism of adolescence becomes the permament stamp of the degraded mentality.

The psychosis frequently commences with depression of spirits, which is, however, usually not so absolute as in original melancholia, and is very often associated with an almost hysterical desire for sympathy, obtrusiveness replacing the peculiar reticence of true melancholia. Very often the mental deterioration is rapid, and it may be so complete as to simulate a dementia. Abnormalities of sexual life are almost universal. In the great majority of cases the patients are inveterate and excessive masturbators, and especially in women life is sometimes given up to a neurotic dream of love, which centres about some person—usually of high rank—with whom the subject may not even have acquaintance.

LATE PARANOIA.—Late paranoia develops after the completion of puberty, and very frequently not until the fortieth or fiftieth year of life. Almost invariably, however, the subject of the disorder has been from early life eccentric in thought and in action. In a large proportion of cases the disease develops gradually, the delusions forming in the mind so slowly that it is almost impossible to say when their seeds have germinated. On the other hand, in some cases the first distinct mental aberration is a violent attack of delirium which may simulate an acute mania. Again, a severe catarrhal attack, a sudden uterine disorder, a pregnancy, a typhoid or other constitutional fever, may be the abrupt starting-point for a paranoia which may readily be mistaken for a confusional insanity.

The cases of paranoia differ very much in the nature of their delusions and in their general symptoms, but for the purposes of discussion they may be arranged in groups, it being remembered that these groups merge into one another, and that sometimes the character of the individual case changes during its course.

The most common form of paranoia is that attended with delusions of persecution. In the beginning the subject feels that the world is becoming hostile to him, or suspicion attaches in his mind to a certain individual or individuals as viewing him with distrust: as time goes by suspicion becomes more intense; a look, a whispered word, a momentary gesture, a sermon, are in his thought proof of hostile intent. The manner becomes unquiet, whilst the face perchance puts on a hunted, anxious look. Little by little suspicion increases to belief, and gradually or suddenly, as the case may be, the paranoiac *knows* that he is the object of persecution, that attempts are being made by poison or in other ways upon his life, that he is accused of crime and threatened by the police, etc. In its delusions the mind often responds to its environment.

Thus, in Europe the paranoiac frequently believes himself the object of political persecution: in America private individuals rather than government officials that oppress him.

Very frequently the delusions have a sexual tinge: the world is conspiring against the man's sexual life or sexual power, or suspicion is directed against the fidelity of the wife or on the part of the wife against the fidelity of the husband. Usually, about the time when the delusions become fixed, hallucinations appear, and in a majority of cases they are chiefly or wholly confined to the sense of hearing: voices of denunciation or reproach, of threatening, of obscene allusions,—these fill life with terror. Less common are hallucinations of feeling: insects crawl over the surface, snakes inhabit the interior, unclean embraces terrify the nights. Somewhat less frequent are hallucinations of taste: especially in cases with delusions of poisoning, the food smacks of arsenic, chloroform, etc. The sense of smell is sometimes implicated, but it is curious how rarely, except in active delirium, optical delusions appear: to *hear* the persecutor is most common, to *see* him is most rare.

In persecutory paranoia the emotional state is one of depression, but in its depth and in its relations this depression differs entirely from that of true melancholia. The victim of melancholia believes himself worthy of all his sorrow—to himself acknowledges his guilt and is humiliated by remorse. The paranoiac, depressed though he may be by his persecutions, knows that these persecutions are undeserved and rebels against them. Again, except in periods of excessive fury, the melancholic lunatic is not dangerous to others, whilst the paranoiac is always an object of danger to his fellows: his impulse is to resist persecution or to revenge himself for wrong, and so, in self-defence or driven to fury by a sense of injustice, he assaults his fancied persecutor. The danger is especially great when the paranoiac believes that his persecution originates in one individual; nevertheless, there is some danger when the persecution is thought to be general, and at any time the upbraiding voice may, in the mind of the paranoiac, attach itself to a passer-by.

Closely allied to the paranoia just described is that form of the disorder in which the subject believes that his earthly possessions rather than himself are attacked by mankind. At first quarrelsome and litigious only, little by little this paranoiac, having spent his time in attempts at lawsuits, becomes when these fail a violent denunciator of judges and judiciary systems, and finally, posing to himself as a martyr or saint militant, fights society for his own rights, or perhaps, taking a wider or more ambitious sweep, contends in every possible way for the general rights of mankind. At large such paranoiacs may be anarchist leaders; shut up in an asylum, they may be most troublesome by their efforts for liberty through the law.

Of different type is the religious paranoiac. Almost invariably this madness has blossomed out from an early character of excessive piety and religious zeal, founded in a great majority of cases on a neurasthenic as well as neuropathic constitution. In very early life sedentary and retiring in habit, unwilling and perchance unable to labor, careless of social duties, these subjects from

the age of puberty give themselves up to religion and to onanism, for the relation between religious and sexual ecstasies is remarkable, and sexual perversions are in these people very frequent. At last, when the nervous system has been weakened by fasting, by sexual excess, by acute illness, or perchance without apparent cause, the visions which constitute the pathognomonic symptoms of the disorder appear, hallucinations of sight being in this form of paranoia much more frequent than those of other senses. The religious paranoiac sees the heavens open, the Virgin and the Son Himself smiling on him, or with strange ecstatic joy looks out upon a procession of the blessed. Later, voices of rapturous singing, of prophecy, or of commendation, it may be from God Himself, add to the overpowering joy of the vision.

Egotism underlies alike the paranoiac hallucinations of persecution and of ecstasy, but in the one case the ego is the persecuted of mankind, the other the praised of Heaven. Often, however, the religious paranoiac has his depressed moments, when the soul contends with devils for eternal salvation.

Allied to religious paranoia is erotic paranoia, in which the subject believes himself to be in love and to be loved by some person usually higher in political, professional, and social life. Such a paranoiac may spend the day in weaving to himself a romance of love and the night in erotic dreams. Hallucinations of hearing seem in this form of paranoia much more frequent than those of seeing.

Paranoia finally ends in a condition of mental enfeeblement, through which may be preserved much of some peculiar talent or power ordinarily possessed. Not rarely, before this stage is reached, the form of the paranoia changes. Thus, by a gradual or by sudden transformation the persecuted paranoiac becomes to himself the child of fortune, a person of high birth, an inheritor perchance of a throne, whilst the erotic paranoiac, disappointed and repulsed, saddens into a victim of undeserved persecution.

Diagnosis.—It may seem easy to determine the existence of an insanity which is attended by delusions, but in the case of paranoiacs great difficulty sometimes arises in deciding how far an opinion held should be classed as a belief or how far as a delusion. This is particularly the case when the belief or delusion is concerning some matter or matters which are incapable, from their very nature, of positive demonstration. Thus, in the matters of religious belief every man who holds strongly to a certain faith might consider every person who believes in a different faith to be insane. The history of modern Spiritualism seems to offer an almost insoluble problem: are those whom the mass of mankind believe to be the dupes of modern Spiritualism, and who themselves believe that they habitually hold communion with spirits, to be considered insane? And how far distant is their mental plane from that of the woman who is convinced that Providence has, as the result of her prayers, put back the ordinary course of nature, caused a cyclone to cease, or put far from her an incurable affection?

In a case of which I had cognizance a successful administrator of affairs of moment and detail upon the frontier of the United States said to a friend:

"My life in this frontier post, from its monotony, would be absolutely insupportable if it were not for my daily mail which I get from my dead friends. I had a letter from your brother last week, and every morning I live in expectation of receiving a letter from some deceased friend or relative." The letters really came from a "medium" in the Eastern United States. On another occasion the gentleman said: "There is one thing that gives me great comfort, and that is that I am a descendant of the Virgin Mary." Some one intimating a doubt of that genealogy, he continued: "I know that I am a descendant of the Virgin Mary, because I had a letter from her, and she says so; and she certainly ought to know." Most alienists, I think, would claim that this man was a paranoiac, and yet how narrow is the space between him and the ordinary believer in Spiritualism!

The delusions of paranoiacs may, on the other hand, be so conformable to what is seen in ordinary life that the recognition of their true nature becomes very difficult. I was once met in the boudoir of a palatial mansion by a handsome young woman, perfectly lady-like and self-restrained in her manner, who, with much quiet show of modest reluctance, told me the story of her having been infected with syphilis by her husband, and of the death of their child through the hereditary disease. Thorough examination convinced me that neither the woman nor the child had had syphilis. When I assured my patient that she was not diseased, she expressed herself much relieved, but was unrelenting toward her husband, whom she accused of all manner of infidelities. At first I entirely believed the woman's story, but after a time it became evident that her charges against her husband were untrue, and that she was a paranoiac with fixed sexual delusions. This woman's intellectual powers were good, she held her place firmly in society, but a study of her past revealed abundant traces of the ungovernable egotism which underlies much of paranoia.

Periodical Insanity.

Definition.—A condition dependent upon original or acquired neuropathy, in which attacks of insanity occur at regular or irregular intervals.

In some women the attacks of periodic insanity have a tendency to recur at the monthly epoch, hence the so-called *menstrual insanity*. Epileptic and certain other diathetic insanities might well come under the heading of periodical insanity, but for convenience' sake have been treated of in other parts. (See page 617.)

The cases of true periodical insanity are naturally arranged for study into two classes: first, those in which the recurring attacks are of similar type; second, those in which the attacks vary in form.

In the first form of periodical insanity the earlier attacks may be so light as to apparently differ from those that follow, but when the paroxysms have once been fully developed they maintain an extraordinary constancy, even as to the details of symptoms. The same imperative conceptions and impulses, the same delusions, hallucinations, and even the same language, are repeated with an

almost stereotyped regularity. During the intervals the patient may be reasonable, capable of attending to business, and neither in conduct nor in conversation give distinct evidences of his disease; but usually a peculiar nervousness, a ready excitability, a persistent hysteria, or some other distinct evidence of the neuropathic diathesis, can be made out, and, as the disease advances, almost invariably there is a deterioration of character and of mind, so that even during the intermission the individual is indifferent, apathetic, with blunted emotions and decreased mental energy, and yet, it may be, at the same time more irascible.

The paroxysms of periodical insanity usually develop more abruptly than do attacks of simple mania and melancholia, from which affections the fully-developed periodical insanity may also usually be distinguished by the presence of pronounced imperative conceptions, of strong propensities or impulses, accompanied by a peculiar perversion of the moral sense.

Mania periodica may begin abruptly or may be ushered in by a very short period of depression. The peculiarity of the mania is that the exaltation is accompanied by a peculiar moral perversion which leads to what is sometimes known as the delirium of action. In other words, the subject is driven by impulses coming from within to perform various forms of impulsive criminal acts—to steal; to expose the person in public; to commit sexual crimes or to enter upon an orgie of sexual excesses; to burn, to wander from place to place, to commit murder, or to end the scene by suicide. Delusions are not common, but hallucinations are more frequent. The mental disturbance is often accompanied by palpitation, rushes of blood to the head, cold extremities, disturbances of secretion, myosis, mydriasis, nystagmus, insomnia, loss of appetite, or other evidence that the whole nervous system takes part in the general process.

The relations between the emotional exaltation and the morbid impulses which play so important a rôle in this form of insanity vary. Sometimes the mania predominates, sometimes the imperative conception; and under the latter circumstances are developed those cases in which criminal acts are performed by persons perhaps not otherwise evidently insane. Some instances of kleptomania (impulse to steal), of pyromania (impulse to burn), and of periodical sexual perversion come under this variety of insanity. If the history of "Jack the Ripper" be ever known, it will probably be found that the man is the victim of periodical mania, chiefly showing itself in sexual perversion, and that the murders which he performs are in obedience to the abnormal sexual impulses and are attended with a furious, almost epileptic, orgasm.

Habitual drunkenness is usually the result of vice, the habit having been formed through self-indulgence at a time when the impulse to drink could have been resisted successfully; but there does appear to be a neuropathic state in which at intervals the subject is drawn by an impulse beyond his control to indulge in a narcotic orgy—a state which may properly be viewed as a periodical insanity, to which the name of *dipsomania* is applicable, and in which the law should give authority for restraint. In one case of my own in which the narcotic impulse recurred at intervals circumstances made it possi-

ble to confine the patient, and after some weeks of almost absolute insomnia, with furious struggling and almost manaical raving, the narcotic impulse disappeared without being drowned in gratification.

Periodical Melancholia is a very rare affection, in which the emotional depression may or may not be accompanied by delirium. The symptoms may closely simulate those of ordinary melancholia, except in the great constancy and severity of suicidal impulses. The diagnosis must rest on the recurrence of the attacks.

In Circular Insanity, or Cyclothymia, the cycles vary in length from a few days to many months: as a general rule, the more violent the symptoms the shorter is the time required to complete a cycle. The arrangement of the cycle varies in different individuals, but is constant in the one case. In this way a melancholia may be followed by a mania, and this by a lucid interval, or the mania may first appear, or the lucid interval may follow the melancholia. The passage from one mental condition to another may be abrupt, but more commonly it is gradual. The mania may be violent, resembling in all its symptoms an attack of ordinary acute mania. It may be so mild as to simply amount to a condition of mental exaltation in which the subject is dominated by all sorts of immoral impulses and tendencies, which lead to a line of conduct that has been aptly spoken of as insanity of action. In like manner, the melancholia varies in intensity from the most profound, hopeless, despairing apathy to a slight depression of spirits. Sometimes the lucid interval is wanting, and mania follows melancholia and melancholia follows mania in perpetually-recurring alternation. There are certain cases in which the symptoms of a circular insanity are so slight that the patient does not at any time, to the eye of the ordinary observer, overstep the bounds of sanity. Such individuals are avoided by their friends as moody and unreasonable: to-day sanguine, talkative, energetic, and extravagant, to-morrow they are taciturn, apathetic, or full of vain regrets for acts that they have done or enterprises that they entered upon while in the condition of exaltation.

The individual attacks of circular insanity offer in their symptoms no peculiarities; indeed, the mania or melancholia of a circular insanity seems in every symptom to correspond to a similar insanity not of the cyclical type.

FUNCTIONAL NERVOUS DISEASES.

By HORATIO C. WOOD.

Neurasthenia.

Definition.—A condition of lack of power of the nerve-centres, not dependent upon the existence of organic disease in any portion of the body.

Synonyms.—Nerve-weakness; Nerve-exhaustion.

Etiology.—Neurasthenia is not, in the proper sense of the term, a disease, but a bodily condition, which may or may not be secondary to organic diseases not directly connected with the nervous system. The common cause of primary neurasthenia is overwork, combined with excessive mental emotion, especially of a depressing character, such as anxiety. The power of creating nerve-energy varies indefinitely in the human individual, whilst an enormous expenditure is required for the performance of the vital functions of respiration, circulation, and digestion. There are persons who, on account of original feebleness, are scarcely able to afford the nerve-force necessary for merely living—such persons are neurasthenics from birth. Overwork is not an absolute, but a relative, term. Whenever the daily expenditure of nerve-force is greater than the daily income, physical bankruptcy and neurasthenia must result.

Nervous exhaustion may involve a few nervous centres, constituting a local neurasthenia, or may implicate the whole nervous system and produce a general neurasthenia. A local neurasthenia is always, however, attended by a general lack of tone and has a great tendency to pass into a general neurasthenia. Sexual excesses produce spermatorrhœa or other forms of exhaustion of the sexual nerve-centres, in most cases soon followed by general neurasthenia, constituting a group of cases which may be known as sexual neurasthenics. Writer's cramp is a local neurasthenia which I have frequently seen to be the herald of a general breakdown. Cerebral asthenia, the result of mental overwork, commonly soon develops into general loss of power.

Symptomatology.—The symptoms of a developing neurasthenia vary greatly in accordance with the part of the nervous system which is the first to give way under strain. When the overwork has been chiefly mental, loss of the disposition to work is usually the first symptom, a more and more painful effort of the will being each day necessary for the performance of the allotted task. The power of fixing the attention upon the kind of work which has produced the disturbance is at first interrupted, but by and by it becomes difficult to hold the attention to any subject. Weakness of memory, disturbance of sleep, sense of weight or of constriction in the head, or other abnormal sensations soon appear.

A developing local neurasthenia may be masked, however, by functional excitement, probably due to active congestion of the nerve-centres. The working power of the failing brain is often for the time markedly increased: the subject may glory in a wild intellectual exaltation, accompanied by an uncontrollable brain-activity, and an almost complete insomnia—a condition which is prone to end in some serious and sudden breakdown.

Gradual as is the development of the neurasthenia in most cases, an almost abrupt paroxysm may occur after unnoticed premonitions. Thus I have seen a man fall in the street with an overpowering vertigo, the first pronounced neurasthenic disturbance.

In general neurasthenia the spirits may be moderately good, but usually there is marked depression, which may deepen into a decided melancholy and a condition bordering on insanity. The will-power becomes weak, morbid feelings develop, and more and more dominate the patient, who at last may become a confirmed hypochondriac. Neurasthenia does not, however, often lead to complete insanity unless there be an inherited taint.

Cerebral asthenia may coexist with the power of enduring physical labor, but sooner or later physical power fails. In this stage (or not rarely as an earlier local neurasthenia) weakness of sight, with loss of power in the ocular muscles, often develops. The nature of the optical trouble can usually be recognized by noticing that vision is good at the first look, but fails when the eye is steadily used for a few minutes, although the organ is optically perfect.

Neurasthenic vaso-motor weakness is often pronounced. Excessive blushing on the slightest provocation, great flushing of the face after the use of alcohol or other stomachic irritant, waves of heat passing over the body, occasional pallors provoked by exertion or apparently causeless, and cold extremities,—these are some of the phenomena which mark the lack of power in the centres that control the blood-vessels. Secretion also is affected. Night-sweats are frequent or the hands and feet are bathed in perspiration, whilst any emotion or excitement produces a violent perspiration or often a sudden diarrhœa.

Excessive irritability of the heart is very common in neurasthenia. Excessive quickness of the pulse, especially exaggerated increase upon exertion, is an ordinary phenomenon. Excessive palpitation, and even pronounced shortness of breath, are not rare. Irregularity and intermittency are not rare, and are usually especially pronounced during excitement or under the influence of gastric or intestinal irritation. When the tobacco habit has been acquired the tobacco heart is almost always present. An apical systolic murmur may often be heard, even when there is no distinct anæmia. These murmurs are usually soft in character, and may persist for months without change. Often, but not always, they can be made temporarily to disappear by putting the patient to absolute rest in a horizontal position.

Gradually the strength fails. Under the influence of excitement much exertion may be possible and not felt at the time, although after one or two days it is followed by complete prostration. Atonic dyspepsia is not rare.

Itching, formication, various paræsthesiæ, violent neuralgia, nervous headaches, often render life a burden. Especially is neuralgia prone to be present when the neurasthenic is of gouty parentage. Very frequently neurasthenia is the underlying foundation of hysteria, and almost invariably in prolonged neurasthenia there is some hysteria, so that the line between the neurasthenic and the hysteric is scarcely more than an imaginary one.

Treatment.—In nervous exhaustion recovery can only be obtained through rest and food, aided by the use of remedies for stimulating nutrition. Minor disagreeable symptoms may be met as they arise by drugs. Strychnine, arsenic, and phosphorus given for a length of time are often of service as alterative nutrients, but the chief reliance must be upon hygienic treatment.

Local neurasthenia, whether existing by itself or as the foundation of a general neurasthenia, requires rest of the organ primarily worn out. Thus in sexual neurasthenia sexual abstinence is absolutely essential. In brain-tire it is the brain which must be rested. To rest an overwearied, excited brain is often not an easy task. In attempting it the effort should be to obtain the following results: first, the removal of all cares, anxieties, and all brain-work, especially brain-work of such character as has been connected with the breakdown; second, the maintenance of the interest of the patient, so that the past shall for the time being be forgotten, and the present not overweighted with irksomeness; third, invigoration of the physical health of the whole body, and especially of the nervous system. In order to obtain the first of these measures of relief isolation of some sort is essential; for the second, mental occupation is usually required; for the third, fresh air, exercise, or some substitute is to be superadded to abundant food and rest.

The proper method of meeting these indications varies greatly, not only with the varying physical conditions and idiosynerasies of patients, but also with their diverse domestic and pecuniary relations. To give detailed directions for every case is impossible, and I shall therefore limit myself first to simple cases of brain-tire in which the muscular strength is preserved; second, to cases of profound general neurasthenia.

In brain-tire travel is usually recommended, and travel affords, when properly directed, separation from old cares and thoughts, a maintenance of interest by a succession of novel sights and experiences, and the physical stimulation of fresh air and exercise. In bad cases general travel is too stimulating. Ocean-voyaging gives complete isolation, fresh air, mental stagnation, and, if the patient be fond of the sea, complete enjoyment. Camping in the wilderness offers also all of these advantages, and as a further good the possibility of obtaining exercise in exactly the amount desired. The subject may live in his tent and be nursed and fed by his guide or may do the work of a day-laborer. Quiet travel in the mountainous districts of foreign countries is often very efficient, but sight-seeing, and even visiting cities, must be avoided. The quiet of Switzerland or the Tyrol may bring restoration when the bustle of London and Paris might complete the ruin. In all cases strict attention must be paid

to the individual tastes of the sufferer in deciding what measures should be carried out.

There are cases of neurasthenia in which the slightest exercise, or even the unconscious effort and excitement of seeing personal friends, is an injury. In these cases the so-called "rest-cure" often acts most beneficially. Rarely does it itself give permanent relief, but it often lays the foundation for later complete restoration by means of outdoor life and exercise taken after a certain amount of strength has been gained. A word of caution seems necessary against the routine employed in this rest-cure. It is simply the carrying out of a principle, and although, in the pages of a book like this, it is necessary to give a fixed formula, success in practical life will depend upon the skill of the practitioner in modifying this, and adapting formulæ to the needs of the individual case. The principles of the rest-cure are absolute rest, forced feeding, and passive exercise. The rest must be for the mind as well as for the body, so that in severe cases complete and absolute isolation must be insisted upon; and especially when there is a decidedly hysterical element is it necessary to separate the patient entirely from her friends. Under these circumstances there must be a well-trained nurse who is personally agreeable to the patient. The confinement would be very irksome to any except the most exhausted patient were it not for the daily visits of those engaged in the treatment. To further provide against ennui the nurse should be a good reader, so that under the definite instructions of the physician she can occupy a certain portion of the time in reading to the patient. In the worst cases the patient should not feed himself or herself or perform any of the acts of the toilet. Directly after breakfast the sponge-bath should be given by the nurse, the patient being between blankets. Hot water should be used or hot sea-brine, and after each part has been sponged over it should be momentarily rubbed with a piece of ice, followed by brisk friction with a Turkish towel. The greatest care should be given to the question of feeding. The end to be attained is to give as much food as can be digested without overdoing and deranging digestion. It is usually better to give the food, which must be both light and nutritious, at short intervals. In most cases milk should be used very largely, sometimes exclusively. Often, especially when there is a tendency to obesity or when the digestive powers are feeble, the milk should be skimmed. Frequently koumiss, matzoon, or other fermented milks are advantageous. Rarely peptonized milk may be given. Beef and other concentrated meat-essences are valuable as stimulants, and may be used, especially as the basis of soups. Various farinaceous articles of food may be added to them, or if an egg be broken into the concentrated bouillon or beef-essence just as it ceases boiling, a nutritious, and to many persons palatable, dish is obtained. When constipation exists, oatmeal porridge, Graham bread, fresh or dried fruits may be allowed if readily digested by the patient. In order to give a general plan of the dietary the following schedule of the daily life is given. Such a schedule should always be put into the hands of the nurse, who should be required to follow it strictly. It must be altered from day to day, so as not to weary the patient with monotony. It is especially

important to remember that the diet must be carefully studied for each patient, and be adapted to the individual requirements of the case. Success will in a great measure depend upon the practical skill and tact of the physician in this adaptation:

8 A. M. Rolls or toast; cocoa or weak coffee, or roasted wheat coffee; beefsteak tenderloin or mutton chop.

9 A. M. Bathing.

11 A. M. Oatmeal porridge, with milk, or else a pint of koumiss.

12 M. Massage.

2 P. M. Dinner: bouillon with or without egg; beefsteak; rice; roast white potatoes; dessert of bread-pudding, blanc mange, or similar farinaceous article of diet.

4 P. M. Electricity.

5 P. M. Milk toast.

9 P. M. Half pint of skimmed milk or koumiss.

In many cases the patient at first can take very little food, and it is very frequently best to begin the treatment with an entirely liquid diet, giving milk every two hours or using Liebig's raw-meat soup, with milk or plain farinaceous food, and only after a time gradually accustoming the patient to solid food. Not rarely a prolonged milk-diet is of great service. The rest-cure is indeed largely based upon a careful regulation of the food; but a full discussion of the various dietaries to be used would require a treatise upon dietetics.

Exercise is of value in health by its stimulating the general nutrition, aiding the flow of blood back to the heart, and increasing the excrementitious output from the emunctories. In the rest-cure these effects are obtained in a more or less imperfect manner without the expenditure of the patient's nerve-force by the use of electricity and massage. The electrical current produces not only muscular contractions, but probably affects the tone of the minute blood-vessels. Its action is so decisive that, as has been shown by Dr. S. Weir Mitchell, it will often temporarily elevate the temperature of the whole body. The faradic current alone is used. It is applied in two ways: first, to the individual muscles; second, to the whole body. The séances should be daily, the operator beginning at the hand or foot and systematically faradizing each muscle of the extremities and trunk. The slowly-interrupted current is generally preferable, but advantage is sometimes gained by varying the rapidity of the interruptions. The general rule is to select that current which produces most muscular contraction with the least pain. The poles should be applied successively to the motor points of the muscles, so as to contract each firmly and thoroughly. This process should occupy from thirty to forty minutes. The electrodes are then to be replaced by large sponges well dampened with salt water: one of these should be placed at the nape of the neck and the other against the soles of the feet, and a rapidly-interrupted current, as strong as the patient can bear, should be sent through the body for twenty minutes or half an hour. In some cases the electrical programme may be varied so as to get a local stimulant action from the general current; thus, when the digestion is

enfeebled and the bowels costive, for a portion of the time one of the sponges may be placed upon the epigastric region. In women, when there is great abdominal and pelvic relaxation, one pole may be placed high up in the vagina. I have seen long-standing uterine prolapse cured in this way. Some electro-therapeutists claim great advantage from galvanization of the cervical sympathetic ganglia, but I do not myself believe that they have ever succeeded in reaching these ganglia with the current.

Massage, like electricity, affects greatly the peripheral circulation, empties the juice-channels, and gives tone to the muscular system. It must be clearly distinguished from rubbing of the skin. It consists in manipulations of such of the muscles as are not too deep to be reached, and of the cellular tissue. In order to lessen as much as may be the skin-friction by these manipulations, it is often well to anoint the surface with cocoanut or other bland oil. In practising massage it is essential to remember that the natural course of the venous blood and the juices of the cellular tissue is toward the centre of the body; therefore all general massage movements should be practised in this direction. The manipulations are percussion, rolling, kneading, and spiral. They consist of movements made with the pulpy ends of the fingers and thumbs, and spiral movements with the whole hand so folded as to adapt its palm to the limb. In percussion the strokes should be from the wrist and should be quick and short. It is probably not possible, even by long, strong strokes, to affect deep muscles. In the rolling manipulations the effort is to roll the individual muscles beneath the pulps of the fingers. This manipulation may be varied by pinching the muscles, not the skin, and kneading. In each case it is intermittent pressure upon the muscles that is aimed at. The circular movements are to be in opposite directions with both hands simultaneously, the limb being grasped by one hand a little above the other, and a spiral sweep made up the limb, the ball of the thumb and the palm of the hand resting upon the patient, and the pulpy parts of the thumb and the fingers grasping the limb. It is especially such motions as these which affect the circulation of the flesh-juices.

The length of time in which a patient should be kept in bed varies from three to six weeks. The getting up should be gradual, the time of sitting up and the amount of exercise carefully increased from day to day. The electrical treatment should be rapidly withdrawn, but often massage may be continued with advantage every other day for some time. So soon as can be the patient should be sent out of the city, to consolidate by outdoor life that which has been gained.

Hysteria.

Definition.—A functional disorder of the nervous system, characterized by depression of the will-power, exaltation of the emotional nature, and an infinitude of shifting, polymorphic nervous disturbances more or less clearly simulating various organic diseases.

Etiology.—Although the name "hysteria" is derived from the Greek ὑστερος, a womb, there is no direct connection between the disease and the sexual organs, except only through the tendency of sexual disturbances, and

especially sexual excesses, to produce nervous exhaustion and irritation, which in turn may aid in the development of hysteria. The affection is vastly more common in females than in males, simply because the nervous system of the female is less robust, more excitable, more sensitive, and more readily thrown off its balance than is that of the male. Race and racial habits are of even more importance than sex as an etiological factor. In barbarous countries the disease is practically unknown. In Northern races, with a tendency to phlegmatic temperament, hysteria is comparatively infrequent and of minor severity, whilst the mobile Southern temperament favors its development. Thus the Latin races, as exemplified in the French and Italian, are much more hysterical than the English and Teutonic, and in the extreme southern portion of the United States, where the Latin blood predominates, severe hysteria is much more frequent than in the North.

In the majority of cases the disease first manifests itself between fifteen and twenty-five years of age, but it is not rare before puberty, and occasionally occurs even in very young children. I have seen it in young boys as frequently as in young girls. In boys it is often connected with or dependent upon masturbation, adherent prepuce, or other irritation of the sexual organs.

The influence of heredity, especially neuropathic heredity rather than direct heredity, in the production of hysteria is very great, whilst education and habits of life are almost equally powerful. Luxury, license, and indulgence during childhood, indoor rather than outdoor life, any method of education or of life which renders the nervous system more sensitive and less robust, tend very strongly to the development of the hysterical temperament.

Hysteria may unexpectedly appear as the result of nervous exhaustion produced by overwork, depressing emotions, long-continued severe pain, or exhausting dissipation. Moral influences are often very effective in their action in persons of nervous temperament. The disease not rarely illustrates the contagiousness of example: a single hysterical patient will sometimes inoculate a whole school, infirmary, or hospital ward, transforming, it may be, the quiet retreat or educational institution into a pandemonium of nervous explosions. During the Middle Ages, when by misery, poverty, and religious excitement the ground had been especially prepared, whole communities became involved in epidemics of hysterical madness; hence the Flagellants, Children's Crusade, etc.

When the hysterical temperament exists, local injury or local disease is prone to bring about a local hysteria in the affected part, and when the original local disease is of such character as to wear heavily upon the general nervous system, local hysterical manifestations may develop after an organic lesion or disease in a person who previously had not shown any distinct hysterical symptoms.

Symptomatology.—The symptoms of hysteria are so infinite in their number, their variety, and their collocation, the history of hysterical cases is so widely diverse, the course of the disease is so absolutely without rule, that it seems impossible to give any concise description of the affection within the

space allotted in this volume. It must be remembered that hysteria exists in nature in every possible degree, and that the majority of cases, as seen in this country, are those of minor hysteria, grading up from the slightest hysterical tinge in temperament or in disease. It must also be borne in mind that the ingrained hysteria due to heredity is far deeper in its seat than what may be known as accidental hysteria; that is, the hysterical condition developed by the accidents of life. Under the latter circumstances the ordinary signs of hysterical temperament are often wanting.

Hysteria often reveals itself in certain physical peculiarities: the large, full, liquid eye, the mobile pupil, the clear skin, the vivacious movement of ever-changing whims, or the slow, languid movements of the self-conscious beauty may furnish unmistakable signs of the hysterical temperament.

Mental Symptoms.—The basis of the hysterical character is selfishness—a selfishness which sometimes shows itself in the indulgence of the grosser appetites and desires, but which more commonly seeks self-gratification in applause, in admiration, and in being the centre of sympathetic attention. Indeed, this selfishness often leads its possessor to great lengths of apparent self-sacrifice, the desire for praise and attention overmastering sensibility, pain, and even present contumely. The hysterical woman is self-conscious and self-centred, dwelling ever on her own personality, its needs, its wishes, its life, its ailments, its everything. Excessive sensitiveness in all that regards herself is the natural outcome of this mental state. Self-indulgence goes hand in hand with self-consciousness, while the will is without power to assert itself and the individual knows not at all the path of self-control and true self-sacrifice.

The weakness of the will is not the cause of the mental attitude, but only one of the concomitants: unwillingness to make the effort necessary for self-control leaves the individual largely to the play of outside forces, especially when these forces touch aright the dominant chords of character. Hence, suggestions of conduct, sympathy with suffering, and even questions as to symptoms, have an inordinate influence. The pain that is not, when asked for soon becomes.

The morbid desire for attention and sympathy leads to intentional simulation of disease, so that the hysteric will pretend what does not exist, but the mimicry of disease in hysteria has in the majority of cases a deeper seat than this. It is an unconscious simulation. Not merely do emotions dominate the forces of life, but ideas formed in the mind may express themselves in a physical enactment of a disease which has been thought of. The dread of some disorder, or of the disablement and suffering which it causes, is almost as powerful as desire in multiplying the symptoms of an hysteria.

A very extraordinary rupture often takes place in hysteria between consciousness and will. Thus I have seen a patient declare that she could not see with the left eye, and respond correctly to every test for the existence of blindness in the affected eye until the separate prisms were used after she had been told that prisms make a person see double with one eye. Under this belief the double images were seen and located correctly, proving that there

was vision all the time with the left eye. I do not believe that the patient purposely lied throughout, but that the belief that she could not see in the left eye so dominated conscious perception that there really was no consciousness of the image until belief in that image was established.

Emotional instability, lack of control of the will over the emotional nature, is one of the most characteristic manifestations of the hysterical state. With or without reason, but always without control, the subject laughs and cries, the emotional storms rising rapidly from the most inadequate causes.

The desire for sympathy leads always to exaggerated statements, and in the examination of an hysteric this must always be borne in mind. It leads also to innumerable forms of deception. Thus I have known an hysterical woman to raise an alarm in her country-house, and be found upon the ground beneath an open window, apparently greatly injured by the fall which she asserted she had received during a somnambulistic walk, when in truth she had simply walked out of the door and laid upon the grass. Especially common is it for these simulated symptoms to take a shape that will bring great personal attention by the young and inexperienced physician, and mayhap minister to the morbid sexual desire of the patient. To swallow pins and needles, or to thrust them into the tenderest parts of the body that they may be withdrawn by the doctor, is common enough. To retain urine, with absolute recklessness of the suffering involved, for two or three days, that the catheter may be used, is very frequent. I have seen the rectum and lower large intestine secretly filled day after day with starch jellies, to the utter astonishment of the practitioner, especially when the true nature of the fæcal discharge was revealed by the microscope of the consultant. A very common trick, which imposes with extraordinary frequency upon the credulity of doctors, is the placing of small bones by the woman over night in the uterus, to be removed by the doctor the next day as parts of a dead fœtus.

Disturbances of Consciousness and Motion.—Besides the major and minor hysterical paroxysms, spasms, choreic movements, and paralytic disturbances occur in hysteria. The hysterical spasms may be localized in any portion of the body; the choreic movements are sufficiently described elsewhere in this book. (See page 634.)

Into *minor* hysterical paroxysms enter all the elements of the major affection, but usually some of the symptoms are wanting in individual attacks, and not rarely a single stage constitutes the whole paroxysm. The aura is not usually present, unless the so-called *globus hystericus* (a sense of constriction or the rising of a ball in the throat) be considered to represent it. The emotional state is usually well developed, and is especially prone to express itself by uncontrollable laughter or equally uncontrollable sobbing or crying. A very characteristic performance which I have seen, especially in children, is that which may be termed *beast-mimicry*, in which the patient bites or snaps or snarls like a dog or crows like a cock, or in some other way imitates the movements and the vocal acts of the lower animals. Among these cases belong the not rare attacks of *spurious hydrophobia*, in which, either with

or without severe general convulsion, the subject shows profound dread of water, great emotional disturbance, often crying out to be held lest he bite some person, and continually snarls and barks and attempts to bite. These symptoms do not closely resemble those of true hydrophobia, in which disease the subject never offers to bite, and does not make any noises resembling those of the dog or any other lower animal. Beast-mimicry may be considered as diagnostic of hysteria.

The convulsive symptoms of minor hysteria are tonic rather than clonic. More or less persistent rigidity is very frequent and very characteristic. It may last for hours or may pass by in a few moments. The disturbances of consciousness are similar to those of major hysteria (see page 598), only usually less severe.

In the *major* hysterical convulsion the tendency is to rigid contractions of muscles which lock the body in positions like those of voluntary life. Consciousness may be abolished, but is usually only perverted. Thus a patient, apparently unconscious during the fit, narrates after recovery all that has occurred during the paroxysm; or there may be the so-called *automatic* consciousness, in which the patient during the paroxysm seems to understand all that is said, but nevertheless after the paroxysm has no remembrance of what has taken place.

Commonly the major convulsion is preceded by some warning, such as a special feeling of malaise, epigastric sensation, palpitation of the heart, giddiness, globus hystericus, or an aura which appears to arise from a hyperæsthetic ovary. The patient falls, but usually gently and not with the suddenness of true epilepsy. Not rarely there is at this time an initial scream, which may be repeated during the paroxysms. The pallor of the face may now be marked. A simple tonic spasm develops, lasting two or three minutes. In it the limbs are usually rigid, with the toes pointed downward and the arms extended or lying at the side of the patient. It is at this period that the respiration becomes arrested, and there is developed the stage of asphyxia of some writers. The face is swollen, with turgid veins, and suffocation seems imminent. This condition may be followed by a furious clonic convulsion, in which bloody foam gathers about the mouth, although the movements preserve, to some extent, the appearance of wilfulness, and the head or the arms are struck violently and with seeming purposiveness against the floor or dashed against pieces of furniture. Following these clonic convulsions, or not rarely replacing them, is the characteristic stage of opisthotonos, in which the person lying upon the back is bent violently into the arc of a circle, so that the body rests upon the head and feet, with the central portion arched from the ground. The muscular contractions may be so severe that the head is drawn completely backward and the upper portions of the body rest upon the face, which looks toward the floor, whilst the lower end of the arc is supported on the toes. This condition of opisthotonos may last for some minutes. In some cases it is interrupted or replaced by violent purposive clonic spasms, the patient suddenly leaping from the bed or rising into a sitting position, and

as quickly falling back again in opisthotonos. This to-and-fro movement may take place with extraordinary velocity. In some cases the body is bent violently laterally instead of backward. The opisthotonic stage may be interrupted by various emotional actions, or it may gradually subside into what may be called the emotional stage, when the patient assumes some attitude of intense emotion, and not rarely the so-called posture of the crucifix, in which the subject lies upon the back, absolutely quiet, with the legs stretched out side by side and the arms firmly extended at right angles to the body in the position of a cross. The widely-opened eyes, with dilated pupils, appear to be looking into indefinite distance, whilst a beatific smile is settled upon the face, so that by the ignorant the convulsant is often believed to be seeing visions of heavenly joy. Usually the emotion changes from time to time: the light of religious beatitude upon the countenance deepens into an intense voluptuousness, attended, it may be, with lustful words and gestures; or terror becomes supreme, and is manifested with equal intensity; or, in a passion of penitence, the convulsant, with sobs, bitter cries, and broken words, begs for mercy. Again the scene shifts, and, now singing, now weeping, reproaching alternately herself and her care-takers, the woman passes on to a slowly-perfected consciousness.

Hallucinations occur during and after the fit, and are always correlated to the emotional state. Thus during the terror the subject sees rats and other disgusting objects, which, according to Charcot, are usually upon the side that is anæsthetic between the paroxysms.

The character and mental states of the confirmed hysteric approach in many respects those of a paranoiac (see Hysterical Insanity, p. 558), whilst the delirium of a major hysterical paroxysm may simulate an acute mania. I have indeed seen recurring attacks of hysterical epilepsy replaced by a furious outbreak of acute mania, lacking in none of the symptoms characteristic of that disease. It seems to me that in such a case the maniacal explosion must be looked upon as the direct outcome of the hysterical neurosis, and that therefore the existence of an hysterical acute mania not in itself distinguishable from ordinary acute mania must be acknowledged. In most cases in which such maniacal symptoms exist the neurosis is so thoroughly engrafted upon the constitution that permanent recovery is not possible, the patient during life suffering from various forms of hysterical attack, and being always possessed of the peculiarities which have already been spoken of as characteristic of the hysterical temperament. Hysterical symptoms may occur during almost any form of insanity, but do not warrant our looking upon such a melancholia or mania, or whatever form the affection may take, as hysterical, scarcely more than we should be warranted in considering pneumonia when associated with hysterical symptoms as hysterical. At the same time, the relation of the hysterical temperament to monomanias and to general insanities is distinct; and, according to my belief, it is entirely possible for any form of insanity to be simulated by symptoms which have their origin in the original faulty organization that is the basis of chronic hysteria; moreover, such faulty nerve-

organization is closely allied to the peculiar neurotic temperament which is the basis of much insanity.

Closely allied to the major hysteria is *hysterical somnolence*, which may take the form of a *true narcolepsy* (the patient being continually drowsy, falling asleep at all times, but passing only the nights in profound slumber), or it may assume the shape of the lethargy or trance.

Hysterical trance usually, but not always, commences with marked hysterical symptoms which leave the subject in absolute repose. The face may be red and hot, especially in the first days of the attack, but usually it is pale. The pulse at first may be regular and slow, but after a long sleep it is rapid and feeble. The respirations, generally quiet, may at times become hurried, irregular, and even stertorous. In severe cases the movements of the thorax may be so slight as to be traceable with difficulty. The muscular system, often thoroughly relaxed, may be rigid, and in many cases muscular relaxation alternates with muscular contractions or even contractures. The eyes are opened or closed; very frequently minute tremors affect both the lids and the eyeballs. The jaws are often set, and sometimes an excess of saliva, or even foam, gathers about the mouth. In the profoundest cases there is complete anæsthesia of both the common and the special senses, so that neither pinching nor cutting, neither cold nor heat applied to the skin, elicits response. The pupils are usually dilated, and often respond to a powerful light, which, however, calls forth no other signs of life. Sometimes the patient can be readily fed by means of a spoon, but generally in severe cases it is necessary to use the œsophageal tube. Usually digestion is good, but the stools are at long intervals and scanty. The urine is in most cases scantily excreted and is passed involuntarily. Considering the small amount of nourishment taken, the bodily nutrition is often surprisingly maintained, but in prolonged cases there comes, sooner or later, great emaciation. The bodily temperature may in the earlier parts of the attack be somewhat elevated, but ordinarily it is distinctly subnormal. The awaking is usually, but not always, sudden. During the course of such a lethargy the subject may pass into a condition which has been mistaken for death. The bodily temperature falls, the respiration becomes so passive that no movement of the thorax or abdomen is perceptible, and, unless a feather or other light object be held over the mouth, breathing may seem to have ceased. The beats of the heart diminish in frequency and in force, so that they become imperceptible even upon auscultation. The face takes on the waxy whiteness of a corpse. The muscular system is in complete relaxation, the dilated pupil no longer reacts to light, and even the cornea is filmy as in a corpse. This death-like condition may last for only a few hours, or may continue during from one to several days, after which, little by little, respiration and circulation are re-established. After such a crisis the subject may awake immediately or pass into a new sleep.

Catalepsy is a form of hysterical lethargy characterized by the peculiar condition of the muscles, owing to which the body or the limbs remain for an indefinite time in any position in which they are placed. It may come on

gradually or abruptly as the result of a powerful emotion, but usually develops during a lethargy, the paroxysms being of irregular duration and sometimes continually recurring. The facial expression may be that of apathy; in some cases it is that of devotion, of rage, or of whatever passion the subject was in at the time of the fixation of the muscles. The eyes are wide open, with quiet lids. The body is motionless in the posture in which it has been placed or in which it has settled during the arrest of active motion. There is no power of voluntary movement, but the limbs are not rigid or contracted. When taken hold of they bend with the plasticity of wax. In any position in which the body or limbs are placed they remain for a long time. Berger (quoted by Barth) is said to have seen the most bizarre and difficult attitudes steadily maintained for seven consecutive hours by a young cataleptic woman who was constantly under observation. During the whole of the cataleptic state there is complete anæsthesia of both the common and the special senses, so that the most violent irritations of the skin produce no reaction. Respiration is regular, the pulse maintains its normal rhythm and rate, and the general bodily functions appear to go on unaffected.

If the patient be regularly fed with liquid food at intervals, hysterical sleep may last uninterruptedly for many weeks, months, or even years. Sometimes the patient will occasionally wake to take food.

Hysterical Paralysis may simulate almost any form of organic palsy. Paralysis of the whole body is exceedingly rare, but such cases are reported. The face also is not often affected, and the ocular muscles usually escape. Nevertheless, hysterical strabismus and hysterical inequality of the pupil are occasionally seen. Hysterical monoplegia is not frequent; hysterical hemiplegia is very common, but the most frequent variety is hysterical paraplegia.

Hysterical Paraplegia may coexist with muscular relaxations or contractions (see page 596), with normal, abolished, or exaggerated knee-jerk, and even with ankle-clonus. The sensory nervous system may or may not participate in the disturbance—in some cases there is excessive hyperæsthesia, with or without pain; more frequently the sensibility is lessened or abolished; usually the muscular sense is at least as much affected as is cutaneous sensibility. Sometimes electro-sensibility is abolished. If true girdle sensation ever occur in hysterical paraplegia, it must be very rare: the real, not suggested, presence of such a sensation is almost pathognomonic of organic disease.

In *Hysterical Hemiplegia* one extremity is in most cases distinctly more affected than the other, and the face is very rarely implicated; the presence of facial palsy tells strongly against the probability of an hysterical origin. The palsy is rarely complete, so that a patient unable to walk or even stand may be able to raise the foot when in bed. There is usually, but not always, a more or less pronounced loss of sensation in the paralyzed part, and the coexistence of a hemianæsthesia with hemiplegia should always arouse suspicion.

Hysterical Disturbances of Sensation may take the form of hyperæsthesia, anæsthesia, or paræsthesia. *Hysterical Hyperæsthesia* may follow the regional distribution commonly seen in anæsthesia, but is usually irregular in its dis-

tribution and often interrupts anæsthetic tracts. Certain local hysterical hyperæsthesias are so important as to require special notice. Hyperæsthesia in the genitals is very common in the female, is usually associated with loss of sexual desire, and commonly lies at the foundation of the condition known as vaginismus, in which any attempts at coitus produce an overpowering vaginal spasm. Hyperæsthesia of the mamma is usually attended with swelling, excessive tenderness, and violent pain, sometimes shooting down the arm. (See Diagnosis.) Hyperæsthesia with vaso-motor swelling, and even true exudation, may in almost any of the larger joints mimic organic disease. (See Diagnosis.)

Hyperæsthesia of the special senses is a very common hysterical symptom: especially is photophobia both frequent and severe. Indeed, photophobia without distinct disease of the eye is almost always hysterical. A special-sense hyperæsthesia may show itself simply in the pain caused by the natural stimulus of the affected organ, but may also take the form of a true functional exaltation, so that vision or hearing becomes much more acute than normal. In my experience this form of hyperæsthesia has been especially frequent in regard to hearing. Not rarely, hysterical women will understand and repeat conversations spoken in apartments at such distance from their own that the ordinary ear catches no sound.

Hysterical Anæsthesia may exist in any portion of the body, but in the majority of cases it takes the form of hemianæsthesia. This hemianæsthesia is apt to be interrupted by spots of hyperæsthesia, especially in the region of the groin or in the ovary itself, or in the dorsal and lumbar regions posteriorly, or in the limited vertical space from one to two inches wide stretching from the lower cervical region upward. The hyperæsthesia in these cases may be superficial or may be only elicited by deep pressure.

In neurasthenic women, especially young women, hyperæsthesia or superficial tenderness all over the vertebral column is so common, and exists in so many cases without very pronounced hysterical symptoms, as to be commonly spoken of as a disease, the so-called *Spinal Irritation* or *Spinal Anæmia*. There is not, however, any reasonable foundation for the theory that the condition depends upon anæmia or any other recognizable lesion of the spinal cord, or that it is entitled to a distinct place in nosology. The cases shade from the slightest form of vertebral tenderness to major hysteria.[1]

Hysterical anæsthesia may be limited to one organ, like the cornea of the eye, may involve the mucous membranes as well as skin and deeper tissues, and may be complete or incomplete. Thermo-anæsthesia is common, whilst analgesia, existing by itself, is almost invariably hysterical. Hysterical anæsthesia is usually accompanied by the so-called *ischæmia*. In this condition the surface is pale and the needle prick or even an extensive superficial

[1] The treatment of spinal irritation is that of neurasthenia and a mild hysteria. It is true that sometimes benefit is obtained by local appliances of belladonna plasters, or even by mild blistering, but it is impossible to determine how far this treatment acts through expectant attention and how far it has any direct influence.

incised wound does not bleed. Anæsthetic ischæmia appears to be specially pronounced in the violent epidemic forms of hysteria, such as occurred in the *Convulsionnaires* of the Middle Ages; hence the miracle that superficial wounds were not followed by loss of blood.

In hysterical hemianæsthesia the special senses are usually affected, and there may be loss of hearing, smell, taste, and vision. Usually, however, the special senses are not completely set aside. *Amblyopia* is commonly shown by a concentric narrowing of the field of vision and a peculiar loss of color sense, the *achromatopsia* of Galezowski. In some cases the power of seeing the colors is entirely lost, so that all objects appear of a uniform sepia tint. When the achromatopsia is not complete the colors disappear in a constant order. The first color that an hysterical person ceases to see is violet: usually, but not always, blue is lost before red, the intermediate tints fading out in regular succession.[1]

The *digestive*, *circulatory*, and *respiratory* systems are frequently deranged in hysteria.

Cardiac irritability is very common, the slightest emotional or other excitement producing violent tumultuous palpitation, which in some cases is accompanied by great discomfort, and even irregularity and interruption of the heart's action, with more or less cardiac distress. In some cases a violent pain in the cardiac region, shooting down the arm, closely simulates angina pectoris, the simulation being rendered more complete by the excessive rapidity and smallness of the pulse. I have seen this pseudo-angina pectoris more frequently in young men than in other hysterics, and when in such cases the general signs of hysteria are not pronounced a false diagnosis may readily be made. "Flushings," with a sensation of intense heat and a sudden outburst of perspiration, occur in hysteria, but are especially connected with the climacteric period in women. Unilateral flushing, local œdematous swellings, and similar phenomena, though rare in hysteria, demonstrate the possibility of local vaso-motor disturbance.

Possibly as the result of vaso-motor relaxation are the hæmorrhages from the nose or stomach, which are specially prone to be severe when menstruation is suppressed. Care is often necessary to avoid mistaking for a true hæmoptysis the bleeding which a designing woman produces by sucking or otherwise irritating the gums.

[1] It seems necessary to advert to the so-called metallo-therapy first originated by Dr. Burk, who found that different hysterical individuals have such relations with different metallic substances that when a small disk of the appropriate substance is bound over the anæsthetic part a sensation of warmth, with slight redness and disappearence of the ischæmia and anæsthesia, follows. In some cases not only is the sensibility of the skin restored, but if the plate be in the neighborhood of the orbit, vision returns. Also, often the so-called "transfer" phenomena appear—*i. e.* loss of sensibility and ischæmia develop upon the unparalyzed side in the position corresponding to the seat of the metallic application. Powerful magnets have been found to have a similar influence to the metal, and even disks of wood have been successfully employed. It is very difficult in this country to obtain transfer phenomena, and it seems almost certain that they are the result of expectant attention.

The bodily temperature rarely departs from the norm in hysteria, and in simulated acute disease advantage can often be taken of the fact for diagnostic purposes; nevertheless, hysterical fever does occur. According to M. Briand and other French writers, there are three types of it—in the first form the paroxysms are irregular, of long duration, accompanied by various nervous disturbances; in the second variety the fever continues from one to four weeks and is accompanied by disturbance of the nutrition, in some cases the whole course of the affection closely mimicking a typhoid fever; in the third form the paroxysms of fever occur with more or less regularity, so as to give the appearance of a true intermittent fever. Exaggerated temperatures, 120° or 130° F., have been recorded from time to time as occurring in hysterical patients. Most, if not all, of these high records have been due to skilful manipulation of the thermometer by a designing patient, but there is reason for suspecting that extraordinary local elevations of temperature happen in hysteria.

Hysterical disturbances of respiration are common. Intensely rapid breathing, 50 to 150 per minute, the so-called "*hysterical dyspnœa*," may occur without alteration of the pulse-rate, and may be associated with thoracic symptoms misleading to the inexperienced practitioner. Hoarse, croaking, laryngeal cough, seemingly almost uncontrollable, is a frequent hysteric phenomenon, as is also aphonia from laryngeal palsy. Violent paroxysms of acute dyspnœa may occur from hysterical laryngeal spasm, simulating attacks of true laryngismus stridulus.

Secretion is often affected in hysteria. Excessive sweating is very common, and a profuse sweating may be accompanied with a sufficient hæmorrhage to color it deep rose-red (*hæmatidrosis*). One of the most characteristic symptoms of an hysterical paroxysm is the free discharge of limpid, light-colored urine, evidently due to vaso-motor and secretory disturbances in the kidneys. More serious is the partial or even complete suppression of urine (*anuria*), which may for many months almost completely prevent the excretion of the urinary solids through the normal channel. Under these circumstances the sweat, the vomit, and other secretions become loaded with urea.

Disturbances of digestion are almost universal in hysteria. Constipation is very common and not rarely very obstinate. Diarrhœa is more rare. Flatulence, gastric and intestinal, is sometimes accompanied by extraordinary distension of the bowels, and occasionally by irregular spasmodic contraction of the abdominal muscles, producing strange internal noises. Hysterical œsophageal spasm may for a while prevent swallowing, whilst hysterical vomiting is one of the most frequent of symptoms. This vomiting may be excessive and continue for days and weeks, so severely and so persistently that the patient seems to retain no food whatever, the appetite being replaced by an absolute loathing for food. Such cases constitute the so-called "fasting girls" who from time to time become the centre of attention and wonder on the part of whole communities. The ability of the hysteric to live upon the smallest quantity of food is often extraordinary, but there can be no doubt that

in the notorious instances of alleged fasting deception has played an important rôle. The vomiting may be accompanied by reversion of intestinal peristalsis, resulting in the discharge of fæcal matter from the mouth, and cases are on record in which rectal injections were in a short lime thrown up from the stomach.

Diagnosis.—The diagnosis of major or minor hysteria occurring in general paroxysmal form requires no further discussion. The recognition of the true nature of an hysterical paralysis may be very difficult.

The presence of other distinct symptoms of hysteria, either in the past or in the present, is of importance. Nevertheless, a violently hysterical person may be attacked by organic palsy, and I have also seen hysterical paraplegia occurring without other symptoms of hysteria and without an hysterical history that could be made out. The hysterical palsy is apt to be transient and shifting in its character, to go and come suddenly, and not to conform in its minor phenomena with the sequences and coincidences of organic palsy. Again, the hysterical palsy is often accompanied by symptoms that do not occur in the organic paralysis which is simulated. Thus an hysterical hemiplegia or a monoplegia may be attended with paralysis of the bladder, of the intestines, or of the rectum, although paralysis of the visceral walls is very rarely if ever present in organic hemiplegia or local paralysis; or an hysterical hemianæsthesia is not properly situated in its relations with the coexisting motor palsy; or electro-sensibility is lost when general sensibility is preserved, etc. etc. An atypical paralysis should always be viewed with suspicion—in women it is usually hysterical; in men it is usually syphilitic, but may be hysterical.

An hysterical monoplegia is not infrequently attributed to an injury. If contractures come on immediately after a real or an alleged injury, the paralysis is probably hysterical; but complete relaxation may exist in an hysterical monoplegia. When after a traumatism the paralysis and the relaxation are complete and there is no wasting of the muscles, the affection is usually hysterical, since in all cases of total or nearly total loss of power from injuries to a nerve the muscles rapidly change. Irregularities in the anatomical relations between the disturbances of sensibility and the alterations of mobility indicate an hysterical origin, but these relations may, in hysteria, conform to the organic type.

In consentaneous organic palsies of sensation and motion sensation almost always improves first—in mimicking hysterical states motion usually improves before sensation. In organic hemiplegia aphasia is frequent; in hysterical it is very unusual.

Hysterical affection of the larger joints sometimes so closely simulates chronic inflammation as to make the diagnosis a matter of some difficulty. The presence of other hysterical symptoms is important, and usually the true nature of an hysterical joint can be recognized by attention to the following considerations: first, the muscular rigidity or contraction can be overcome by mildly persistent efforts while the patient's mind is diverted, yields readily

during natural sleep, and disappears during slight anæsthesia or even under a full dose of chloral or opium; secondly, there is no rise in temperature in the joint, although the part looks red and inflamed; thirdly, the reaction of the contracted and apparently atrophied muscles to the faradic current is normal.

The knee is the part most frequently implicated, but mimetic disease of the hip-joint is especially misleading. It should be noticed that the limp varies from day to day as the patient's attention is directed to or diverted from the joint—that it is exaggerated by fatigue and nervous exhaustion, and hence is usually more pronounced in the evening than in the morning. Moreover, in the onset of an organic disease the patient usually begins to limp before he complains of pain, whilst in the hysterical disorder pain generally appears first.

Hysterical lateral curvature is especially prone to deceive practitioners, owing to the fact that true lateral curvature is very frequent in neurasthenic women. The hysterical curvature, being the outcome of spasm, disappears during anæsthesia; the organic lateral curvature remains unchanged by the anæsthetic.

The nature of the hysterical breast is to be recognized by the excessive superficial tenderness, so that merely brushing or handling the breast causes as much pain as hard pressure; by the diffusiveness of the swelling; by the constant variation in size and in hardness; and by the recurrence of the symptoms at the menstrual period, at the approach of stormy weather, or after general fatigue. Not rarely in neurotic girls, and sometimes in neurotic boys, at the period of sexual unfolding one breast will suddenly become hot, exceedingly painful and tender, and perhaps secrete a few drops of sero-lacteal fluid.

The so-called *phantom tumor* of hysterical women is a localized swelling in the abdomen, probably the result of local muscular spasm. The sensation imparted to the fingers may be exactly that of a hard tumor. Usually the presence of percussion clearness renders the diagnosis of the phantom tumor easy, but in obese women this sign may fail. The true nature of the phantom tumor is always revealed by its disappearance during anæsthesia.

Great care is sometimes necessary to prevent mistakes when organic nervous disease develops in nervous women suffering from hysteria. Among the most difficult cases that I myself have ever met with are those in which inherited syphilis has just revealed itself after puberty in an hysterical girl. Basal meningitis, poliomyelitis, myelitis, or any organic disease may develop in an hysterical person, and unless cases be thoroughly examined grave errors will be made. I have seen the diagnosis of major hysteria persisted in by good medical practitioners up to within a few hours of the death of a patient, when an examination of the urine would have demonstrated the uræmic nature of the disease. Usually, watchfulness will result in the detection of choked disk, trophic change, or other unmistakably organic symptom.

It is usually easy to recognize the nature of an hysterical anæsthesia through the existence in the past or present of other hysterical manifestations,

and especially by the fact that frequently when motor and sensory paralyses coexist, they do not conform in relative position to the organic type; further, the organic anæsthesia is fixed and does not vary from time to time in its limits, as does the hysterical anæsthesia; and the organic anæsthesia is not interrupted by islets of normal sensation or of hyperæsthesia, as is the hysterical disorder.

The diagnosis of hysterical *blindness* can often readily be made by noting the absence of the causes of organic blindness and the presence of hysterical manifestations, with the conformity of the amblyopia to the hysterical characteristics already given. (See page 594.) A simulated monocular blindness can usually be detected by means of the Graefe prism test: if a prism held before the eye in which sight is admitted cause double vision, or if, when its axis is held horizontally, a corrective squint develop, vision exists in both eyes. Another equally certain test is to let the patient read with both eyes at sixteen or twenty inches, and slip a glass of high focus in front of the eye alleged to be sound. If the reading continue under these circumstances, the amaurosis is feigned. L. Müller's test for mimetic *deafness* in one ear is to have different words spoken simultaneously in a low voice in two tubes, each of which is connected with one ear of the patient. If the apparent deafness be real, the patient will only repeat that which is spoken into the healthy ear; if there be simulation, confusion and repetition of the words spoken into the alleged deaf ear result. In any case of simulated paralysis of special sense betrayal will often occur during the semi-conscious stages of artificial anæsthesia.

Prognosis.—Death from hysteria is almost infinitely rare, although Sir William Gull has described under the name of *anorexia nervosa vel hysteria* a condition with great emaciation, feeble pulse, fading respiration, and low temperature in young patients, ending sometimes in death. It is hardly probable that these cases were purely hysterical. Proper forced feeding will almost invariably prevent a fatal termination in hysterical patients who refuse food.

When the hysterical temperament is once thoroughly developed, it is rarely if ever set aside completely, though it may be held in abeyance. The chances of complete recovery are therefore much smaller in those cases in which by original inheritance or by faulty education the person has become an ingrained hysteric than in those cases in which by transitory emotional pressure, excessive mental and emotional labor, or by other active cause a previously and inheritedly feeble nervous system has been merely for the time being thrown off its balance.

Pathology.—Hysteria is based upon no anatomical peculiarity of the nervous system sufficiently gross to be recognized by any test that we are at present capable of applying. Theories almost innumerable have from time to time been suggested to explain the phenomena, but in accordance with the general rule followed throughout this work no discussion of these theories will be here entered upon. All that we know is that hysteria is a neurosis—*i. e.* a peculiar nervous state which may be the result of inheritance or of more temporary causes.

Treatment.—For the purposes of discussion the treatment of hysteria naturally divides itself into preventive and curative.

The preventive treatment of hysteria consists in the proper education of the young, it being possible to largely overcome the results of inheritance if the attempt be begun early enough. In a volume like this there is not space for the consideration of the details of this subject, but certain general principles which ought to guide all efforts can readily be stated. The attempt should be —first, to increase the robustness of the whole person, and especially of the nervous system; second, to reduce excessive sensitiveness by accustoming the nervous system to moderate exposure and hardships; third, to develop in the child the habit of obedience (first to those who are above her, and afterward to her own personality, led by a sense of right and wrong; in other words, to teach the young child the habit of subjection to control from without, in order that the power of self-control from within may later be developed); fourth, to bring about so much of intellectual development as shall give to the patient abundance of interest outside of herself and her immediate surroundings, and shall form a basis for character; fifth, to inculcate unselfishness and to develop other traits of character, such as are recognized as worthy of imitation throughout the world. The when, the where, and the how these things shall be done depend upon the circumstances of the individual child. Country life is usually preferable to city life; a moderate living to the home of luxury; home training to training in boarding-schools or other institutions; plain food to high living.

But the environment of the individual patient may change these things. Thus if the mother in the home be weak and hysterical herself, it is essential that some one else guide the young life. Again, if it be impossible for the child to be reared in the country without its leaving a home where all the influences trend toward good, it may be better to sacrifice the country life and to attempt to gain its advantages by gymnastics and athletic outdoor sports.

In the treatment of developed hysteria it is essential to remember that in the majority of cases the hysterical person is a neurasthenic, and that the basal treatment in most cases must therefore be that of neurasthenia. The rigidity with which this treatment must be enforced depends upon the needs of the individual case, but the removal from home, the putting to bed, the whole course of the so-called rest-cure by means of the isolation it requires affords opportunity for that domination and control by the physician and nurse which are so necessary in the treatment of hysteria.

The success of the moral treatment of the hysteric depends upon the tact of the physician and of the nurse; and, as the latter functionary is in continual contact with the patient, she is very important. Unless the nurse be properly selected with view to the work at hand, all efforts at cure must fail. The object of the moral management is to develop, first, a willingness to be unselfish; second, the habit of self-control. In some intelligent hysterics a careful, skilful putting before them of their own nature, of its difficulties, dangers, and possibilities, has a most happy effect, but great care is necessary in the selection

of the individual to be managed in this way. Very frequently such treatment will do harm. In more severe cases absolute control from outside is necessary: the habit of obedience or submission, once formed, becomes the basis of advance of character. The first thing to do is to let the patient see that complaints will not bring sympathy, but will rather excite disgust in the mind of the hearer and disregard for the patient. The next point is to make the hysterical attacks as disagreeable as possible to the patient. This is not simply because the attacks may be simulated or brought on by a direct effort of the will, but that a motive may be furnished the patient which will aid the will in preventing an attack of hysteria. The hypodermic injection of apomorphine will usually cause vomiting and arrest of an hysterical paroxysm, and where the attacks recur at short intervals the apomorphine treatment is often advantageous. When a hospital resident physician I found that in women who were brought in from the street by policemen the production of a pair of shears and the commencing of cutting the hair off, preparatory, as was loudly stated to the policeman standing by, to the putting of a blister on the scalp, would have a more quieting influence upon a furious hysterical paroxysm than would the efforts of several strong men. Even though the patient seemed unconscious, invariably the convulsive movements and the delirium ceased. In an epidemic of hysteria which occurred in one of our Philadelphia charity schools two most obstinate cases resisted the cold douche, blisters, and even the hot iron: no procedure had the slightest effect in preventing the continual recurrence of beast mimicry and other hysterical paroxysms. The two children were finally kept without food for three-quarters of a day, and then fed as much as they would take. To one of them, in the presence of the other, was then given ether, as slowly and as disagreeably as possible, so as to provoke screaming, fighting, and excessive vomiting. This put an end to the symptoms in the one that suffered and the one that witnessed the suffering.

The effect of a motive in hysteria is sometimes most extraordinary. The well-known sudden restorations of power to limbs that have been palsied for years, or to patients that have been bedridden for decades, through strong religious excitement and faith, are supplemented by cases in which the emotion has been fear or rage. I have known a woman who had been motionless in bed for many years, enraged by the treatment of her physician, leap from the bed and tear the clothes off his back before she could be restrained. These cases illustrate, however, rather the result of emotional excitement than of a motive. Not so an instance in which a poor but very beautiful hysterical girl, engaged to a rich man, was informed by the latter that though he loved her he could not live with a woman who vomited continually, and that the marriage would have to be postponed until the vomiting ceased; and behold! how soon that incoercible vomiting ceased! In this case the patient did not vomit purposely, but the strongly excited will finally controlled the stomach.

In endeavoring to carry on the moral management of the hysterical patient it is essential that the physician do not intensify the symptoms by too much attention. The hysterical woman often craves for medical sympathy, and

occasionally has toward the doctor distinctly sexual feelings, so that caution is sometimes necessary not to do her more harm than good. In those cases in which paralysis, vomiting, or other active functional derangement exists the patient should be taught that constant effort will often overcome the evil.

Hypnotism is sometimes of great value in the treatment of hysterical symptoms, but the greatest care is necessary in using the method, as the attention which it brings to the patient and the state itself may do grave injury. I have, however, seen rapid cures by it of hysterical contractures which had resisted all other treatment. Hypnotism is especially dangerous when the hysteria is deeply seated in an original lack in the nervous system: in accidental hysteria it may be used more freely. I do not believe at all that the cure in these cases is due to any suggestions made in the hypnotic state: the result of treatment is rather the outcome of a mental impression.

The same may be said of mock surgical operations, which may occasionally be used with excellent result, but which are of course especially liable to abuse. Bread pills given with absolute carefulness as to detail of administration sometimes work wonders. Of exactly the same character are the so-called faith cures, pilgrimages, etc. One of the worst cases of chronic hysteria I ever knew, who has been much in the newspapers in recent times as having been cured by a pilgrimage to Our Lady of Lourdes, was the subject of a clinical lecture by myself, published ten years ago, in which lecture it was stated that the woman would probably some time be relieved, but not by the doctors.

Certain drugs belonging to the so-called class "antispasmodics," notably asafœtida, valerian, musk, and camphor, are useful remedies in the treatment of mild hysteria, since they often abate the symptoms, quiet the nerve-excitement of the patient, and never do harm. The bromides have more influence than any one of these remedies, but are distinctly more capable of harm, and should only be allowed at the discretion of the physician. Chloral, morphine, and other narcotics may sometimes be used with advantage, but the danger of the production of the narcotic habit is very great, and neither of these remedies nor alcohol should under any circumstances be prescribed to the patient with knowledge on her part of what she is taking.

Local treatment in hysteria is always accompanied with a danger of increasing the symptom by calling attention to it; nevertheless, in many cases it is necessary. Pains which seem agonizing will often be forgotten in a moment under some pleasurable excitement, and the attempt to relieve pain by narcotics always endangers the formation of the narcotic habit. The headaches of hysteria can sometimes, however, be much benefited by the use of the bromides or of cannabis indica.

In pseudo-hysterical angina it is probable that the pain itself is not altogether purely hysterical, but the result of an hysterical spasm or loss of power in some portion of the cardiac apparatus. Certainly this pain is often relieved by nitro-glycerin or nitrite of amyl, and the paroxysms prevented by the persistent use of cardiac tonics, such as digitalis and caffeine. Counter-irritation sometimes will relieve the pain of hyperæsthetic ovaries, and occasionally is

of value against the tenderness of the so-called spinal irritation. Usually, however, in these cases the application of a belladonna plaster is more successful. In severe cases of pain hypodermic injections of water sometimes have a marvellous effect. This effect of such injections is especially pronounced in hysterical insomnia, deep sleep being frequently produced by a few drops of a fluid which the patient believes is a saturated solution of morphine.

In the treatment of painful special-sense hyperæsthesia it is essential that the patient be not allowed to be shut up in dark rooms or in excessively quiet apartments, but to be forced to endure at least a moderate amount of the natural stimulus which it is affirmed causes so much pain. I have cured severe hysterical blindness on one side by closing the sound eye hermetically with sticking-plaster and putting blisters, one after the other, behind the ear and on the temple of the blind side, the patient being informed that the fastening up of the well eye and the blistering would be kept up indefinitely until sight returned to the affected organ.

In hysterical paralysis faradization and massage are often of great service, provided the patient be impressed with the belief in their power for good. Hysterical contractures are among the most obstinate of symptoms: occasionally they can be affected by static or faradic electricity, by blistering, or by other procedures which produce a distinct moral or physical perturbation. I have found in obstinate cases very marked benefit from the subcutaneous section of the tightened tendons. How far such operations do good directly, and how far they produce their effects by causing a mental impression, is uncertain. As already stated, hypnotism is of special value in this form of hysteria.

Hysterical retention of the urine requires the exercise of great tact in its management. On the one hand, the long distension of the bladder may produce organic disease; and, on the other hand, a large element in the production of the retention may be the desire of the woman to have the doctor draw off her urine. The catheter should always be passed by a female nurse, the doctor positively refusing to do it.

Hysterical vomiting is to be, at first at least, totally disregarded. If, however, it persist and become severe enough to attack nutrition, the various antiemetics, especially cocaine, may be tried, but artificial feeding should soon be resorted to. Semi-liquid, half-digested food should be given by means of the nasal œsophageal tube, the process being made distinctly disagreeable for the patient.

VERTIGO.

Vertigo, or the sensation of moving or of an appearance of motion in surrounding objects which are really at rest, varies from the slightest swimming of the head to the attacks in which the victim seems involved in a whirling chaos of motion. In the more severe attacks the ground undulates or rises or sinks; houses move; hills, trees, and rocks slant hither and thither; and in some instances the whole landscape inverts itself and hangs

above the head, threatening ruin. In these severe cases there may be distinct perversion of special senses. The term *vertiginous status* is applied to the condition in which paroxysms of vertigo occur in rapid succession.

Vertigo may be *organic*, dependent upon organic disease of the nerve-centres; *cardiac*, due to disturbance of the circulation; *epileptic*, the outcome of idiopathic epilepsy; *hysterical* or *neurasthenic*, including cases due to nervous exhaustion; *peripheral*, due to a peripheral irritation; *special sense*, due to derangement of the special senses; *toxæmic*, due to the presence in the blood of some poison; *essential*, including cases whose pathology is at present unknown.

Organic Vertigo may arise from almost any form of brain disease: it is exceedingly common in the invasion of multiple cerebral sclerosis, and may be the outcome of a specific or of a true locomotor ataxia.

Concerning *Epileptic Vertigo* see Epilepsy.

Cardiac Vertigo is usually seen in fatty degeneration or other disease of the heart accompanied by failing power, and is usually to be recognized by the fact that it is produced by any sudden severe exertion or is accompanied by cardiac distress or by fear. *Mal de montagne* (headache, vertigo, dyspnœa, with nausea and vomiting), produced in some persons by the rarefied air of high mountains, is probably a cardiac vertigo, as is also the swimming of the head seen in persons of advanced age with atheromatous arteries.

Any variety of vertigo may be closely counterfeited by the *hysterical* affection, which, however, frequently takes some bizarre atypical form. The vertigo of neurasthenia is rarely severe, and is especially apt to be provoked by sudden intense peripheral sense-irritation, as flashing of light, etc.

Among the *peripheral* vertigoes may be mentioned the affection described by Prof. Charcot under the name of *laryngeal vertigo*, in which laryngeal paræsthesiæ with spasmodic cough are followed by a brief vertigo. Some of the cases reported of laryngeal vertigo have probably been a form of idiopathic epilepsy or of ataxic laryngeal crises, but the cure of some of the cases by removal of a laryngeal polypus or other gross laryngeal lesion has proven that the brain symptoms may be reflex.

Gastric Vertigo may be the result of an acute gastric irritation, produced, perchance, by indulgence in strawberries, lobsters, shell-fish, or some other articles of diet out of harmony with the digestion of the individual. This vertigo is often attended with intense headache, partial blindness, or double vision, and is relieved by vomiting.

Chronic gastric vertigo, due to persistent dyspepsia, is a much rarer affection than was supposed by Trousseau and his followers. In those cases of chronic dyspepsia in which the more or less constant vertigo is at its worst two to four hours before eating it seems to me as rational to ascribe the vertigo to the presence in the blood of products of imperfect digestion as to attribute it to gastric irritation. In some dyspeptics, however, there are more or less frequent paroxysms of vertigo, with ocular disturbance and sick stomach, closely simulating those of an acute gastric vertigo. It is possible that the vertigo which occurs long after eating in chronic dyspepsia may sometimes be due to an intes-

tinal irritation, as is undoubtedly the giddiness with a sense of weight over the brows, or even of burning in the eyes, which may be the only manifest symptom of tapeworm.

Vertigo of the Special Senses.—It is well known that certain rapid changes of position produce giddiness with nausea and vomiting, notably rapid whirling, as in the waltz, swinging, as in the play of children, and the rocking motion of the ocean. The giddiness in these cases may be in part due to the disturbance of circulation, but it seems also to be in part caused by the special-sense irritation.

Paralysis of the external rectus, and more rarely of other eye-muscles, often produces a vertigo which is probably the result of the confusion caused in the nerve-centres by the non-agreement of the eyes and other special-sense organs in their representation of objects. Even when one eye is closed the object may be perceived in one direction by vision and in another by the sense of touch or of the muscular sense, so that closure of one eye does not always arrest the vertigo. Nystagmus sometimes produces giddiness by the rapid changes in the position of sensory impressions on the retina. It is important to remember that whilst an acute paralytic squint is almost invariably accompanied by double vision and giddiness, a concomitant[1] squint rarely produces either of these symptoms.

Vertigo is sometimes produced in man by injections into the external ear of very hot or very cold water; also by mechanical interference with the Eustachian tubes. It is as yet uncertain whether such vertigo ought to be considered as really due to the disturbance of the external ear or as a purely reflex phenomenon. Any disease which involves the aural labyrinth is very prone to produce pronounced vertigo; and intense sudden congestion of the inner ear or apoplexy into the semicircular canals may cause a sudden violent vertigo, accompanied by extreme pallor of the face, excessive sweating, and violent symptoms of imminent syncope or even death. Almost all forms of aural vertigo are spoken of by writers as *Menière's disease:* the name should, however, be restricted to the sudden apoplectic cases, such as were described by Menière in 1861. *Aural vertigo* may be essentially chronic and persistent. I have seen frequent vertiginous attacks produced by a gunshot wound in which the bullet lodged in the vicinity of the semicircular canals. The nature of an aural vertigo is to be made out by noticing the deafness or other evidence of local disease of the ear. Menière's disease must be distinguished from *Voltolini's disease,* which is a purulent, labyrinthic otitis. In Voltolini's disease the attack is accompanied with violent pains in the ear, the unconsciousness becomes complete, and high fever and delirium indicate the gravity of the affection.

Toxæmic Vertigo may be the only decided symptom of a mild uræmia, is very commonly of lithæmic origin, or may be produced by alcohol or various

[1] A concomitant squint is one which is produced by defect of the eye itself. The fact stated in the text is probably owing to the slow development of the concomitant squint and the habitual disregard by the brain-centres of the visual image in one of the two eyes.

other poisons. The lithæmic vertigo is often severe and attended with some mental confusion. Its nature is to be recognized by detecting the lithæmia.

Essential vertigo represents a class of infrequent cases in which no known cause of vertigo can be discovered. It is probable that in the brain there are undiscovered centres of equilibration disease of which may give rise to vertigo.

Treatment.—The treatment of vertigo resolves itself into the treatment of the disease which produces the symptom. In those cases in which no distinct cause can be discovered remedial measures should be directed to the thorough building up of the general health. There is no known specific treatment of the vertigo itself.

Epilepsy.

Definition.—A disease of unknown pathology, in which at irregular intervals and without obvious existing causes an abnormal disturbance of nerve-force occurs, in most cases accompanied with loss of consciousness and very frequently by convulsive disturbance.

Synonyms.—Idiopathic epilepsy; Fits; Falling sickness.

Etiology.—The importance of heredity in the production of epilepsy is shown by the fact that of 4300 cases collected from various sources by Prof. H. A. Hare, 26 per cent. afforded a distinctly neurotic family history. Very frequently the inheritance is direct, the epilepsy attacking various members of successive generations; but perhaps in the greater number of cases the epilepsy is an expression of a neuropathic root-stock. Especially does the disease interchange in different generations with insanity. Alcoholism in the parent is frequently an active cause; consanguineous marriage has a distinct but less powerful influence. Scrofulosis, rachitis, extreme poverty, or dissipation, anything which exhausts the vitality of the parent stock and tends to the production of nerve-degeneration or of imperfect development of the nervous system, certainly has an influence in the production of the epileptic diathesis.

The production of what may be termed accidental epilepsy by poisons, alcoholism, extreme dissipation, violent emotion such as fright, peripheral irritation, etc., may result in a permanent epilepsy, it being an established clinical fact that when, through the action of some removable cause, the nervous system has become accustomed at irregular intervals to discharge paroxysmally nerve-force, the habitual discharge is very prone to continue after the removal of the original cause.

Epilepsy is somewhat more frequent in males than in females. It is especially a disease of early adult life, but once established is permanent. Probably about one-third of the cases have their beginning under thirteen years of age, two-thirds under nineteen, and the remaining third under thirty years of age; the number of cases occurring after thirty being so few as scarcely to affect statistics.

Symptomatology.—In the typical, fully-developed epileptiform convulsion the first symptom is a peculiar sensation, first felt in some part of the body, and rising from its seat of origin up to the head, to be lost in uncon-

sciousness. This so-called *aura* is succeeded at once by a peculiar wild, harsh scream, known as the *epileptic cry*. With the first unconsciousness a general tonic spasm comes on, producing rigidity of the whole body and violent distortions of the head, limbs, and face. The muscles of the trunk and abdomen are rigidly contracted. Often a turning of the head and eyes to one side is the first evidence of this condition, and in some cases not only the head but the whole body rotates. The facial muscles are violently contracted, usually most markedly on the side toward which the head turns; the jaws are fixed and often drawn to one side; the arms are almost always flexed at the elbow, and still more strongly at the wrists; whilst the fingers are flexed at the metacarpo-phalangeal joints and extended at the others, the thumb being adducted into the palm or pressed against the first finger. The position of the fingers is similar to that of grasping a pen, and is due to conjoint spasmodic contractions of the interosseous and flexor muscles, as in the so-called athetosis. The legs are extended and the feet inverted. The position of the arms, legs, hands, and feet is usually that which is assumed in a case of universal tonic spasm, the members being drawn always in the direction of the muscles of superior power; but in some epileptic convulsions this is departed from, showing that certain of the muscles are more affected than others. Thus, the fists may be clinched or the legs may be violently flexed and drawn up on the abdomen.

The stage of tonic spasm is usually accompanied by marked pallor of the face, and lasts from a few seconds to one or even two minutes, when it is succeeded by the stage of clonic spasm. Usually the coming on of this is marked by vibratory tremors passing into vibrations, which continually grow both slower and more severe until the intermissions become long and complete, and the limbs are alternately relaxed and jerked in movements as wild and bizarre as they are violent. During the period of clonic spasm the face becomes red, congested, even bloated, and often livid. The expression changes continually, since the spasm involves all the muscles of the face, including those of mastication and of the tongue, the soft palate, and the larynx. Owing to the violent working of the muscles of mastication the saliva is forced from the mouth in the form of froth. The tongue is continually thrust in and out by the spasm of its muscles, and is apt to be caught between the convulsively moving jaws and severely bitten. If the tongue happens to be between the teeth during the period of tonic spasm in an epileptic convulsion, it is bitten in the first stage of the fit.

The blood-stain which is so characteristic upon the froth is due to hæmorrhage from the tongue. The pupils at the beginning of the fit are sometimes contracted; absolutely immovable dilatation occurs, however, very early, if indeed it be not present from the onset, and is the characteristic condition during the whole fit. The return of the pupils to the normal state is often one of the earliest evidences that the paroxysm has exhausted itself. In some cases after the fit the pupils undergo remarkable oscillations. During the height of the attack both the pupillary and the conjunctival reflexes are abolished. The sphincters are in the majority of epileptic convulsions not relaxed, but it is not

rare for the urine and fæces to be passed, and Gowers affirms that this is more apt to occur in nocturnal fits. The pulse, in the beginning feeble or of normal force, during the height of the paroxysms is greatly increased in frequency and in force. M. Magnon states that during the tonic stage the pulse-rate falls, and the rhythm is altered so that a complete systole and diastole may occupy six times the normal period. During the clonic convulsion the respiration is noisy, stertorous, slow, or even irregular: often the pauses between the acts are so long that the patient seems to have stopped breathing, and when death occurs in a fit it is by the persistence of such arrest of respiration.

During the convulsion of epilepsy the bodily temperature may remain about the norm, but, if the attack be prolonged, usually rises, very rarely, however, going above 102°. During the status epilepticus the temperature of 107° may be reached.

The stage of clonic convulsion lasts from three to four minutes, when it merges into the condition of quiet coma, and this in turn passes into a heavy sleep, which may continue for a few moments or for hours. After the waking the patient suffers from headache and general muscular soreness.

The description which has just been given represents the epileptiform convulsion as it is seen in what may be considered typical epilepsy; but even in the majority of cases of epilepsy some of the phenomena are wanting, and almost any of them may be absent. The essential or central idea of the epileptiform convulsion is the occurrence of complete unconsciousness, with nervous discharge taking the form of a clonic spasm, in which the movements have no relation, apparent or real, to those of ordinary life.

It must never be forgotten that the epileptiform convulsion in its most typical manifestations may arise from causes other than epilepsy, and also that epilepsy may give rise to convulsive and other nervous disturbances replacing the epileptiform convulsion, but entirely different from it in their phenomena. Before considering these anomalous epilepsies I shall discuss in more detail one or two of the more important symptoms of the convulsive attack.

The *epileptic cry* is probably due to a forcing of air by the convulsive contractions of the respiratory muscles through a glottis narrowed by spasm of the vocal cord. It is commonly single, but may be repeated, although much repetition should always raise the suspicion that the attack is hysterical.

The *aura* is often absent. When present it usually arises in one extremity or in the stomach, although psychical and special-sense auras do occur, and in some cases warnings are given by bilateral tremors or starts in the limbs, or by widespread indefinable sensations, which may perhaps be looked upon as generalized auras. Various as the auras are in different individuals, they are remarkably constant in the one subject, each epileptic paroxysm conforming to those that have preceded it.

An aura which commences in an extremity is usually first felt in the hand, but it may begin in the foot. From the hand it rises up the arm as an indescribable sensation, and is not rarely traced by the patient to the neck, where it disappears in the development of unconsciousness. The gastric aura is very

frequent. It is variously described—as pain, as burning, as a sense of coldness, as trembling, but more often as an indefinite distress. Usually there is no sensation of rising connected with it, but in some cases this occurs. An aura may be first felt in the chest, and ascend to the throat, when it gives rise to choking sensations. It may also begin in the face, tongue, larynx, pharynx, or indeed in any part of the body. In psychical aura the emotion is almost always that of alarm or excessive terror. In very rare cases a very peculiar idea ushers in the epileptic convulsion, constituting a true intellectual aura.

Special-sense auras are rare, the gustatory being the most infrequent, the ocular the most frequent. The ocular aura may consist in seeing colors; in an apparent increase or lessening in the size of objects; in indescribable visual sensations; in double vision, or in loss of distinctness of sight, deepening, it may be, into complete blindness. In a few cases there are actual visions, either simple or complex. In the auditory aura abnormal sounds are heard, such as hissing or the whizz of rushing steam, or intermittent, pulsating noises, such as beating of drums or music, and in very infrequent cases even a spoken word. The olfactory aura seems always to take the form of a bad smell.

The rate of the aura varies very greatly. When it is slow enough to allow of the institution of proper measures the fit can usually be aborted.

According to the observations of Westphal and of Gowers, none of the myotatic contractions can be obtained immediately after a very severe epileptic fit, but after about half a minute the knee-jerk reappears, and frequently becomes excessive; ankle-clonus may also be temporarily present.

The most important of the *anomalous epilepsies* is that which is known as "*petit mal*," or the little sickness, in contrast to the larger attacks, which are known as "*gros mal*." In its more ordinary form petit mal consists of a momentary loss of consciousness, accompanied by pallor of the face, which is not, however, invariably present. The sufferer, in the midst of a conversation, suddenly stops, is quiet for a few seconds, and then takes up the thread of discourse as though nothing had happened, being in fact unconscious that anything has happened. Sometimes the period of consciousness is followed by a state of confusion of thought.

It must be borne in mind that every grade of attack occurs in nature between the mildest and briefest paroxysm of petit mal and the most severe convulsion. Sometimes the unconsciousness is accompanied with great muscular relaxation and a fall to the earth, without further symptom. Sometimes the petit mal is ushered in by a distinct aura or even a single loud, piercing scream, which may not be followed by motor disturbance. So variable and so frequently absent is the convulsive portion of the epileptic paroxysm that the unconsciousness is usually considered as the essential portion of the epileptic paroxysm. I am sure, however, that in an epileptic attack consciousness may be preserved. In a case which was probably one of epilepsy, and in which, so long as I had opportunity for watching the symptoms, there was no change, the patient had a distinct aura in the hand, rising up the arm in the usual manner, but suffering arrest in the neck, at which time, without any loss of con-

sciousness, there were violent convulsive movements of the muscles below the position to which the aura had reached.

Further, I am sure that the epileptic paroxysm may show itself simply as a sensory disorder which resembles an epileptic paroxysm cut off at the end of the aura stage. Thus I have seen in various children paroxysms in which the child would cry out with a sudden painful sensation in its stomach, become extremely pallid, run to its mother, be held for a moment, and the whole attack would be over, there being, at least in some of these attacks, no loss of consciousness. That these cases represented true epilepsy has been demonstrated by their continuance in spite of all treatment; by the regularity of their occurrence; and, beyond all, by the fact that I have watched them develop into fully-formed, unmistakable epilepsy.

When the epileptic paroxysm occurs only at night (*nocturnal epilepsy*), its existence may be entirely overlooked. Sometimes the patient wakes before the occurrence of the paroxysm, but very frequently he passes directly from the unconsciousness of sleep to the unconsciousness of the epileptic convulsion, and on waking in the morning has no knowledge of what has occurred, although usually there is much malaise and general physical weariness. A bitten tongue in such cases ought to reveal the occurrence of the night. Frequently, but not by any means universally, the urine is passed during the attack, and whenever a new habit of wetting the bed at night is formed during late youth or early adult life suspicion should be aroused.

The most characteristic feature of the movements of ordinary epilepsy is the absence of apparent purposiveness, but in anomalous epilepsy this characteristic may be wanting, as in *procursive epilepsy* and *epileptic automatism*.

In *epilepsia procursiva*, either with or without a primary epileptic cry, the subject starts on a run, either forward or in a circle, and after a greater or less time wakes up or falls in a violent clonic convulsion. I have seen the arrest of such a patient by force change the attack of running into a clonic convulsion. This procursive epilepsy is rarely preceded by an aura; is often but not always associated with organic disease of the brain; occurs usually but by no means universally in young subjects; may continue for years unchanged or be transformed into an ordinary epilepsy, during the transformation the attacks being now this, now that, form. It is said, also, to be frequently associated with moral degradation.

In a paroxysm of *epileptic automatism* the subject performs simple or complicated acts apparently involving the possession of consciousness, and yet is in the condition in which he has no proper control or knowledge of himself or of his surroundings. The relations of epileptic automatism to double consciousness are very close. The condition may precede or may follow the convulsive attack, the patient running, singing, dancing, laughing, gesticulating, or doing other bizarre actions, and then falling in a convulsion. Epileptic automatism is, however, usually a post-paroxysmal phenomenon, and occurs more frequently after a minor than after a major epileptic attack. In its simplest form the automatism consists of doing something which is usually incongruous, such as

undressing regardless of surrounding circumstances, seizing and secreting about the person small objects, cutting bread and buttering it and eating it as fast as possible, etc. etc. Sometimes the series of acts are so apparently rational and purposive that it is almost impossible to persuade bystanders that the patient is not conscious. Gowers relates a case in which a London cabman would drive through the most crowded streets of London without accident; and a woman under my own care would continue whatever act she was doing at the time of the convulsion. Thus, when preparing a meal she would fall into a convulsion, get up in two or three minutes, and continue to dish up the dinner, arrange the plates, etc. in an apparently natural way, but after a time would suddenly wake up and have no knowledge of what she had been doing.

In many cases of epileptic automatism no display of emotion is made: sometimes, however, the patient is hilarious, and even aggressively affectionate, and still more frequently rage or violent emotion is manifested. It is through cases in which violent passion asserts itself that epileptic automatism passes into the so-called *epileptic mania,* which, indeed, may be very logically considered as a form of the automatism associated with excited emotions.

In maniacal epileptic automatism, so called, there is violent excitement and delirium, which may take the form of an acute mania or of an agitated melancholy: in either case the incoherence is usually less than in the corresponding non-epileptic affection. Not rarely after a primary period of violent disconnected speech the patient is seized with an ambitious or mystic delirium, or sometimes a delirium of persecution, or, more rarely, with an erotomania, in which sentence after sentence flows out with extraordinary volubility. The attack usually comes on suddenly, and is always accompanied by hallucinations, which sometimes develop brusquely or, more rarely, in the course of a few minutes. The hallucinations affect all the senses and give rise to delusions which conform with the type of the emotional disturbance. The delirium may last for a few moments or several days. It is especially characterized by the tendency to acts of extreme violence—to suicide in the melancholic form and to homicide in the maniacal variety.

In epileptic fury the subject has no control over his actions, and when murder and other crimes are committed it is important that the medical jurist recognize the true nature of the attack. When the mania is of mild type the danger of overlooking its character is greatest. The diagnosis is to be made by obtaining the history of previous attacks of epilepsy, by the brutality and causelessness of the crime, and especially by the fact that the patient has no memory of occurrences which took place during the mania. In a certain proportion of the cases the attacks of epileptic mania are repeated in exact counterfeit one of the other. The maniacal outbreak may, however, not recur for a great length of time. The difficulties of the expert are increased by the fact that the first paroxysm of an epilepsy may take the form of a furious outbreak of epileptic mania. Under these circumstances it may be essential that the patient be kept for a length of time under surveillance, since, although the circumstances of the paroxysm may satisfy the mind of the medical expert, they may fail to

carry conviction to judge and jury. Esquirol states that the homicidal mania of epilepsy is never radically cured, and that its subject is always liable to a fresh outbreak. Whether this be absolutely true or not, it is certain that the recurrence is sufficiently habitual to demand the perpetual surveillance of the epileptic criminal.

Epilepsy frequently leads to mental degradation, which may end in complete dementia. More rarely a permanent insanity develops in the epileptic, although it is doubtful whether the convulsions in these cases are not simply the outcome of an original neurotic vice which is also the cause of the insanity. The type of such insanity is said to be usually melancholic, with delusions of persecution and suicidal impulses. The characteristic mental state of chronic epilepsy is progressively lowered mental power, with a peculiar irritability and brutal selfishness, and outbreaks of furious anger on the slightest provocation. Even while the mental powers are still active epileptics very frequently are peculiarly irritable and revengeful. After a paroxysm these tendencies are increased.

The term *Cardiac Epilepsy* has been given to a peculiar form of paroxysmal attacks with convulsive movements in which it is doubtful whether the nervous or the circulatory disturbance should be considered primary. There are two forms of this affection.

In *Syncopal Cardiac Epilepsy* the habitual pulse-rate is much below the norm, and at the moment of the attack diminishes to twelve, ten, or even five per minute. The paroxysm may be ushered in by an aura; the face, at first pale, afterward becomes congested; the respiration, at first often quickened, is labored and stertorous; the bodily temperature usually, if not always, falls, in some cases very distinctly. Not rarely, directly before the paroxysm, the patient complains bitterly of intense coldness. Whilst unconscious the patient may be quiet, but general or more frequently partial convulsions, with or without biting of the tongue, are not infrequent phenomena.

In the *Congestive Cardiac Epilepsy* there is during the attack excessively violent heart-action, with intense congestion of the head, giving rise to deep flushing, to the formation of punctate ecchymoses, and even to general oozing of blood from the face. The conjunctiva is usually extraordinarily congested and swollen, and often bleeds freely. Not rarely violent hæmorrhage from the nose occurs. I have never seen the attack commence with an aura, but the convulsive movements may be very violent.

Diagnosis.—In discussing the diagnosis of epilepsy it seems best, first, to consider the anomalous forms of epilepsy; second, the relations of idiopathic epileptiform convulsions to other forms of convulsions.

The diagnosis of anomalous epilepsy becomes easy when it is recognized that the *essential character of idiopathic epilepsy is a tendency to an abnormal discharge of nerve-force at irregular intervals and without obvious cause, but dependent upon some persistent, usually irremediable, state of the nervous system.* Such being the fact, whenever during late childhood or early adult life peculiar paroxysmal attacks occur, evidently not of hysterical origin nor yet

due to irritation, to abuse of alcoholic or sexual pleasures, or to any other assignable cause, the practitioner should suspect the presence of an aberrant epilepsy. It is, however, very unwise to express such an opinion too hastily, and only after the failure of long-continued treatment to effect a cure should the probable nature of the attacks be explained to the parents or immediate friends of the patient.

The convulsions produced in childhood by the peripheral irritations, etc. are distinguishable from epileptic fits only by the failure of repetition. It must be remembered that what may be called the convulsive diathesis in the child is closely associated with the epileptic diathesis, and that in a large proportion of cases of epilepsy there is a history of repeated convulsions during childhood. Some children are evidently born with a convulsive tendency so firmly fixed in the nervous system that its possessor is doomed from birth to a hopeless epilepsy. On the other hand, there are individuals in whom the epileptic tendency originally exists, but in so slight a degree as to be amenable to hygienic and medicinal treatment. Such an individual may during childhood suffer from repeated attacks of accidental convulsions and become epileptic, or by great care the early convulsions may be prevented, the diathesis or tendency be overcome, and the nervous system be allowed to harden into the normal mould.

The characteristics of the hysterical convulsion, as contrasted with the epileptiform, are the peculiar disturbances of consciousness (see Hysteria, page 597); the presence of emotional disorder; and the tendency of the muscular contractions to affect only a part of the body, to simulate in an exaggerated form natural movements, and to become tetanic. Persistently clonic spasms pertain especially to the epileptiform convulsion, whilst persistent tetanic rigidity is highly characteristic of hysteria.

Although usually the true nature of hystero-epilepsy can be recognized, there are cases in which it is necessary to reserve the diagnosis until the patient has long been watched. It must also be remembered that pronounced hysterical phenomena may immediately follow a purely epileptic convulsion. Nocturnal epileptiform attacks, in which the patient passes without waking into the convulsion, are probably never hysterical.

It is necessary also to distinguish from idiopathic epilepsy epileptic convulsions due to peripheral irritation, or "reflex epilepsy;" epileptiform convulsions due to violent poisonings from within or without the body, or "toxæmic epilepsy;" and epileptiform convulsions due to organic brain disease, or "organic epilepsy."

The age at which the epileptic paroxysm has first appeared is a matter of vital importance in the diagnosis between idiopathic epilepsy and the diseases which simulate it. It may be laid down as a rule of sufficient accuracy for practical guidance, and having very rare if any exceptions, that *an epilepsy which develops after the thirty-fifth year of age is not idiopathic, but is due to some organic brain disease, to the abuse of alcohol, reflex irritation, or other causes, which in some cases may be so hidden as to be exceedingly difficult of*

recognition. An epilepsy which first appears after the thirteenth year should be viewed with great suspicion. In my own experience epilepsy occurring between the age of thirty-five and fifty-five, not dependent upon assignable causes unconnected with organic brain disease, has in at least 80 per cent. of the cases been due to brain syphilis.

The nature of an *Organic Epilepsy* is often indicated by the character of the attack. In idiopathic epilepsy the convulsion rarely begins habitually in one extremity. Such mode of onset, and especially the confinement of the movements to one limb, one side of the face, or other muscular territory, should arouse the grave suspicion of Jacksonian epilepsy due to organic focal brain disease. An idiopathic epilepsy, or at least an epilepsy in which no change can be demonstrated in the nerve-centres, *may*, however, take on the Jacksonian type, so that in any case, before giving a positive opinion, it is wisest to wait for other symptoms of organic brain disease; but here it must also not be forgotten that a temporary aphasia and a hemiplegic or monoplegic paresis may follow a paroxysm of idiopathic epilepsy.

Reflex Epilepsy is not to be distinguished by any peculiarities in the convulsion, but only by finding the cause of the irritation and noting the effect of its removal. It must, therefore, be an invariable rule for the practitioner to search thoroughly every epileptic individual for points of irritation. Wounds of the head or other portions of the body, astigmatism and other imperfections of the eyes, diseases or malformations of the nasal cavity, carious teeth and retained milk teeth, aural disease, adherent prepuce or other irritation of the genital organs, intestinal worms,—are among the irritations which have in very many cases provoked a reflex epilepsy. The importance of thorough examination is increased by the fact that the reflex epilepsy may engender the epileptic habit.

Of the *Toxæmic Epilepsies* the most important is the alcoholic, which may simulate very closely not only major epilepsy, but the simple epileptic vertigo or petit mal. Not rarely the attack is ushered in by headache, gastric embarrassment, troubles of vision, excessive tremors, or some similar prodrome which may closely resemble an aura, and probably is of the nature of an aura. The alcoholic convulsions often occur in paroxysms, two, three, four, or more, one after the other, at intervals of a few moments, and are not rarely followed by a temporary mental derangement which may take the form of acute dementia, during which the subject is reduced to the condition of an automaton, obeying immediately and mechanically all impulses from without. Uræmic and plumbic convulsions may also closely simulate an idiopathtic epilepsy. In all cases of toxæmic epilepsy the diagnosis must rest upon the history of the case and the presence of other symptoms of poisoning.

Much aid in the diagnosis between uræmic, hystero-epileptic, and epileptic convulsions can be obtained by a study of the temperature. Uræmic convulsions are usually, but not always, accompanied by fall of temperature. In the severe isolated epileptic attack the temperature often rises very distinctly, and when there is a prolonged series of fits, connected by coma and occurring at short intervals, the temperature rises steadily. The single hystero-epileptic

attack is accompanied only by a slight rise of temperature, and when a series of convulsions are the expression of the hystero-epilepsy, the temperature falls very rapidly immediately after each convulsion, and does not after successive attacks reach distinctly higher than in the first.

Prognosis.—So far as the continuance of the paroxysms is concerned, the prognosis in idiopathic epilepsy should always be very guarded. Traumatic, toxæmic, reflex, and organic epilepsies may be cured, but absolute cures of idiopathic epilepsy, if they ever occur, are most exceedingly rare. Much, however, can be done to ameliorate the attacks and render them less frequent.

Death is exceedingly rare in a true epileptic fit, although occasionally a patient dies in the status epilepticus. A fatal termination from the accidents produced by the fit, such as falling into the water or fire or from a height, or suffocation from vomited food getting into the larynx or the patient lying upon his face in a soft bed, is more frequent. Notwithstanding these facts, in the majority of cases epilepsy does not very materially shorten life.

The question as to the intellectual future of an epileptic patient is always a very serious one. In a considerable proportion of cases the disease ends in mental and moral degeneration. Although the resistance of the brains of different individuals to retrograde changes from epileptic disturbances varies very greatly, three general rules may assist the practitioner in casting the horoscope: first, the younger the age at which the epilepsy commences the greater the probabilities of serious mental deterioration; second, very rarely if ever is there any distinct recovery of power, so that symptoms once established are usually permanent; third, the more frequent and severe the fits the greater the chances of intellectual ruin.

Epilepsy does not, however, always end in mental degradation even when the attacks are frequent and violent and have come on early in life. I have known very bad epileptics to pursue with activity and success a business, or even a professional, life. When the epilepsy has existed some years without producing any serious intellectual results, the chances are always in favor of the escape of the patient from such results. It is also necessary not to mistake hysterical symptoms, such as emotional excitement, pseudo-convulsions, and even pseudo-mania occurring in the epileptic patient, for symptoms truly epileptic and evidences of permanent intellectual and moral change. Chronic epilepsy may be so closely associated with the hysteria which it probably produces as to make it almost impossible to unravel the mixed symptoms. The importance of this lies in the fact that hysterical mental disturbance in epileptics often disappears.

Pathology.—Almost every conceivable form of organic lesion has been found in the brain of epileptics, but the mere variety and inconsistency of these lesions demonstrate that they are results or complications, and not the causes, of the epilepsy—a conclusion which receives positive confirmation in the fact that very frequently no grave lesion can be found in the brain of epileptics. Whether the recent conclusions of Chaslin[1] will be eventually estab-

[1] Charcot's *Archives experimental*, Juin, 1891.

lished is at present writing uncertain, but these conclusions are sufficiently remarkable and plausible to be cited here. This observer claims to have found in epileptics, as a characteristic lesion, a form of cerebral degeneration in which the changes in the neuroglia are the result of a vice of development which is ordinarily dependent upon heredity. According to the author, this "*gliosis*" is a non-inflammatory degeneration in which the neuroglia of the brain is transformed into an abnormal tissue composed of bundles of fibrillæ much longer and much more distinct than those in normal brain-tissues. The nerve-cells are reduced in size and number, with their processes shrunken or altogether removed; the capillaries are for the most part completely intact, without that cellular infiltration of their walls, and especially of their sheaths, which is so pronounced in inflammatory sclerosis. Some of the capillaries, however, are dilated, and occasionally there is one with its walls thickened.

The scope of the present volume does not allow space for discussion of the various theories which have been and are in vogue as to the cause of the epileptic attacks. A brief mention of one or two of these, however, seems necessary.

In accordance with the vaso-motor theory, the convulsion is due to a sudden over-action of the vaso-motor centre in the medulla, and consequent brain anæmia. This theory is not plausible, and really fails entirely to account for anything, as it fails to show any reason for the disordered action of the vaso-motor centre. It seems to me certain that no disorder of the vaso-motor centre could produce an epileptic attack. A second theory is that of the discharging lesion in the brain cortex. In accordance with this, the cortical cells become at irregular intervals so surcharged with nerve-force that an overflow occurs and produces a general nervous disturbance. This theory, like the one previously mentioned, fails to show the cause of the epilepsy, but plausibly explains the mechanism of the paroxysm.

Treatment.—In the so-called *reflex epilepsy* the removal of the cause of the irritation is of primary importance. When in any case the disease has followed blows upon the head severe enough to cause laceration of the scalp or injure the skull, the old cicatrix of the wound should at once be removed, and if during the operation evidences of depression should be discovered, trephining should be performed. When no evidences can be obtained of local injury to the skull, and no good result has followed removal of the scar, the question as to whether trephining should be performed or not becomes a very serious one. My own opinion is that unless some reason for doing otherwise exists the case should be operated upon. In the majority of instances the operation will probably fail to do permanent good; occasionally it will give brilliant results; and, as it is not possible to decide beforehand in which category any individual case will eventually fall, and as the operation carefully performed in accordance with modern methods is very rarely fatal, it seems to me that in so incurable a disorder as epilepsy it is wisest to give the patient the benefit of the doubt. In every case, however, the situation should be carefully explained to the parents of the patient, or to the patient himself if of sufficient age, and no operation

should be performed contrary to the wishes of the person or persons concerned. Careful antiepileptic treatment after the removal by operation of the original irritation is most essential. The epileptic habit, once set up, has in itself the probability of life, and very commonly the only effect of the removal of the original cause is to make a case amenable to treatment. It may well be that, left to itself, such a case will soon lose all the possibility of the benefit which might have been derived from the operation by the judicious after-use of remedies.

In *Idiopathic Epilepsy*, when it is possible, the epileptic paroxysms should be arrested in the first stage by mechanical or medicinal means. This, however, can only be done when there is sufficient time for action between the beginning of the aura and the coming on of the unconsciousness. When the aura begins as a slowly-rising sensation in one of the extremities, the patient should be taught instantly to grasp with the hand or encircle with a tight band previously prepared the limb above the point of attack: under such circumstances the aura will often be unable to get past the constriction. If the attack manifest itself first as a local spasm, such as the crooking of a finger, the breaking of this spasm by forcibly stretching the part should be tried. The inhalation of nitrite of amyl will, in many cases of slow aura, if promptly and efficiently performed, arrest the fit. The patient should always carry ten minims of the nitrite in pearls or in a little homœopathic vial in a pocket which can be quickly reached. The pearls are to be crushed or the bottle emptied on the handkerchief, and the deep inhalation proceeded with. As soon as the sensations ordinarily produced by the nitrite of amyl appear, the inhalation may be interrupted, but there does not seem to be any danger of injurious effects, since no case of serious poisoning by the nitrite is on record.

In the great majority of epilepsies the aura is instantaneous and the attack cannot be cut short. The patient should be placed in a horizontal position, if he has not already fallen, on a mattress or other soft body, all tight bands or clothing be loosened, and the paroxysm allowed to have its course; except that it is well to thrust something soft between the teeth, so as to prevent the biting of the tongue, such as a large flat piece of cork or India rubber, with a string tied to it, so that if it should by any means get into the back of the throat it may be withdrawn. In practice, however, it is rarely possible to prevent the biting of the tongue. The inhalation of ether often lessens the severity of the convulsion, and seems never to produce any injury. I have seen it habitually practised with very good results, and it has the great advantage of satisfying the natural craving of nervous mothers to be doing something. Chloroform is more prompt and more efficacious than ether, but its use is attended with appreciable danger. Except when in position of peril, force should not be employed in arresting the epileptic convulsion, resistance aggravating the motor disturbance. After the attack the patient should be allowed to sleep quietly until spontaneous awaking occurs.

The general management of the epileptic case should be both hygienic and medicinal. In the regulation of the habits of life and of the diet of the patient moderation in all things is especially desirable. Sufficient sleep and moderate

exercise, both intellectual and physical, should be strictly enjoined. In my opinion it is distinctly a mistake in the case of children or young adults to attempt to do away with all efforts at intellectual culture. So far as is possible the patient should conform in his daily life to the habits and customs of the non-epileptic individual, excepting that in all things he should avoid excess. Especially also is it important to the growing epileptic child that habits of discipline and self-control be enforced even more carefully than in the ordinary child. Excessive punishment must of course be avoided, but by light punishments never intermitted, by moral means, and in every possible way the moral nature of the child should be most assiduously cultivated. The fear of publicity, of physical injury from a fall during the fit, and a false sense of shame, all tend to an unnatural withdrawal of the patient from society, from business, and other pursuits. So long as it is possible this tendency should be combated, and it is much wiser to take even the risk of physical injury during the convulsion than to unnecessarily seclude the patient. Seclusion means of necessity self-introspection, and self-introspection helps most woefully in the production of mental disorder.

The diet of the epileptic should be chiefly, but not altogether, vegetable. Abstinence from meat, which has been advocated by some authorities, is certainly of no value, and flesh may be allowed twice a day in moderate quantities without any evil results whatever. Tobacco, tea, and coffee are forbidden by authorities, but I do not myself believe their use in small quantities does any injury in the adult.

The question as to the propriety of marriage is frequently put to the medical practitioner. The answer should be that marriage is not capable of doing the patient any good; that if care be exercised to avoid any excess of sexual indulgence it does no harm to the patient; but that a large proportion of the progeny of such marriage will suffer from epilepsy or other grave nervous disorder.

Laxatives and other drugs may be used in epilepsy whenever required for the relief of perverted local function; and whilst the belief frequently expressed by parents that the attacks are of gastro-intestinal origin is rarely true, constipation certainly tends to increase the frequency of the convulsion.

A very large number of drugs have been employed from time to time as specific antiepileptics: amongst these alleged specifics valerian, artemisia, belladonna, oxide of zinc, sulphate of copper, nitrate of silver, may all be mentioned as absolutely worthless. Borax has of recent years been recommended by a number of practitioners, but in my experience has proven itself almost worthless. I have taken a wardful of epileptics, and, after medicine had been withheld for weeks, given borax in as large a dose as the stomach could bear, without any apparent effect upon the aggregate number of weekly fits; and I have never seen an individual case in which the borax treatment attained any brilliant results. The only remedies which I have seen do any good are the bromides, antipyrin, antifebrin, and sulphonal. My experience with antifebrin and sulphonal has not been sufficient to satisfy me as to their value as

contrasted with antipyrin, but they certainly have some power, and probably may at times be used with advantage as a substitute to antipyrin. In a small proportion of cases antipyrin (ten to fifteen grains a day) has a remarkable effect in controlling the attacks, and I have seen individuals in whom it acted more happily than the bromides. I know of no way of clinically determining, except by trial, whether antipyrin will suit any individual case. The chief value of antipyrin, sulphonal, and antifebrin is, however, as coadjutors of the bromides rather than as the main basis of the treatment.

Of all the remedies against epilepsy, the bromides are the most serviceable. Although Albertoni has shown by direct experiment that they diminish decidedly the irritability of the cerebral cortex in the motor zone, they are palliative rather than curative, and act only while present in the cortex. They do not remove the tendency to epileptic attacks, but antagonize the action of such tendency, and must therefore be in most cases administered continuously for many years after the occurrence of the last fit. The bromides of potassium, sodium, lithium, ammonium have been chiefly used. Of these the bromide of potassium has been most employed. I have no reason for believing in its superiority to the bromide of sodium. The bromide of lithium has yielded in my hands results not distinguishable from the bromide of potassium, but is so expensive that in the absence of any advantageous power it should scarcely be given. The bromide of ammonium, I feel very positive, is superior to the bromide of potassium in being less apt to produce either physical or nutritive depression. It is capable of causing all the ordinary symptoms of bromism, but these symptoms are less severe than those produced by the bromide of potassium.

Whatever bromide is selected, it should at first be given in ascending doses until the occurrence of acne, mental depression, foul breath, somnolence, or excessive weakness shows that bromism has been produced; unless, indeed, the paroxysms are absolutely controlled before this condition is reached. Later through the case the effort of the practitioner should be to keep such a condition of bromic saturation that the patient will be continually just within the limit of distinct physiological manifestation of the drug. The non-recurrence of the fits for a month or several years gives no reason for the withdrawal of the remedy. The effect of the continuous administration of the bromide upon the general health seems to be entirely unimportant. In severe cases it may be necessary to maintain a distinct mild bromism, but this is always unfortunate, and to be avoided if possible. The tendency of the bromides to produce severe skin eruption is to some extent counteracted by the simultaneous use of arsenic. Again, the combination of antipyrin with the bromides is most effectual in its results. After very large clinical experience I am sure that a mixture of bromide of ammonium, antipyrin, and Fowler's solution affords the best combination of known remedies in the majority of epileptic cases. As the bromides act by accumulation in the system, it is not necessary to give them more than twice in the twenty-four hours—a great boon, since the frequent taking of medicine is often exceedingly irksome. The dose of

the bromide of ammonium may be from twenty to thirty grains, with five to seven grains of antipyrin and two to three minims of Fowler's solution. I have also found the combination of bromide of ammonium and sulphonal efficacious. It will probably be found that the administration of the mixture of the bromide of ammonium and antipyrin cannot be kept up indefinitely; at least I have seen cases in which after several years of use the thermo-genitic functions of the body were very much disturbed, and a tendency to excessive coldness of the extremities, with colliquative sweating, became so marked that it was found necessary to replace the antipyrin by sulphonal.

In rare cases of epilepsy the epileptic attacks take on the Jacksonian form without there being detectable organic lesions of the brain. The question as to the propriety in these cases of surgical removal of the cortical brain-centre cannot as yet be positively answered. *A-priori* reasoning seems to me to lead to the probable conclusion that such removal will be followed by secondary sclerosis, and that any benefit which may be obtained must be temporary. Moreover, the clinical results so far obtained do not seem encouraging. Nevertheless, in clear cases, with the consent and complete understanding of the situation by those persons who are specially interested, the surgeon may be justified in trephining and cutting out the centres believed to be diseased.

Convulsions.

Convulsions are symptomatic conditions which have already been spoken of in various places in this work, but their importance necessitates a brief general discussion of the subject.

The *Hysterical Convulsion* may simulate either the tetanic or the epileptiform, but can usually be distinguished by the peculiar disturbances of consciousness, and by its partial distribution, and by the persistent rigidity which attends it.

Tetanic Convulsions may be due to strychnine or other poisonings, and to tetanus, traumatic or idiopathic. The diagnosis is to be made out through the history of the case and by paying attention to the following points: In strychnine-poisoning the whole muscular system is invaded almost simultaneously, and the muscles of the jaw are the last to be affected and the first to be relaxed; whilst in tetanus the symptoms usually commence in the back and neck with pain and stiffness, and the jaw-muscles are the earliest to set and the last to relax.

In the case of *Epileptiform Convulsions* the first thing that the practitioner has to make out is whether the convulsion is one of a series or not. If the convulsion be one of a series, it is probably due to idiopathic epilepsy or organic disease of the brain, but may be the outcome of uræmic, alcoholic, plumbic, or other persistent poisoning, and in rare cases has its origin in repeated cardiac failure or a persistent peripheral irritation. It does not seem necessary to discuss here the diagnosis between these affections, as they have been sufficiently considered elsewhere.

An isolated convulsion may, of course, be the commencement of an idio-

pathic, organic, cardiac, or toxic epilepsy, but is usually due to animal, vegetable, or mineral poisoning or to peripheral irritation. In the adult it is most frequently uræmic, but may represent another poisoning. In young children the convulsion is often a prodromic symptom of some exanthematous disease, but still more frequently is it the outcome of an irritation caused by teething or by indigestible substances in the gastro-intestinal tract.

The nature of a convulsion ushering in scarlet or other fever can usually be recognized by the peculiar expression of illness and the general vital depression which attends it, and by the disturbance of the temperature, aided in some cases by knowledge of exposure to contagion. In all cases of acute convulsion of doubtful etiology the gums of the young child should be carefully examined, and if they be found swollen and inflamed should be lanced. It should also be a uniform practice to administer as rapidly as possible an emetic by the stomach, or, better still, to give a hypodermic injection of apomorphine, unless some distinct cause for the attack other than gastric irritation can be discovered, as gastric convulsions are in nowise peculiar. Moreover, the vital depression and fall of temperature produced in a susceptible child by gastro-intestinal irritation (aided perhaps by absorption of some organic products, probably the result of fermentative changes, as after eating a stale cream-puff, etc.) may closely simulate the oncoming of a systemic fever.

The general **treatment** of convulsions in children may be summed up to be—lancing of the gums if necessary, and emptying the stomach if the cause of the convulsion be obscure; the administration of the bromides freely, of chloral very carefully; the use of the hot bath, and careful administration of ether, alcohol, digitalis, and strychnine when the circulation fails. I have secured recovery in cases in which a child had ceased to breathe, and was apparently dead, by placing it in the hot bath and using artificial respiration, the emetics which had failed to act producing free vomiting as soon as the bodily temperature was raised and the accumulated carbonic acid pumped out of the system. In such cases the hot bath acts not merely as a relaxant, but as a stimulant to the circulation and restorative to the failing bodily heat. A little mustard in the bath is sometimes useful.

St. Vitus's Dance.

Definition.—A non-febrile disease, not essentially dependent upon demonstrable organic affection of the nervous system; usually occurring in childhood; characterized by choreic movements involving the whole body, with general loss of nerve-tone and of muscular power.

Synonyms.—Chorea;[1] Chorea minor; Chorea of childhood.

[1] The names of St. Vitus's dance, the dance of St. John, chorea minor and chorea major, and chorea germanorum, have been used with very varied meanings in regard to their signification. It appears that the Phrygian bacchantes, in their wild worship, were affected with furious uncontrollable automatic movements, accompanied by more or less disturbance of consciousness, and it is certain that the sect of the Suffi, in Persia, shortly after the origin of Mohammedanism, were accustomed in their sacred ceremonies to pass into a condition of raging excitement, with furious dancing, convulsive tremblings, and even general convulsions. About

Etiology.—Neuropathic heredity has some influence in predisposing the child to St. Vitus's dance. The same may be said of race, the disease at least being more rare among negroes than among whites. Any habits of life which lessen the robustness of the nervous system are also effective; hence city children are more frequently affected than those living in the country. In children under five years of age the disease is rare, though it may occur in infants. About four-fifths of the cases are reported as having been in children between the ages of five and fifteen. Girls are much more frequently affected than boys. In this climate chorea occurs more frequently in the spring months after the exhaustion of the hard winter.

So large a proportion of the sufferers from chorea come from distinctly rheumatic stock or have had rheumatism themselves before the chorea, and so frequently does chorea seemingly interchange with rheumatism, that some relationship must exist between the two diseases. This mysterious relationship is also shown by the frequency of organic heart disease, and even of acute endocarditis, in choreic children.

In most cases no distinct exciting cause can be found for an attack of chorea. Sometimes, however, the affection commences abruptly under the influence of a depressing emotion, especially of fright. Chorea is prone to recur; not, however, because one attack predisposes to another, but because a persistent, fundamental weakness renders the nervous system liable to be easily thrown off its balance time and again. The attacks may recur at short intervals or be separated by periods of years.

Pathology.—Various lesions have been found in the brain and in the spinal cord after death from St. Vitus's dance, such as minute cerebral embolism, softening of the spinal cord, interstitial proliferation of nuclei, and hyperplasia of the spinal neuroglia with serous exudations into the central canal, etc. It must be remembered, however, that chorea may be caused in a few minutes by

the year 1000 a sect of the Suffi found numerous followers and imitators throughout Asia Minor, Persia, and Egypt, and even in Greece. In Christian lands the so-called dance of St. John was already at the time of the Crusades an observed custom; and when the influence of the Suffi spread itself by the returning waves of the Crusades, the epidemics of religious excitement and automatic dancing became more and more violent. It was not until the outbreak, in 1418, of a fresh epidemic in Strasburg that the term dance of St. Veit began to be freely applied to these religious disorders—a name which appears to have had its origin largely in the fact that in these later epidemics children were especially affected. St. Veit was a boy who, born in the island of Sicily, suffered martyrdom in the year 303 during the persecution of Diocletian, and whose body, carried hither and thither for a considerable length of time, found its final resting-place in the cloister of Korvey.

By Paracelsus these epidemics were called chorea sancti viti and chorea lasciva. The disease of childhood now known as St. Vitus's dance has no connection either etiologically or in its nature with these epidemics, but modern custom enforces the application of the name to it as used in this book. By many German writers the affection of childhood is known as chorea minor, whilst the term chorea major, or chorea germanorum, is used to express affections more or less closely resembling in their phenomena those of the epidemic furies of the Middle Ages. By some German writers any very bad case of ordinary chorea is spoken of as chorea magna. Again, the term chorea major or chorea germanorum is sometimes used as a name for the automatic chorea described on page 635.

a fright, and that in a majority of cases it is recovered from in a few weeks. It is absurd to suppose that serious organic change of the nerve-centres can be present in these cases. Moreover, competent observers have failed to find alterations in the nerve-centres after death from chorea. A large number of observers have, however, especially noted changes in the spinal cord, the ganglionic cells appearing shrivelled, their protoplasm granular, their nuclei obscured, and their processes indistinct or absent. I have found exactly similar lesions in choreic dogs, and have noted that when the animals were killed in the beginning of an attack the spinal ganglionic cells showed no change: a little later the only alterations in the cells were the very frequent absence of the nuclei, the failure of granulations in the protoplasm, the loss of power to take staining fluids, and rarely the occurrence of sharply-defined vacuoles. Then the processes began to drop off; and finally it was found that the places of the cells were occupied by irregular, globose, crumpled-looking masses, without sharp outline and taking carmine staining very faintly. No granulations, no nuclei, no processes, were apparent. Evidently there is in the spinal ganglionic cell of the choreic dog an altered nutrition, which first manifests itself solely in disorder of function with choreic movements; after a time, as the nutritive process continues, the structure of the cells becomes sufficiently affected for change to be recognized, and in fatal cases some at least of the cells have undergone total degeneration. Similar structural changes have been noted in the ganglionic brain-cells of choreic dogs and cats. In the choreic child during life the will, the intellect, and the emotional faculties are often markedly abnormal, whilst after death structural change has been found in the spinal cell. It seems to me clear that the pathology of St. Vitus's dance is an alteration in the nutrition of the ganglionic structures of the whole cerebro-spinal axis; which altered nutrition may fail to develop structural changes sufficiently great to be recognized by the microscope, or may go on until it produces pronounced structural lesions.

Symptomatology.—The onset may be sudden or gradual in its development. The attack may come on in the midst of apparent health, but ordinarily it is preceded by languor, irregular action of the gastro-intestinal tract, and a pronounced nervous irritability. The motor disturbance may be first indicated by a peculiar restlessness of the child, who is not rarely punished for fidgeting. The true choreic movements usually appear first in the fingers, afterward in the face, and then spread until they involve the whole body.

In a large proportion of cases very often the two sides of the body are not equally affected, and sometimes the symptoms are limited to one side (hemichorea), or in rare cases are almost confined to one extremity. A distinct increase of electric irritability has been noted in both nerve and muscle of the affected side, and even qualitative changes have been described, it being stated that the closure contracture at the negative pole may become equal to the opening contracture. Sensibility is usually not influenced, except that tender points over nerve-trunks are said often to be demonstrable. If there be distinct pain present, rheumatic or organic complications should be suspected. The pupils

are usually somewhat dilated and react slowly to light; very rarely they are unequal. Anæmia is frequent. When marked fever occurs, rheumatism, endocarditis, or other complication probably exists; but slight elevation of temperature, apparently of nervous origin, is sometimes present.

In severe attacks of St. Vitus's dance the arms are in almost constant movement, the fingers opening and closing, the wrists flexing and extending, and the elbow-joints in almost incessant activity, so that every imaginable position of the hand and arm is rapidly taken and lost. During the violence of the disease it is impossible for the child to control the movements of the arm sufficiently to dress or feed himself or to perform any act requiring precision of motion. At this time the legs are similarly affected, so that walking is gradually interfered with or may be rendered impossible. The steps are irregular, jerking, often with lateral movements, now rapid, now slow, and if progression occur at all it is zigzag and uncertain. The face and head are no less affected: there is a constant, ever-changing distortion of the countenance, giving rise to fleeting expressions of sadness, terror, grief, rage, etc., and to grimaces innumerable. The mouth is opened and shut, the corners jerking up and down; the tongue is protruded or sometimes moved rapidly in the mouth, so as to produce a peculiar clacking sound. Articulation grows indistinct, the child speaks irregularly and badly, perhaps only in monosyllables, and finally the voice may be converted into a succession of irregular, unintelligible sounds. In very bad cases mastication becomes almost impossible, and even the muscles of deglutition are involved, so that the child is unable to swallow at the proper moment, and the food is spluttered and spilled about. The head itself is moved rapidly to and fro, backward and forward, sometimes laterally, sometimes in perpetual rotation. In the most violent cases all the muscles of the body are in a condition of furious action. The rolling, twisting movement of the trunk and the perpetual beatings and thrashings of the extremities render it almost impossible for the patient to lie in bed unless fastened down, and the utmost care is necessary to prevent severe bruises and excoriations of the skin.

The respiratory muscles are the last to be affected, but cases have been reported in which hiccough, crowing inspiration, irregular respiratory rhythm, and other evidences of choreic action of the respiratory muscles were abundantly present. The choreic movements cease at night, or at least during sleep, but in the most severe cases by keeping the patient awake they produce an insomnia which constitutes an additional factor in the rapid wearing out of the strength and the bringing about of a fatal result. That the brain-cortex does not entirely escape is shown by the peculiar nervous irritability which forms an almost essential symptom of the disease. The general intelligence is ordinarily well preserved, but there can often be noted a temporary weakness of memory, and the loss of the power of fixing the attention upon any one subject for a length of time is usually very decided.

Hallucinations are very rare, and usually indicate that a chorea is hysterical. They may, however, occur in typical St. Vitus's dance. In fatal cases

the mental disturbances are very pronounced; there may be even an acute dementia: sometimes the patient is seized with maniacal delirium, which is always of exceedingly serious import.

The muscles of organic life may participate in the choreic disturbance. This is especially true of the heart. Chronic valvular lesions are frequent among choreic patients, and an acute endocarditis occasionally occurs during an attack of St. Vitus's dance; but cases are not rare in which mitral or even aortic murmurs are heard during an attack which are not due to any organic lesion of the heart and are not hæmic in their origin. This is shown by the fact that these murmurs occur when there is no anæmia, that they vary from day to day and from hour to hour, and at times may be absent, and that when the child recovers from the chorea the murmur disappears entirely.

Further, fatal cases have been reported in which no valvular lesion was found in the autopsy, although marked cardiac murmurs had existed during life.[1] The most rational explanation of these murmurs is that they are due to the irregular contractions of the chordæ tendinæ preventing the proper closure of the valves. It is the duty of the practitioner always to auscult the heart of the choreic child, and if murmurs be present to decide, if possible, their significance. If the history of a previous endocarditis or of previous chronic valvular lesions can be obtained, the probabilities are always that the murmur is due to an old lesion. The absence of such history is, unfortunately, no proof of the previous non-existence of cardiac disease. Supposing that the murmur is recent, it is often a very difficult matter to decide whether it is neurotic or inflammatory. The neurotic murmur rarely, if ever, manifests itself in irregularity of the pulse; it is not associated with cardiac pain nor with elevation of the general temperature. If these exist, the diagnosis of acute endocarditis may be considered made out. The presence of even one of these symptoms should lead the practitioner to treat the case as one of endocarditis.

The course even of so-called acute chorea is always slow and almost indefinite in its duration; rarely is recovery complete under five weeks. The ordinary result is a complete cure, but in rare cases permanent loss of power in muscle or mind remains, evidently because of organic changes in the nerve-centres which have been brought about by the chorea. Death is exceedingly rare.

Diagnosis.—Reflex chorea often so exactly resembles St. Vitus's dance that its true nature can only be made out by the detection of the point of irritation. Especially are eye-strain and nasal difficulties apt to cause in childhood persistent chorea, and it is therefore essential in every case which resists treatment to thoroughly examine these organs. The teeth and the sexual organs should also be carefully looked at: adherent prepuce with irritation of the glands in the male, and a similar condition of the clitoris in the female, often produce a form of (probably) chorea. Hysterical chorea may closely simulate ordinary St. Vitus's dance. The nature of such a case is to be recog-

[1] *Revue mens. des Maladies de l'Enfance*, 1884, ii. 421.

nized by the existence of marked concomitant symptoms of hysteria, and by occasional persistent rigidity in the affected muscles.

The true choreic neurosis is closely allied to the hysterical neurosis, and it is often equally correct to speak of a case as one of hysterical chorea or of choreic hysteria. The choreic movements of hysteria are also more apt to be rhythmical than in St. Vitus's dance. (See Convulsive Chorea, p. 633.)

Treatment.—The treatment of St. Vitus's dance should be both hygienic and medicinal. As the underlying condition is one of lowered nerve-tone and nerve-nutrition, rest, fresh air, exercise, careful feeding, and tonics must be employed in order to increase nutritive activity. The judgment of the practitioner must decide in each individual case how much of the time the child shall spend in the bed and how much in the open air. The general error is, I think, in allowing feeble children to take more exercise than their strength permits, and certainly indolence rather than activity must usually at first be urged upon the choreic. Many hours a day ought ordinarily to be spent on the bed, while in the severe cases a rest-cure treatment may be necessary. At the same time the child should be kept in the open air as much as possible. When the weather permits swinging in a hammock or lying upon a couch the patient may at the same time obtain rest and fresh air. The food should be nutritious, but not stimulating, thoroughly digestible, and given in as large quantities as the alimentary canal will assimilate—milk and farinaceous articles, with a very restricted use of meat and of sugar. Bitter tonics and alcohol in small quantities may be administered to increase the activity of the digestive organs, whilst cod-liver oil and iron, if well borne, may be employed as nutrients. At one time purgatives were much used, and they are certainly valuable if given only in such doses as will keep the digestive tract thoroughly cleaned out and stimulated. The bitter vegetable purgatives, I think, are more useful than salines.

The special medicinal treatment of chorea is naturally divided into the palliative and the curative. The palliative treatment consists in the use of drugs which depress motor activity. It is important to remember that these remedies have no directly curative influence; that they are rarely to be employed except in very severe cases; and that in severe cases they accomplish permanent good only by procuring rest and sleep. The bromides are not very effective, are distinctly depressing to the nutrition of the nervous system, and are only to be used under peculiar circumstances. Chloral will, for the time being, quiet almost any choreic movements; especially is it active when combined with morphine; and in all cases of chorea threatening life a combination of these drugs, in the proportion of ten grains of chloral to one-eighth of a grain of morphine, should be given at night in such amounts as may be necessary to procure quiet sleep.

There are two remedies which seem to have a specific action in chorea. Arsenic is extraordinarily effective: it should always be given by itself, so that its dose can be altered independently of other remedies, and must be administered in ascending doses up to the limit of toxic action. A child five

years old may commence with three drops of Fowler's solution, given after meals in milk, the dose being increased every third day one drop until distinct puffiness of the face or gastro-intestinal disturbance is produced, when the medicine may be temporarily withdrawn until its effects have subsided. Cimicifuga sometimes succeeds after the failure of arsenic: a freshly-prepared fluid extract, having a strong odor of the drug, should be given in increasing doses until it causes headache or vertigo. Thirty minims may be the commencing dose for a child nine years old.

After a chorea has been subdued the habits of life of the child should be arranged with the greatest care in order to change, if possible, the inherent feebleness of nerve-constitution which is usually the basis of chorea.

Reflex Chorea.

Definition.—Local or general chorea, due to some peripheral irritation.

Local, unilateral, widespread, or generalized choreic movements may, under conditions of the nervous system not well understood, arise from local irritations. In rare cases the symptoms may closely simulate those of St. Vitus's dance. Intestinal parasites, diseased teeth, neuromatous tumors, nasal deformities—with their consequent disease of the mucous membrane—and other irritations have in numerous cases given rise to choreic disturbances, which have subsided rapidly or at once upon the removal of the point of irritation.

Chorea of Pregnancy seems to be produced by the conjoint influence of a predisposition to chorea, inherited or acquired inanition of the nerve-system incident to the hydræmic state of the blood during pregnancy, and various potential irritations, especially in connection with the sexual organs. It is a very serious affection, in which the movements are often so excessively violent and incessant that they deprive the sufferer of sleep, and materially aid in the production of a rapidly progressive exhaustion which not rarely ends in death.

After the removal of the irritation the treatment of reflex chorea is the same as that of St. Vitus's dance, excepting that much more is to be expected from motor sedatives—*i. e.* palliative remedies. In the chorea of pregnancy no time should be lost in bringing the patient as rapidly as possible under the influence of chloral and opium, whilst the strength is maintained by moderate stimulation and high feeding up to the point at which the digestive system refuses to do more work. When these measures fail the general consensus of the best obstetrical opinion is in favor of producing abortion before the patient's strength is too much exhausted.

Convulsive Choreas.

Definition.—Paroxysmal affections in which violent choreic movements occur, which are not distinctly purposive, or at least do not simulate complicated purposive acts, and are not attended with loss of consciousness.

Under the present heading are included groups of cases in which the symptoms, though more or less similar, have different origin. Various names have

been given to these cases—local chorea, habit chorea, convulsive tic, etc. Most of the cases are arranged in one of three groups.

Group First: Hysterical Chorea.—Almost any form of movement may occur in hysteria. When the whole body or a portion of it is the seat of more or less rapidly repeated clonic, peculiarly brusque spasms, resembling those produced by an electric shock, the case is sometimes spoken of as *electric chorea.* The choreic movements of hysteria are apt to be very rapid and more or less rhythmical. They frequently attack extremities distorted by hysterical contractures. Thus in a leg violently flexed by contractures I have seen the knees vibrate laterally over a considerable arc at the rate of one hundred and twenty times a minute. By tracing a series of cases it will be seen that disorderly choreic movements insensibly pass into vibrations, and these into true rhythmic spasms. Rhythmic spasms may affect any portion of the body. The limbs, normal or distorted by contractures, may be agitated with regular movements. The face may be attacked rhythmically, and facial grimaces, with or without the consentaneous thrusting forward of the tongue, occur. Occasionally the muscles of the larynx and of respiration are also affected, so that each spasm is accompanied by a quick, strange utterance. This rhythmic chorea again passes by insensible degrees into the purposive movements of hysteria.

Group Second: Choreic Tic.—In this form the choreic movements may simulate single purposive acts. It is probable that in many of these cases the movements have originated during childhood, at a time when they might have been controlled by a strong effort of the will, but, this effort not having been put forth, the bad habit has grown in a neurotic temperament into a fixed neurosis entirely beyond control of the will; hence the term "habit chorea." At first perhaps remedial by hygienic and moral treatment, aided by the use of arsenic, they at last are not affected by any treatment. On the other hand, some of these cases of tic are from the onset uncontrollable. The tic or spasm may involve a single nerve-distribution or a widespread area; may be irregular, having no apparent relation with life; or may continue the form of a purposive act in which perchance it has had its start. A brow may be lifted at intervals, an eye winked, a jaw dragged forward, a shoulder shrugged, a trick of gesture incessantly repeated, even a cough or a snuffle perpetually indulged in. When the paroxysm is widespread and accompanied by a diaphragmatic contraction, which by forcibly expelling the breath produces some bizarre sound, the case may assume the appearance of an automatic chorea, but is essentially different from those cases in which the movements are directed toward an end and are the outcome of a dominating impulse.

The treatment of the cases usually ends in disappointment, and must be chiefly hygienic and symptomatic. It is probable that in cases which fail to yield some minute change has occurred in the nerve-centres.

Group Third: Cases in which the Choreic Movements are Due to Various Organic Diseases of the Nervous Centres.—Under this heading also may be included the so-called senile chorea and the chorea of

insanity, although these affections are usually more widespread and more closely resemble in their symptomatology the chorea of childhood than do most spasmodic choreas.

Most of these organic choreas are hopeless, many of them undoubtedly being connected with changes in the cerebral cortex. I have seen, however, a senile chorea unexpectedly get well, the patient at the same time recovering mental health.

Automatic Chorea.

Definition.—An affection in which paroxysms of apparently purposive actions occur independently of the will of the person, as the result of an impulse which arises spontaneously in the individual or which occurs in response to something received from without the individual.

The above definition covers two series of cases: first, those cases in which the paroxysms arise spontaneously, the chorea major or chorea germanorum of some authors, also the salaam convulsions (tic salaam); second, those cases in which the paroxysms are the result of some external irritation, constituting the affection which has been described in America as the "jumpers," in Southern Asia under the Malay name of latah, in Eastern Siberia as miryachit, in France as tic[1] convulsif (in part) and as Gillis de la Tourette's disease.

Symptomatology.—In chorea major, or chorea germanorum, the outbreak is usually preceded by prodromes, such as melancholy, apathy, feeling of nausea, malaise, cramps or tonic convulsions, disturbances of the circulation, palpitations, etc. The paroxysms usually come on with a general excitement, which perhaps ought to be considered as a form of aura. During the paroxysms the affected person dances, sings, springs from the ground, rolls himself from side to side, hammers violently with the hands, stamps with the feet, or in a fury of motor excitement whirls with mad rapidity until, completely exhausted, he falls to the ground. The excitement is not confined to the motor sphere: songs are sung, affairs recited, foreign tongues spoken, in a manner entirely beyond the normal power of the individual; events, languages, poetical quotations, which seemingly never have been engraved upon the memory, are recounted or recited in eloquent or incoherent ravings. In the height of the attack consciousness is usually lost, but sometimes it is in a measure preserved, especially in the sporadic cases. As an instance of the sporadic variety may be mentioned a case reported by Robert Watt, in which a girl ten years old turned herself round and round in paroxysms; later, she had attacks in which she would roll from end to end of the bed violently backward and forward, then, lying upon her back, her feet and head would be forcibly jerked together ten or twelve times a minute. A single paroxysm of these movements often lasted fourteen hours a day.

Some cases like those described in the preceding paragraph have probably been instances of epileptic automatism, whilst others have been forms of hys-

[1] Many of the cases described as convulsive tic by French and other authors represent spasmodic chorea. (See page 634.) Further, as automatic chorea was well described before the first article of Tourette, there is no justification for attaching his name to the disease.

teria, allied to the hysterical religious epidemics of the Middle Ages and to the performances which have been frequently witnessed at revival scenes during camp-meetings in the United States and among the Howling Dervishes of Mohammedan countries.

Of different character are probably the so-called salaam convulsions of children, in which the paroxysms recur several times a day, last from a few seconds to some minutes, and consist of a bowing forward of the head and body perhaps as many as two hundred times. Allied to these cases is that of a man who formerly lived in this city, who when seized with the paroxysms would spring up, rush to a table, and jam his hat down upon his head several times, uttering at the same time certain words.

The essential feature of latah is an extreme excitability of the patient, which causes him, upon the least abrupt excitation, such as would be produced by slapping him on the shoulder, hallooing at him, slamming a door, etc., to jump or perform other violent disorderly acts, conjoined with a condition of the cerebral nervous system which necessitates a repetition of voices or sounds (*echolalgia*) or the ejaculation of some word, usually obscene (*coprolalgia*). In some cases the impulse of imitation is so great as to force the victim to repeat not only the spoken word, but also any act done by a bystander. Very frequently the sudden nervous excitement is accompanied by an excessive emotion, especially of fear, although such emotion may be entirely foreign to the ordinary nature of the individual. The disease appears to be hereditary. It often affects various members of several generations of one family.

This affection was described by Dr. George M. Beard in 1880, who found in the so-called "jumping Frenchman" of Maine that the hearing of a sudden voice or noise caused a repetition of the words or sounds, with the performance of strange antics, whilst a loud command was always obeyed, often with a cry of alarm not unlike that of hysteria or epilepsy. Two "jumpers" standing near each other when commanded to strike each other did so with zeal. Dr. Beard tested the echo-speaking or repetition by reading portions of Latin and Greek, when the untutored "jumper" repeated the sounds of the words as they came to him in a quick, sharp voice, at the same time jumping or making some bizarre motion.

M. O'Brien makes four classes of cases, as he has seen them in Southern Asia:

Class first, comprising those individuals in whom an unexpected noise produces great alarm, with an irresistible impulse to rush upon the nearest object, and at the same time forces an exclamation which is always obscene.

Class second, comprising those persons in whom certain words when suddenly pronounced will produce an excessive paroxysm of sudden terror. Thus, in an individual noted for his courage and who faced the living alligator without a sign of fear, the sudden pronouncing of the word "buaya" (Malay for "alligator") produced a paroxysm of overpowering terror.

In *class third* the individuals imitate the words, gestures, or sayings of those in their neighborhood.

In the *fourth class* the individuals become completely abandoned to the will of some other person, performing every act, however *outré* or improper, which they are commanded to do by such individual—standing on their heads, attacking a spectator, etc. In these cases the person who suffers from latah recognizes his enslavement and is greatly depressed thereby, but is unable to prevent it.

Pathology.—It seems to me that those cases of chorea major which are neither hysterical nor epileptic, and all cases of latah, are more closely related to reasoning insanity than to spasmodic disorders, the paroxysms of movement being produced by morbid impulses similar to those which occur in various neuropathic insanities. (See page 578.) It is probable that the subject of latah is under the influence of a dominating idea which compels him to obey or to imitate as the case may be. I have seen a feeble neuropathic child develop such a mental condition that, under command, the most bizarre positions were taken and maintained for an extraordinary length of time, giving an appearance easily mistaken for catalepsy. The relations of this mental state to certain stages of hypnotism are very evident.

Treatment.—There is no reason for believing that any specific medicinal treatment is of avail in the affection now under consideration.

Hereditary Chorea.

Definition.—A peculiar hereditary affection, characterized by the presence of general choreic movements, usually associated with other evidences of disturbed innervation, and probably due to a developmental organic affection of the nerve-centres.

Synonym.—Huntington's chorea.

Etiology.—So far as our present knowledge goes, this affection always depends for its existence upon direct heredity. It is commonly asserted that, if the disease fail to appear in one generation, all succeeding generations remain free, but clinical experience shows that this is not invariably the case. It is not known that exciting causes play any important rôle in the development of the disease.

Pathology.—Hereditary chorea appears to be very closely allied to hereditary ataxia, and, like the latter disorder, probably depends upon some developmental departure from the norm in the nervous system, although at present we have no positive knowledge as to this point. In the spinal cord of a patient who had been affected with this disorder Dr. Wharton Sinkler found in the antero-lateral columns of the cord an abnormal amount of connective tissue, thickening of the walls of its blood-vessels, and absence of many of the axis-cylinders. The central canal was occupied by a mass of nuclear tissue.

Symptomatology.—Hereditary chorea usually develops in middle life, although in some cases it has appeared at or even before puberty. The choreic movements resemble those of St. Vitus's dance, but are more constant, more rhythmical, and less under the control of the will. While standing or sitting the patient is continually repeating the same irregular jerking movements. The gait is especially peculiar, for the first few steps perhaps nearly normal,

when suddenly it is interfered with by one leg being thrust violently forward and the other one jerked up to it, so that the subject seems to go with a quick, short hop, almost like a dancing step.

The course of the disease is exceedingly slow, and in some cases many years are required before the subject becomes unfit for physical labor. The mental condition is usually but not always abnormal: excessive irritability, moroseness, melancholia, chronic mania, and dementia have all been noted in cases which have been reported as instances of hereditary chorea. The reflexes are often exaggerated, but may be sluggish. The sensations are normal. In some instances a peculiar muscular stiffness has been noted.

Treatment.—So far as is known no treatment is of any avail in this disorder.

Tetany.

Definition.—A chronic affection of unknown pathology characterized by tonic muscular spasm accompanied by tingling and formication.

Synonym.—Tetanilla.

Etiology.—Tetany is more frequent in males than in females, is almost confined to childhood and young adults, is very often associated with rachitis, and has repeatedly followed removal of the thyroid. It is said to be directly produced by excessive lactation, by the puerperal state, by exposure to cold, by prolonged fatigue, by exhaustion from diarrhœa or other cause, by the irritation of intestinal worms, by exposure, and even by the rheumatic diathesis or the infectious fevers. Further, it is affirmed that it may result from excessive emotion and spread from patient to patient as an epidemic. Such epidemics have, however, probably been hysterical in nature.

Pathology.—There is no known lesion in tetany.

Symptomatology.—Tetany consists essentially of successive tetanic convulsive attacks separated by intervals of quiet and repose. The paroxysms may continue for some minutes or for many hours, and may cease gradually or abruptly. Arthralgic pains, formications or numbness in the hands, radiating pains in the fingers, temporary partial blindness, headache, sense of fatigue, etc. are assigned as occasional prodromes. Usually the spasms are most marked in the upper extremities, and sometimes are confined to them; the fingers are often drawn together so as to form a cone. Rarely there is a more accentuated flexion of the fingers, and still more infrequently the hand and the fingers are stiffly extended. The feet may be attacked; sometimes cramps of the calf occur without distortion, but in other cases the feet are violently extended, with the toes pointing downward; more rarely the feet are flexed. The thigh usually escapes, but spasm of the abductors and crossing of the feet have been noticed. Only in the severest cases are the trunk-muscles affected, but opisthotonos and menacing dyspnœa do occur. Even more exceptional than these are spasmodic closures of the jaw and distortions of the face. The course of the disease may be painless; sometimes, however, neuralgic pains run along the nerves, and usually cramp-pains are present in the affected muscle. Anæsthesia and analgesia are ordinary phenomena.

According to Erb, the faradic excitability of all the muscles of the body is increased. The reflexes are said to be usually lessened in adults, but exaggerated in children.

The so-called "facial phenomena" of tetany consists of contractions of the facial muscles produced by a rapid series of taps with a percussion hammer upon the cheek just above and parallel with the horizontal ramus of the lower jaw. Schlessinger affirms, however, that the phenomenon is not constant in tetany, and is often demonstrable in persons not suffering from tetany.

Prognosis.—Tetany usually ends in recovery, although it frequently lasts for months or even years, and has a distinct tendency to relapse.

Diagnosis.—Trousseau discovered that in tetany, during the periods of relaxation, and in some cases even as long as three days after the occurrence of a convulsion, an attack can be brought on by pressing upon the principal nerve-trunk or artery. By this symptom, by the complete relaxation between the attacks, and by the partial character of the convulsion tetany is distinguished from tetanus.

Treatment.—The treatment of tetany should be primarily directed to the relief of the bodily condition underlying the disorder. Chloral, bromides, the anæsthetics, will to some extent control the movements. The value of arsenic has not been determined.

Paralysis Agitans.

Definition.—A disease of advancing life, characterized by tremors continued during waking hours, associated with muscular weakness and rigidity. Pathology uncertain.

Synonyms.—Parkinson's disease; Shaking palsy.

Etiology.—Paralysis agitans rarely occurs under forty years of age, is most frequent between fifty and sixty, more common in men than in women, and is very rarely the result of hereditary influence. Violent fright, prolonged anxiety, exposure to cold, violent physical injury, especially when accompanied with great emotional disturbance, are occasional exciting causes of the disease. In the majority of cases, however, the development of the disease is gradual and without apparent reason.

Pathology.—Some of the most noted neurologists have failed to detect any anatomical change in any portion of the nerve-centres in persons who have long suffered from paralysis agitans. Under these circumstances speculation has been rife as to the nature of the disease, and various theories have been brought forward. None of these theories seem to me very plausible, and certainly none of them are at all established.

Symptomatology.—Paralysis agitans usually comes on insidiously and gradually, although in some cases the symptoms have developed at once after a sudden fright or other emotional storm. The attention of the patient is first attracted by a tremor in the hand or foot, or even in one finger or toe. This tremor at first is transitory, can be controlled, at least temporarily, by an effort of the will, and is suspended by voluntary movement. Little by little, without

any fixed method of progression, it involves more and more of the body, becomes more and more settled, and at last continues throughout all the waking hours, during repose as well as during action, and cannot be controlled at all by the will. It often passes up the arm first invaded, and then descends to the lower limb of the same side, constituting the hemiplegic form; or it may commence in a leg and pass across the body to the opposite leg, and produce a paraplegic variety. Finally, all portions of the body are affected except the head. The face is very rarely attacked by the tremors, although in the later stages it puts on a peculiar fixed, immovable, usually melancholic expression. According to Charcot, the head is never affected, any apparent trembling of it being due to the transmission of motion from the trunk. This absolute assertion is, however, not correct, as I have seen typical cases of paralysis agitans in which the muscles of the neck and the head were in constant tremor; and Westphal[1] is said to have reported similar cases. Loss of power in the lips seems to be not infrequent in the advanced stages, so that there is a tendency to dribbling of the saliva,—a tendency which is also in part due to the peculiar prone position of the head. The speech becomes a little slow and labored, but is not profoundly affected: neither eating nor swallowing is interfered with.

The tremors themselves are short, very rapid, and in some cases distinctly rhythmical, especially in the fingers, where they may assume somewhat the appearance of voluntary actions, as though the patient were rolling something between the digits. I have noticed in some cases a distinct tendency of the tremors to alter their rapidity in accordance with any rhythmical sound, so that their rapidity could be regulated, without the patient's being conscious of it, by altering the rate of vibration in the interrupter of a faradic battery. A peculiar rigidity of the muscles is characteristic of the advanced stages. There are no violent contractures, but a characteristic fixation of the part. To this statue-like rigidity is, at least in some measure, due the position of the patient. In standing the trunk is inclined forward, with the face looking obliquely downward; the forearms usually flexed somewhat upon the arms; the hands a little bent upon the forearms, and the fingers partially closed, so that the hands assume a position similar to that in which the pen is held; hence the term of "writing hand" as given by Charcot. The same tendency to flexion of the legs exists, so that in standing the knees are bent. Occasionally, peculiar distortions of the hands or other portions of the body may be met with. On attempting to restore the normal position of the parts the muscles usually offer but little resistance until the restoration is nearly perfected.

The power of making momentary muscular efforts diminishes very slowly in paralysis agitans, but even early in the disease fatigue follows moderate exertion, so that there is soon a great loss of endurance. In not rare cases there is a marked tendency to *festination* in the walk—*i. e.* to a progressive increase in the rapidity of the gait. The man seems to be in continual danger of falling forward when attempting to walk, so that the leg has to be thrust forward more and more quickly in order to prevent toppling over, and the walk becomes

[1] *Charité Ann.*, 1878, p. 405.

more and more rapid, and in a little while breaks into a run, which grows faster and faster until the patient either falls or arrests his course by seizing hold of some stationary object. The peculiar position of the body would appear to be the cause of the accelerated gait, the head being thrown so far forward as to bring the centre of gravity beyond the line of the feet. That the festination depends upon something more than this is, however, shown by the fact that there are cases in which the tendency is to run backward instead of forward. Moreover, a very markedly bent position is not incompatible with a normal gait.

Sensation is not profoundly affected, and in some cases there is very little suffering. Usually, however, especially as the disease advances, there is a perpetual sense of fatigue in the affected muscles which may amount to a severe aching. Very frequently the patient complains of an habitual feeling of excessive heat, which may also be manifested by a continual sweating. This sensation of heat does not depend upon any elevation of the central bodily temperature, which is of normal intensity. The studies of Grasset and Apollinario, however, indicate that there is an elevation of the temperature of the external surface of the body. These observers found that, whilst the temperature of the surface of the forearm in the normal individual was 33.6° C., in a case of paralysis agitans placed under similar conditions of clothing and exposure the temperature was 36.8° C.

The urine has been chemically analyzed by Regnard,[1] who found the urea normal, the sulphates less than normal. According to Cheron,[2] there is a constant increase in the quantity of the phosphates, which is characteristic and may even precede the development of the tremors. This important observation needs confirmation.

The **course** of paralysis agitans requires many years for its full development, but if the patient does not die of an intercurrent disorder he passes into a condition of hypochondriasis, great depression of spirits, loss of intellectual power, general failure of nutrition, marked emaciation, loss of digestive power, and general marasmus, and at last dies of exhaustion, the end often being hastened by bedsores or other local ailments.

Diagnosis.—The diagnosis of a fully-formed case of typical paralysis agitans is so easy as to need no discussion. In rare cases it is said that the loss of power, the rigidity and fixedness of the limbs, and the peculiar gait develop without the tremors. Under these circumstances the diagnosis must be reserved until the symptoms become pronounced. In certain cases and stages of the disorder senile tremors may be simulated. In the senile tremor, however, the head is especially affected, and there are usually tremblings of the tongue and lower jaw. In rare cases it is necessary to reserve the diagnosis.

Prognosis.—The prognosis of paralysis agitans is absolutely unfavorable, the disease always marching slowly but steadily onward.

Treatment.—The treatment of paralysis agitans is palliative. It consists chiefly of meeting the symptoms as they arise, and of enforcing a quiet, reg-

[1] *Progrès méd.*, 1877.

[2] *Ibid.*, 1877, No. 48.

ular life, with absolute avoidance of physical or mental labor. Various nerve-sedatives, especially morphine, conium, hyoscyamine, and Indian hemp, are accredited by authorities with the power of temporarily quieting the tremors. It is evident, however, that the habitual use of such remedies must in all probability lead to the narcotic habit. Arsenic has been recommended, but is of very doubtful utility. Electricity has been much employed, but does not seem to have real value. In the advanced stages, when there is much suffering, the hot bath is sometimes of service, and not rarely it becomes necessary to use narcotics at night to bring sleep and a measure of relief from pain. I have seen a nightly dose of hydrobromate of hyoscine keep a patient comfortable for several years after the failure of more frequently used analgesics.

Remote Effects of Traumatism.

Blows upon the body or upon the head, as well as violent shaking or other concussion upon the body without actual violence, may produce, first, local injury and inflammation at the seat of the violence; second, traumatic hysteria; third, the condition for which I prefer the name of "traumatic neurasthenia."

In most cases local inflammation and traumatic neurasthenia coexist with a certain amount of hysterical disorder. Before taking up traumatic neurasthenia it seems necessary to discuss briefly the local effects of traumatism.

The paralysis sometimes seen in a muscle which has been violently struck, though not lacerated, is probably due to a suspension of the functions of the nerve-endings. The condition is rare except in the deltoid muscle, whose position renders it exceedingly liable to be severely bruised in falls. If at first local inflammation be set up, local antiphlogistic treatment may be required, and if later the nerve-trunks be found tender, blisters may be applied. If there be any hardening in the immediate neighborhood of a nerve, the latter should be dissected out and thoroughly freed from any cicatricial or otherwise altered tissue.

The hypodermic injection of strychnine, massage, and the electrical currents are to be employed for the restoration of power. One object of the massage is to thoroughly free the muscles and muscular fibre-bundles from binding exudations. The current to be used is that which produces the greatest muscular contraction with the least pain to the patient.

When the blow has been upon the back, the result is often the condition to which I have given the name of "traumatic back." The symptoms are—tenderness more marked upon deep firm pressure than upon slight pressure, also upon jarring; restriction of movement by pain and by spasm of the erector-spinæ muscles. Reflex spasms are also usually producible in the back muscles by jarring, pressing upon the head, or even upon the vertebral column.

The symptoms of "traumatic back" are probably due to deep-seated inflammation primarily situated in the fibrous structure of the vertebral column, and in bad cases invading neighboring tissues and even involving nerve-roots.

The treatment consists in the use of local rest and continued counter-irri-

tation, the general health being, of course, steadily maintained. In bad cases the plaster jacket or some of its substitutes may be essential. I have seen suspension very useful. The human trunk is composed of two cones, of which the shoulders and hips are the respective bases. In the Sayre jacket the upper of these cones is supported upon the latter after the two cones have been dragged well apart by hanging the man from his arm-pits. Some years ago it occurred to me that the upper cone of the body might be used instead of the arm-pits for the purpose of suspending the patient and stretching the back. In accordance with this thought I found that suspension from the upper cone can be sustained without suffering for many hours, and that in diseases of the vertebral column situated low down it is very advantageous. When the traumatism has affected the lumbar region of the back this treatment avails much. In carrying it out the patient should be suspended in the ordinary way for putting on the plaster jacket, and when the first layer of the plaster jacket has been put in place, two broad strong linen bandages, well wetted, are to be so placed, one over each shoulder, that they shall form above a loop, whilst the ends hang down front and back eight inches below the plaster bandage. With new turns of the plaster bandage the linen bandage must now be fastened into its place. After this the loose ends of the linen bandage hanging below the plaster jacket are to be taken up and incorporated into the turns of the plaster bandage necessary to complete the jacket.

Spinal Neurasthenia.

Definition.—A condition of neurasthenia, usually with hysterical symptoms, produced by severe injuries.

Synonyms.—Spinal concussion; Railway spine.

Etiology.—Railroad injuries, falls from hatchways, press of steam from exploding boilers, any violence acting upon the trunk through crushing local force so as to greatly shake and shock the whole system, may produce spinal neurasthenia.

Symptomatology.—The symptoms of traumatic spinal neurasthenia may appear at once after the injury or they may come on insidiously in the person, who has at first believed himself uninjured. The symptoms are subjective and objective. The most important of the subjective symptoms are malaise, loss of ambition, marked increase of nervous irritability, failure of the power of mental and physical labor, depression of spirits, occasional headache, pronounced tinnitus aurium, broken sleep, loss of sexual power, and general failure of health. Almost invariably to these symptoms are added various hysterical manifestations. Probably among these must be classed the extraordinary cerebral attacks which come and go often without obvious cause or explanation. Sometimes these attacks resemble petit mal, in that they consist of short moments of unconsciousness; sometimes the paroxysm is prolonged and consists of an active delirium, which may amount to a furious and aggressive mania. Often the patient has no remembrance of any of these attacks. Distinctly hysterical paroxysms are not rare. Neurasthenic vaso-

motor weakness is common, so that sudden flushings of the face and abrupt outbreaks of sweating are frequent. The muscular irritability is often greatly augmented, and the knee-jerks are exaggerated. Paradoxical contractions may often be produced in the anterior muscles by flexure of the foot; and, as I have seen in some cases, the slightest irritation may cause a general reflex contraction of the erector pilæ muscle, with a consequent "goose-flesh." The activity of the knee-jerk is apt to vary from day to day, and often, when exaggerated, soon becomes exhausted by excitation, so that the muscles by and by fail to respond well when the patellar tendon is repeatedly and rapidly struck. General fatigue will often register itself in the knee-jerk. Ankle-clonus is rare. The sexual power is commonly not altogether lost, but sexual irritability and weakness are usually shown in men by premature emissions. True diabetes may be present and produce its ordinary results. With these various symptoms there are usually pronounced local evidences of the sore back.

The course of this disorder is excessively slow. It has very little influence upon life, but it produces a disablement and much suffering that usually last many years, and which in bad cases may never be recovered from.

Diagnosis.—The diagnosis of this disorder would be very easy were it not for the medico-legal complications which surround most cases. The chief question always is whether the symptoms are real or feigned. Exaggeration of symptoms in many cases is almost a necessity of the situation. The questions to be determined by the physicians are—first, how much exaggeration or feigning exists; second, how much the symptoms are those of hysteria and how much those of neurasthenia; third, how much of local disease there is. The importance of these questions rests upon the fact that traumatic hysteria yields much more readily to treatment than traumatic neurasthenia, and that local symptoms which have already lasted for some time are only to be overcome by very long-continued careful treatment, and are indeed prone to increase rather than decrease.

Treatment.—The foundation of the treatment in these cases consists in absolute rest, mental and bodily. Long-continued rest in bed, with massage and careful use of electricity, is often of the greatest service. This rest must continue for a great length of time, as a little over-exertion will overthrow any good results already obtained. Tonics are of very little service. Narcotics for the relief of pain and malaise often seem called for, but their use is always attended with more or less danger of the narcotic habit. In one of the most successful cases I have ever seen, the patient, a medical man, treated himself, chiefly by drinking three or four pints of strong ale a day. In a person of less resolution this would have resulted in the development of the alcoholic habit. The narcotism of the alcohol and the hops in this case made life endurable, whilst the stimulating effects of the beverage were very useful in maintaining the vital functions.

Results of Excessive Exposure to Heat.

Two distinct bodily conditions, accompanied with disorder of conscious-

ness, arise during exposure to heat: they may be respectively known as heat exhaustion and thermic fever.

Heat Exhaustion.

Definition.—A condition of profound general exhaustion, with paralysis of the vaso-motor system and failure of the general bodily temperature, due to the combined action of heat and exertion.

The sense of weakness which often accompanies exertion in feeble persons during the hot weather represents in the mildest possible form the condition under consideration. In more severe cases there is distinct pallor of the countenance, with failure of the muscular force and of the circulation, accompanied by an overpowering feeling of exhaustion. In the worst cases of heat exhaustion the symptoms develop rapidly, and sometimes with such absolute abruptness that the patient falls in a syncopal condition. Under these circumstances unconsciousness or semi-consciousness may exist, and be accompanied by muttering delirium, great restlessness, facial expression of collapse, rapid, feeble, scarcely perceptible pulse, and a lowered bodily temperature. I have myself known a mouth temperature of 95° F., with complete collapse.

It is essential for the purposes of treatment that heat exhaustion be not confounded with thermic fever, from which it is at once diagnostically separated by the temperature being markedly below instead of above the norm. The only condition readily confounded with heat exhaustion is collapse from cardiac disease, internal hæmorrhage, malarial fever, or other affections occurring in persons picked up in the street and brought to the physician without history. In such cases, however, it is very rare for the temperature to fall as decidedly as in severe heat exhaustion, and peculiar and characteristic symptoms are usually present.

The **treatment** of heat exhaustion consists in the free use of external heat (when it is possible, by means of hot-water baths), the hypodermic injection of atropine, strychnine, and digitalis in order to stimulate the heart and vaso-motor system, with the very moderate internal use of hot alcoholic drinks and ammonia.

Thermic Fever.

Definition.—Fever produced by exposure to heat.

Synonyms.—Heat fever; Sunstroke; Coup de soleil.

Etiology.—The immediate cause of thermic fever is always exposure to heat, natural or artificial. Owing to the interference with evaporation, and the consequent cooling of the body, heat in a moist atmosphere is much more efficient than is dry heat; hence sunstroke is very rare in dry hot climates and frequent in tropical lowlands, as well as in sugar-refineries, laundries, and other places where men work in damp hot air. Exposure to the direct rays of the sun is not necessary, and many of the worst epidemics have occurred during tropical nights.

Whatever lessens the power of the human system to resist external influ-

ences may be a predisposing cause to sunstroke. Chief among these predisposing causes are race, excessive bodily fatigue, and intemperance. The fact that males are much more frequently affected than females depends simply upon the habitually greater exposure of men to heat. Races which by long living in tropical countries have become accustomed to heat rarely suffer from sunstroke.

Symptomatology.—In its severest forms sunstroke is very apt to come on suddenly and without distinct prodromes, although there may be a sense of great distress or of a general burning heat before the loss of consciousness, which may also be immediately ushered in by chromatopsia, or colored vision, the whole landscape being deluged in a blue, yellow, or red light. The unconsciousness ordinarily develops abruptly, and is complete, although very frequently it is associated with muttering delirium. There is usually great muscular restlessness, which in some cases becomes convulsive or is replaced by violent epileptiform convulsions. Sometimes the patient is profoundly relaxed and quiet. The surface of the body, at first dry, often later in the attack gathers upon itself an excessive perspiration, which does not, however, reduce its burning heat. The face is flushed and the eyes are suffused. The rapid pulse is sometimes bounding and apparently strong, although almost invariably compressible; frequently it is feeble and even thready, especially if the symptoms have lasted for some hours. Vomiting is very common; purging is in bad cases almost always present. The whole body is apt to exude a peculiar odor, which is especially strong in the fæcal discharges. The characteristic symptom is the high temperature, which, as measured in the mouth or rectum, may reach 112° or 113°, and is rarely below 108° in cases severe enough for unconsciousness to be present. The urine is scanty, sometimes albuminous, not rarely finally suppressed. The breathing is more or less labored, and often irregular, and toward the last generally becomes more and more shallow. Although at times the patient suffering from thermic fever may be partially aroused by shouting, shaking, etc., the unconsciousness is often absolute. The pupils are variable, sometimes contracted, sometimes dilated.

Even in the most severe forms of thermic fever, as seen in this country, death rarely occurs under half an hour, and usually is postponed for a much longer period. Sometimes it is caused by asphyxia, more frequently by a slow, consentaneous failure of respiration and cardiac action. There is, however, a form of sunstroke rarely seen except in soldiers during battle, in which the death is due to arrest of the heart's action, and is almost instantaneous.

Many years ago, under the name of *ardent continued fever*, the physicians of India recognized a mild form of heat fever, and in 1885, Dr. John Guiteras showed that the typhoid fever of Key West is of this nature. The symptoms are irregular continued fever, without apparent cause or local disease, with a tendency to weakness and the typhoid state, and not rarely with severe but not permanent local, nervous, abdominal, or other disturbance. Writers in India state that in that climate these cases are apt to end in sudden collapse and death.

As was first pointed out by Dr. Comegys, many of the cases of so-called entero-colitis occurring in young children during the hot months are really forms of thermic fever. The symptoms in these cases are high fever, dry tongue and mouth, rapid pulse and respiration, intense thirst, vomiting, purging of greenish, watery, fæcal or serous matters with undigested particles of food, and more or less pronounced evidences of cerebral disturbance, such as insomnia, headache, contracted pupils, delirium, and finally coma. In some cases the bodily temperature rises before death to a point comparable with that which it reaches in sunstroke of the adult.

Pathology.—The results found in the body after death from thermic fever depend much upon the course of the disease and the time at which the post-mortem is made. Owing to the intense heat of the body, post-mortem changes begin in the course of a very few minutes, and some of the lesions described by early writers were really due to beginning putrefaction. When the post-mortem is made immediately, the left heart is found contracted, the right heart usually engorged, the blood semifluid and collected in the venous trunks, with petechial spots upon the arterial coats or scattered through the system.

In an elaborate research made many years ago I proved that the cause of the symptoms and of the lesions of thermic fever is simply the excessive heat. There is in the pons or higher portion of the nervous system a centre whose function it is to inhibit the production of animal heat, and in the medulla a centre (probably the vaso-motor centre) which regulates the dissipation of the bodily heat: fever is due to disturbance of these centres, so that more heat is produced than normal, and proportionately less heat thrown off. Let it be supposed that a man is placed in such an atmosphere and that he is unable to get rid of the heat which he is forming. The temperature of his body will slowly rise, and he may suffer from a general thermic fever. If early or late in this condition the inhibitory heat-centre becomes exhausted by the effort which it has been making to control the formation of heat, or becomes paralyzed by the direct action of the excessive temperature already reached, then suddenly all tissues will begin to form heat with the utmost rapidity, the bodily temperature will rise with a bound, and the man drop over with some one of the forms of *coup de soleil.* Under this view of the case the widespread popular belief, that protecting the back of the head and upper neck from the direct rays of the sun is useful against sunstroke, gains in significance, because it is possible that local heating of the parts spoken of may occur and aid in the production of inhibitory paralysis.

Respiration often ceases in thermic fever through the paralyzing influence of the heat upon the respiratory centres, though in long-continued cases asphyxia may be due to changes in the blood itself. Cardiac rigidity usually occurs directly after death by the coagulation of the myosin, the temperature of the body in sunstroke reaching very nearly the point at which normal myosin coagulates. Excessive exertion so alters the nature of myosin in muscle as to cause it to coagulate much more readily than is normal. During a battle the myosin of the whole body is affected by the excessive effort,

and frequently men are found stiffened in the attitude in which they have been stopped by the bullet, instantaneous death being followed by instantaneous post-mortem rigidity. In sunstroke occurring in battle or in times of excessive exposure death, as has already been stated, may be instantaneous, the man being instantly overwhelmed, because, under the conjoint influence of violent exertion and intense heat, the heart-muscle has suddenly set itself from life into the rigidity of death.

Treatment.—All persons who are constantly exposed to high temperature should keep the bodily health as perfect as possible by avoidance of alcoholic, sexual, or other excesses and of great bodily or mental fatigue. The diet should be largely farinaceous, and the emunctories be kept active by the eating of fruit, the free use of water, and mild salines if necessary. Large draughts of intensely cold ice-water may do harm in heated persons by suddenly chilling the stomach, but cold water taken in moderate quantity, at short intervals, by its action in reducing the general temperature and in aiding free perspiration, does good. The addition of claret or some other substance which mildly stimulates the gastro-intestinal tract and the skin may be of great service in special cases.

In mild cases of continued or subacute thermic fever the basis of the treatment should be the use of the cold bath. The plan adopted by Guiteras at Key West was to wrap the patient in a dry sheet, lift him into a tub of water having the temperature between 80° and 85°, and then rapidly cool this water by means of ice. The time of the immersion lasted from fifty to fifty-five minutes, it being regulated by the thermometer in the mouth of the patient. The patient was then lifted out upon a blanket, the skin partially dried, and the body covered. Guiteras found great advantage by giving a moderate dose of whiskey and thirty minims of the tincture of digitalis twenty minutes after the bath. He states that it is very important to avoid currents of air blowing upon the patient, and to have the bath given in a small warm room. The result of the bath was invariably a lowering of the temperature, a reduction of the rate of the pulse and respiration, and a refreshing sleep. After the second bath the course of the temperature seemed permanently influenced for the better. It was never necessary to give more than two baths in the twenty-four hours, but in some cases they had to be used for many days.

In *acute* thermic fever immediate reduction of the bodily temperature is urgently indicated. Any prodromes should be the immediate signal for withdrawal from exposure to heat, and the use of the cold bath if the bodily temperature be above the norm. As soon as a patient falls with sunstroke he should be carried into the shade with the least possible delay, his clothing removed, and cold affusions over the chest and body be practised. This must not be done timidly or grudgingly, but most freely. In many cases the best resort will be the neighboring pump. In the large cities of the United States during the hot weather hospital ambulances should be furnished with a medical attendant and with ice and antipyrin, so that when a sunstroke patient is reached he may be immediately stripped underneath the cover of the ambulance, and

remedial measures applied during his passage to the hospital. I believe many lives are sacrificed by the loss of the critical moments in the interval between the finding of the patient and his reaching the hospital ward. If circumstances favor, instead of the cold affusion rubbing with ice may be practised. The patient should be stripped and the whole body freely rubbed with large masses of ice. When practicable, a still better plan is to place the patient in the cold bath (50° F.). The employment of enemata of ice-water, as originally suggested by Parkes, may sometimes be opportune. In using these various measures it must be borne in mind that the indication is the reduction of temperature; if the means employed do not accomplish this, they do no good.

The thermometer should always be placed in the rectum or the mouth, the amount of cooling of the axillary surface not being a correct guide. Care is sometimes required not to overdo the use of the cold bath. In the cases which have come under my own observation after the use of the cold bath but little treatment has been required. If, however, the period of insensibility has lasted too long, there may be no return to consciousness, even though the bodily temperature be reduced to the norm. Under such circumstances the case is almost hopeless, but the symptoms may be met as they arise, and a large blister applied to the whole shaved scalp.

When relapses of fever occur, they should be met by the use of cold, but such relapses can generally be prevented by giving antipyrin with small doses of morphine. In thermic fever hypodermic injections of morphine should be given when severe convulsions occur. Venesection may sometimes be advantageously practised in the onset of a severe thermic fever, especially when the means of applying external cold are not immediately at hand; but much care and judgment are required in using the measure. When excessive headache with strong pulse follows immediately upon a sunstroke, free venesection may be required to save the brain or its membranes from an acute inflammation.

Sequelæ.—The mildest sequelæ after thermic fever are inability to bear exposure to heat without cerebral distress or pain, with more or less marked failure of general vigor, dyspeptic symptoms, and other indications of disturbed innervation. In other cases the symptoms are more decided. Pain in the head is usually prominent: it may be almost constant for months, but is always subject to exacerbations. It sometimes seems to fill the whole cranium, but not rarely is fixed to one spot, and I have seen it associated with pain in the upper cervical spine and decided stiffness of the muscles of the neck. With it may be vertigo, decided failure of memory and of the power of fixing the attention, with excessive nervous irritability. When the symptoms approach this point in severity there is usually marked lowering of the general health, loss of strength, possibly some emaciation, and the peculiar invalid look produced by chronic disease. In rare cases epileptic convulsions and very pronounced evidences of chronic cerebral inflammation are present. The symptom which I believe always to be present, and to be of diagnostic import, is the inability to withstand heat. This is shown not only during the summer

months, but in most cases headache and severe general distress are produced by going into hot rooms even in winter.

The lesion underlying these sequelæ of sunstroke is meningo-cortical irritation, with in severe cases distinct chronic meningitis. The treatment is, first, absolute avoidance of any exposure to even moderate heat, combined with intellectual and physical rest; second, the treatment of non-specific chronic meningitis—*i. e.* local bleedings and very free counter-irritation, especially by means of the actual cautery, combined with the internal administration of mercurials and the iodide of potassium in small continuous doses; third, the restriction to a farinaceous, non-irritating diet, and the careful attention to all minor symptoms as they arise. The persistent, merciless use of the actual cautery I have seen achieve extraordinary results in severe cases.

Caisson Disease.

Definition.—A peculiar affection produced by continued exposure to a highly compressed atmosphere.

Synonym.—Diver's paralysis.

Etiology.—The only known cause for this affection is working in caissons during bridge-building or other enterprises in which water is kept out of the caisson by highly compressed air. In passing from these chambers the men go through an outer compartment, so arranged that the pressure can be gradually brought back to the norm. A too rapid passing from the innermost caisson to the outer air is exceedingly deleterious, but no precautions can prevent the disease from attacking a proportion of the workmen.

Pathology.—The pathology of caisson disease is practically unknown. The theory that the symptoms are due to sudden evolution of compressed gas from the blood into the nerve-centres is not proven. It may be that the difficulty lies in the coats of the small blood-vessels. In a few cases in which autopsies have been obtained long after the commencement of the disease disseminated focal myelitis has been found.

Symptomatology.—The symptoms of caisson disease usually develop in from half an hour to two hours after the return of the subject to the surface of the earth. Violent pains occur in the limbs and in the hands, followed in a few minutes by progressive loss of motor and sensory power in the legs. Notwithstanding the anæsthesia may become complete, the pains continue, whilst headache, dizziness, double vision, incoherence of speech, mental aberration, and sometimes unconsciousness, rapidly develop. The patient may convalesce in a few days, or death may take place quickly with apoplectic symptoms, or may follow from paralytic bedsores and cystitis after some months. Usually, however, recovery occurs after a prolonged period of atrocious suffering and motor disablement.

Treatment.—There is no specific treatment of this affection: all that can be done is to meet the symptoms as they arise.

Occupation Neuroses.

Definition.—Localized motor affections produced by the excessive use of groups of muscles in professional or other business pursuits.

Etiology.—Whenever, as in many of the occupations by which men earn their livelihood, there is required an almost indefinite repetition of a more or less complicated set of movements on the part of certain groups of muscles, peculiar local disturbances of muscular action are liable to be developed. The symptoms are usually so entirely local as naturally to lead to the supposition that the affection is purely a peripheral one, but a wider study shows that the disease must be connected with a disordered condition of the nerve-centres. Thus, if the victim attempt to substitute the left hand for the disabled right hand, the disorder usually soon appears in the left hand. Again, general overwork, anxiety, and depressing emotion sometimes play a very distinct etiological rôle, and in a number of instances I have seen the "writer's cramp" appear as the first symptom of a general nervous breakdown. Occupation neuroses may indeed be looked upon as local neurasthenia, having the same relations to general neurasthenia that every local neurasthenia has. (See page 587.)

Occupation neuroses are more frequent in middle adult life than in either extreme of age, and are much more abundant among men than among women, simply because the active period of male adult life is that of labor.

The most common of the occupation neuroses is the so-called "writer's cramp," but the variety of cases which occur in real life is almost indefinite. Professional pianofortists frequently suffer *pianoforte-player's cramp*, which for obvious reasons is more frequent among women than among men. Violinists are liable to a similar affection in either hand; seamstresses, tailors, sailmakers sometimes develop the sewer's cramp. The *telegraphist's cramp* is especially frequent among those who use the Morse machine. *Dancer's palsy*, or cramp affecting especially the muscles of the calf, is very rare among men, being seen almost exclusively in the professional danseuse. *Hammer palsy* attacks chiefly the muscles of the right upper arm, and is especially frequent among gold-beaters, but is occasionally seen among smiths; the latter artisans are also liable to suffer from *chisel cramp*, affecting the left hand, produced by the continuous holding of the chisel or similar instrument. Money-counters, watchmakers, knitters, engravers, indeed the whole list of artisans, are occasionally disabled by peculiar occupation neuroses.

Pathology.—No anatomical changes are known to exist in writer's cramp and allied disorders, and it does not seem worth while to discuss the various theories in detail. The condition is probably one of local neurasthenia, with irritability of the affected centres.

Symptomatology.—The symptoms of occupation neuroses are due to the excessive repetition of movements which require exceedingly fine co-ordination, and differ essentially from the simple muscular exhaustion which occasionally is produced by severe muscular efforts. The characteristic symptom of the

occupation neuroses is, therefore, that, although the disablement for the habitual fine action may be almost complete, muscular power remains, at least at first, for coarse actions. Thus, a man that cannot grasp the pen may readily wield a fifty-pound dumb-bell.

The most marked symptoms of these neuroses are pain and spasm. In 1868, Moritz Benedict stated that there were three forms of occupation neuroses—the paralytic, the spasmodic, and the tremulous. These varieties undoubtedly exist in nature, although not absolutely separated from one another, the distinction between them being simply that in some cases the paralytic symptoms are most marked, whilst in others the spasm or the tremor is the most pronounced. According to my own observation, the paralytic form of the affection is much the most frequent, although some authorities assert that the spasmodic is the ordinary variety.

As it is not possible in the allotted space to describe in detail even the majority of the occupation neuroses, I shall take the most common of them, the *writer's cramp*, as a type of the disorder.

In the paralytic form of writer's cramp the first symptom is usually a painful feeling of fatigue in the arm, which is often associated with formication and numbness, but usually not with true anæsthesia or hyperæsthesia. Only in rare cases can tenderness be found over the nerve-trunks. The pain is always increased by writing, and at last it grows so intolerable as altogether to forbid the use of the pen. With this fatigue and pain there are usually a sense of stiffness and often a distinct muscular resistance when the effort is make to grasp the pen. At first no pain is felt when the arm is not used, and during use the pain is confined to the arm itself; but by and by, if efforts be persisted in, the sense of fatigue becomes more or less permanent, and extends upward from the arm, and may often be felt as a distinct pain between the shoulders. During all this time the power of the muscles for coarse work is in most cases not sensibly impaired, but the execution of any form of fine work is usually interfered with.

Even in the paralytic form of writer's cramp there is a certain amount of irregular spasmodic contraction in the muscles during the act of writing, as is especially shown by the stiffness and, occasionally, by the cramp of the fingers around the pen; but in the spasmodic form of the affection irregular muscular contractions are the dominant symptom. At first these are only simple, slight spasmodic movements of the thumb and first finger, so as to produce an irregular stroke in the writing, but after a time the spasms become stronger and more widespread. By a sudden extension of the finger the pen is dropped, or by a spasmodic action of the opponens pollicis, with abduction and coincident flexion of the index fingers, the pen is rapidly moved from the paper, or occasionally a violent spasmodic flexion of all the concerned fingers holds the pen as in a vice. In extreme cases all the muscles of the forearm are involved; and it is asserted that the muscles of the arm and shoulders may be affected, although I have never seen an instance of this.

Much the rarest form of writer's cramp is that in which tremors are the

most prominent manifestation. When any attempt to write is made, tremblings in the hand and forearm, and in extreme instances in the arm itself, come on. The pen, following the tremors rather than the effort of the will, soon makes nothing but irregular undulating or angular strokes, in which not even the vestige of a letter can be made out. I have never seen a case in which tremors existed as the sole symptom, but I have seen them very marked in the spasmodic form of telegrapher's cramp, and have noted their persistence during almost all forms of voluntary movement, even after the occupation had been abandoned for months.

Prognosis.—The prognosis in writer's cramp is good, provided that absolute rest from the original cause of the disorder can be obtained. The course of the affection is, however, slow, and the disablement has a great tendency to return, even after apparent health has been restored, upon any repetition of the work.

Treatment—In the circumstances which surround most patients the treatment of writer's cramp, as in other occupation neuroses, is troublesome, since, except in the very slightest forms of the affection, total abstinence from writing for a protracted period is essential to the cure. Moreover, the symptoms have a great tendency to recur upon recurrence to writing. Much can be done to prevent the original development of writer's camp, and also relapses, by writing with the arm rather than the hand. Any person who begins to feel discomfort during writing should at once adopt the freer style. A penholder of cork, half an inch in diameter, is of great advantage; the quill pen is said to be superior to any steel pen, and certainly the blunt-pointed steel pen made in imitation of the quill pen is much better than the ordinary sharp-pointed instrument. In free writing the movement is chiefly from the shoulder-joint; for the development of the method Gowers suggests that the learner should draw a line across a sheet of paper with the arm moved as a whole from the shoulder; then that he should make a similar but wavy line; then increase the wavy character of the line and then the slope of the waves, so that at last he forms the line like a series of *m*'s—*mmmmm*—the letters being joined together. From these letters the transition to other letters will be easy. A person learning this method should learn to form a whole line of words without lifting the hand from the paper, the hand holding the large pen-holder lightly. Much better even than this method of writing is the use of one of the type-writing machines.[1] When it is necessary for the subject of the disease to continue the writing at all hazards, the left hand may be employed. In writing with it, it will be found easier to reverse the lines—*i. e.* to write with the slope from

[1] Authors and other persons who compose as they write will find an extraordinary saving of nerve-force and time by the use of the short-hand amanuensis. The habit of dictation can by most persons be readily formed: it must be remembered, however, that the person will dictate as much in one hour as he will write in three, so that the dictation means more expenditure of brain-force in the same period of time than occurs in composing by writing. The author who dictates must work fewer hours a day, but even then will accomplish more than he would with his own pen.

left to right. Usually the left hand soon develops cramp, but occasionally it remains free if great care be taken not to overwork it.

The direct treatment of the arm suffering from writer's cramp yields very unsatisfactory results. No internal medication is of any use, save only as it may benefit the general health of the patient and overcome the neurasthenic tendency apt to exist in these cases. Rest, massage, and electricity are the three agencies at hand. As already stated, rest must be absolute and long continued. Massage seems to be of distinct value. Electricity has been very largely employed, and is by some authorities strongly commended, by others spoken of with despair. It seems, in fact, to do good in some cases, but very often its influence is scarcely perceptible. Faradization may do harm, as the muscles are commonly irritable; it rarely, if ever, does good. The best application is the long-continued use of a mild current of galvanic electricity passed down the nerve of the affected member, of just such strength as to be distinctly but not painfully perceived. A small positive pole should be placed over the nerve-trunks in the groove of the inside upper arm, whilst the hand rests upon a large well-wetted sponge connected with the negative pole.

Headache.

Although pain in the head is a symptom, yet it so frequently constitutes the main complaint of patients that it seems necessary to give it here separate consideration. For the purpose of brief discussion headaches may be arranged in four classes, as follows:[1]

1. Organic headaches, due to disease of the brain or its membranes.
2. Toxæmic headaches, due to a poison either produced within the body or received from without.
3. Sympathetic headaches, due to some peripheral lesion.
4. Headaches which are not included in the other groups, and to which the name of *nervous* may be given, with the understanding that the title carries no etiological significance. In this group are placed many headaches of whose ultimate cause we are ignorant.

Unfortunately, it is not possible, by any character of the headache itself, to

[1] It seems proper here, also, to give anew a warning against mistaking the pain of an acute glaucoma for a headache. The pain of glaucoma, which may develop abruptly, often centres in the eyeball, but may seem to have its chief focus in the supraorbital notch: not rarely it shoots over the forehead and into the cheek and temple, reaching even to the occiput, and filling the whole side of the head with agony. If, as usually happens, there be fever, with severe vomiting, the patient may be thought to be suffering from a bilious or malarial attack, and the eye be irretrievably damaged before the true nature of the paroxysm is discerned. This can be avoided by paying attention to the following points: the eye shows evidences of inflammation in congestion and swelling of the conjunctiva and even of the lids; the cornea is somewhat misty, presenting the appearance sometimes spoken of as "steaminess," and its sensitiveness to the touch of a camel's-hair pencil is diminished; the pupil is sluggish, often somewhat dilated; on palpating the two eyeballs simultaneously with the forefingers the affected eye is felt to be the harder, and the patient often complains of a sense of tension in the ball; vision is less acute in the affected than in the sound eye. In case of doubt it is the duty of the practitioner to call in an oculist at once.

decide to which of these classes it belongs. It is impossible to diagnose the nature of a headache by a study of the headache itself. The organic headache is of course very persistent, but its true nature is only to be made out by the discovery of some other symptom of organic brain disease. Exclusion of other possible causes may lead us to suspect the origin of such a headache.

Toxæmic Headache.—Of the toxæmic headaches the most important are the malarial, rheumatic, lithæmic, alcoholic, caffeinic, gastric, and other headaches due to alterations of the blood produced by diseases of the kidneys, heart, and lungs.

The derangement of the health produced by malaria may cause nervous headache, but the specific malarial headache occurs periodically, usually in the form of the so-called "brow ague," in which an intense pain rapidly develops at fixed hours in the immediate neighborhood of one supraorbital foramen. This pain lasts from five to ten hours, is often of frightful intensity, and may or may not be associated with fever and sweat or other indications of a malarial paroxysm. The nature of such a headache is recognized by its periodicity and by its yielding to very large doses of quinine.

The rheumatic headache is ordinarily heavy, aching, may take the form of the shooting pains of a neuritis, and may be without any character indicating its nature. Usually it is to be recognized by the marked soreness of the scalp which accompanies it. In doubtful cases the effect of the salicylates should be tried.

The lithæmic headache in its usual form is dull, heavy, and often worse in the morning. It is, sometimes, however, atrociously acute, paroxysmal, and continuing with such persistency as to suggest an organic cause. Especially is the organic picture complete in those cases in which the headache is associated with vertigo, staggering, or even epileptoid spells. I believe that usually in these cases there is either a gouty deposit in, or a gouty inflammation of, the meninges. The diagnosis of a lithæmic headache rests on the existence of lithæmia and the exclusion of other forms of headache.

The abuse of coffee and tea, especially in overworked women of neurotic temperament, is a frequent cause of severe obstinate headaches which are not reached by any treatment until total abstinence from caffeinic drinks has been enforced for a month.

The dull, heavy headache of habitual indigestion, with hepatic torpor (the so-called "biliousness"), is usually frontal, may be occipital, and is often associated with defective vision, giddiness, and great depression of spirits. Severe head-pain is sometimes due to gastric acidity. This headache is often ushered in by sudden blindness and dizziness, and usually yields at once to the administration of ammonia and bicarbonate of sodium.

Headache may be the only complained-of symptom of diabetes, uræmia, and the imperfect aëration of the blood produced by cardiac or pulmonic disease. The form of these headaches varies indefinitely: the cause is only to be made out by discovering the distant organic lesion. It must be remembered that in advanced contracted kidneys the urine may be persistently free from

albumin, the disease being revealed only by the persistent low specific gravity. In obscure chronic headache occurring in children the condition of the heart should especially be looked after.

Sympathetic Headache.—Almost any peripheral irritation may in rare cases produce pain in the head, but the ordinary sympathetic headaches are those due to eye-strain and disease of the nose, though it is sometimes doubtful whether the nasal headache is not at least in part produced by imperfect respiration. The headache of eye-strain is extremely frequent, is often frontal, not rarely occipital, may seem to have no connection with the eye, or may take on the character of almost any form of headache, even that of typical migraine. It is usually aggravated by the use of the eyes, is apt to be severe in the morning after an evening spent in a place of amusement, or may coexist with head-pain of other character. The only method of detecting its nature is the discovery and subsequent correction of the optical difficulty. The nasal headache is usually frontal, may be vertical or occipital, and may take on the form of a migraine. Its nature is often immediately to be detected by the tenderness of the inner wall of the orbit when pressed upon by the finger, or by the pain caused by touching the middle turbinate bone with a probe. In any case of chronic headache with chronic nasal disorder the latter should be carefully treated.

Under the title of *nervous headache* may be grouped anæmic headache, congestive headache, the headache of brain-exhaustion, the hysterical headache, migraine, and certain rare headaches whose nature is obscure, but which may be designated by the misnomer of idiopathic headaches.

The headaches of anæmia and of exhaustion are very similar in their nature. Each of them may be accompanied by flushing of the face, reddening of the eyes, or sense of fulness of the head, which may mislead the practitioner into supposing that there is true brain congestion. Often the headache in these cases is rather a sense of distress and weight than a true pain.

The headache of acute cerebral hyperæmia is very rare unless it arise from an exposure to excessive heat, traumatism, or an organic cerebral disease. It is to be recognized by the pulsation of the carotid, the strong full pulse, and the tendency to coma or delirious disturbance.

Most hysterical patients suffer from severe headaches of various character. Almost characteristic is the so-called *clavus*, a pain situated in the middle of the top of the head in a point so small that it can almost be covered with the point of the finger. The hysterical headache is apt to be increased at the menstrual period, and to be suddenly removed by pleasurable mental excitement.

Migraine, *Megrim*, or "*sick headache*," has for its essential feature a paroxysmal headache, which in the great majority of cases appears first at early puberty, and continues in women up to the menopause or in men to advanced middle life. In its details the paroxysm varies in different individuals, but usually conforms more or less to the following type: For some hours before the attack the patient suffers from malaise, often with chilliness and a sense of languor, or in rare cases experiences a condition of peculiar emotional and

mental activity. The attack may or may not be ushered in by distinct prodromes. The pain is unilateral in the great majority of cases, and is referred to the frontal region, having the focus at or about the supraorbital foramen, or more rarely in the eye itself. It comes on gradually, becoming more and more intense for hours, until finally it is unbearable. It is generally described as boring in character, often throbbing, and only in very rare instances as shooting into the jaws and the neck. Sometimes the occipital region may be the seat of the pain. At about the time that the pain reaches its greatest intensity nausea followed by vomiting develops. The vomiting is usually repeated, and is attended with great bodily depression. The matters ejected are the contents of the stomach, followed by mucus and bile. Immediate relief often follows the vomiting. In some cases the patient now falls asleep, and wakes free from the headache; in other cases the headache gradually subsides. The whole paroxysm lasts from five hours to two or even three days. During the height of the attack of migraine there is generally intolerance of light and sound; and yet, according to E. Souila,[1] occasionally there is an intense craving for light, and even for noise.

Although the general features of an attack of migraine usually conform to the account just given, there are certain symptoms which are occasionally present and demand more detailed description. In some cases the prodromes are very marked, and include distinct disturbance of a special sense. The sight is the most frequently affected, and next after it the smell. It is stated that in very rare cases a taste comparable to that produced by passing an electric current through the mouth is prodromic of a paroxysm of migraine; more common is a peculiar bitter taste in the mouth, which is generally referred by most patients to disorder of the stomach. This taste has seemed to me to be closely connected with a peculiar, excessively disagreeable odor of the breath, which in turn appears to be due to the excretion of some sulphuretted compounds. Jewelry about the person may be very distinctly tarnished during an attack.

As the affection has come under my notice in America vaso-motor disturbance is not usually pronounced; but Eulenberg distinguishes two varieties of migraine which he says are typical. In the one, during the height of the paroxysm, upon the affected side the face is pale, the pupil dilated, the temporal artery hard, and the temperature of the external auditory canal is reduced one to two degrees Fahr. Pressure upon the carotid on the side of the pain now increases the pain, while pressure upon the artery on the opposite side of the neck tends to relieve it. Toward the end of the paroxysm the face and ear become red, with a sensation of heat and an absolute rise of the temperature; at the same time there is in some cases a contraction of the pupil. In the second variety of migraine described by Eulenberg there are throughout the paroxysm evidences of vaso-motor depression. Always at the height of the attack the face is red and hot, the conjunctiva injected, and the lachrymal secretion increased. The ear of the affected side is distinctly hotter than its fellow, and the sweat is very abundant at the immediate site of the pain, or sometimes

[1] *Thèse*, Paris, 1884, No. 35.

the sweating is unilateral. By compression of the carotid upon the affected side the pain is lessened, but it is increased by pressure upon the artery of the opposite side. It is affirmed that in some cases the dilatation of the arteries and veins can be detected in the fundus of the eye. Toward the close of the attack the face becomes pale.

Dr. Anstie of London states that the pain-storm or migraine may be accompanied by a temporary whitening of the hair at the seat of the pain, and that this paroxysmal bleaching, so to speak, finally leads to a change of color.

I have never been able to confirm the existence of the described varieties of migraine or of the trophic changes just spoken of, nor yet have I ever seen a case in which migraine has produced, by continually recurring paroxysms, a condition parallel to the status epilepticus, such as has been described by Dr. Féré of the Bicêtre.

Visual prodromes are pronounced in *migraine ophthalmica* (*hemiopia periodica*). The most frequent form of visual disturbance is an amblyopia, accompanied by vivid scintillations passing zigzag, like the lines of a fortification, over the field of vision. When hemiopia occurs it may be either monocular or binocular; sometimes it is lateral; in other cases it occupies the superior half of the visual field. In the binocular form a lateral half of the field is attacked. The vision is completely abolished in the affected portion of the field, although the total acuity of vision may remain normal. This sensory disturbance very rarely occurs except in persons who have long suffered from the migraine. In some cases it is preceded by headache, but usually it develops suddenly as the beginning of the paroxysm; occasionally instead of hemiopia a central scotoma is the dominant symptom. Rarely this scotoma merges itself finally in a hemiopia. In rare cases these disturbances of sight are replaced by distinct visions or hallucinations. The olfactory disturbance which sometimes ushers in a migraine is generally the sense of a peculiar odor, like that of osmic acid, etc. The auditory prodrome has been variously described as like the sound which is produced when a marine shell is applied to the ear, or as a gurgling similar to that which is heard when water enters the ear during bathing.

The psychical symptoms which accompany a migraine are usually not severe, but in rare cases they are very marked, affecting especially the emotional nature, causing in one instance profound melancholy and depression, in another vivacity; in either case there is commonly an excessive irritability. During the attack, according to the measurements of O. Berger, there is a condition of hyperæsthesia of the skin of the face, at least so far as the sense of locality and the electric sensibility are concerned. Certainly in most cases there is no excessive sensibility to pressure, and indeed commonly the pain is more or less distinctly relieved by firm pressure. There is usually no tenderness either during or after the attack at the point of emergence of the nerve from the bone, although in some cases a certain degree of general tenderness of the face is produced by a violent paroxysm. A remarkable but very rare complication of migraine is an aphasia coming on during the height of the attack.

It does not seem worth while to discuss the numerous theories which have been brought forward as explanatory of migraine. The paroxysms are evidently of the nature of nerve-storms. Trousseau many years ago called attention to the relations of epilepsy and migraine, and cases do occasionally occur in which the migraine and epilepsy coexist, or even in which one seems to replace the other. In the vast majority of cases, however, no relation can be traced between the two disorders. Much more definite, though very obscure, seem to be the relations between migraine, gout, and hay fever.

Migraine is to be recognized by its paroxysmal occurrence, by the almost invariable history of inheritance which accompanies it, and by the absence of other causes for the headache. Relief from the paroxysm can often be obtained by the use of antipyrin, phenacetin, and caffeine alone or in various combinations, taken during the prodromic stage. Most migraine subjects bear opium badly, but the combination of deodorized tincture of opium and the bromide of potassium in proportionate doses (♏xx to gr. xlv) almost invariably gives relief without causing narcotism or vomiting. Of course great care is necessary to avoid the production of the opium-habit.

In the general treatment of the disorder it must be remembered that, while migraine cannot be cured, the nerve-storms become infrequent during high health, and that between the attacks care must be taken to build up the patient and to correct gouty or other disease tendencies. The continuous, prolonged administration of the extract of cannabis Indica, in dose just insufficient to produce physiological effect, has in many cases of migraine great influence in abating the number and severity of the paroxysms. Peripheral irritations, such as eye-strain, may greatly aggravate the disorder, and must be carefully prevented.

Sleep: its Disorders and Accidents.

Led chiefly by theoretical considerations, some neurologists have distinguished sleep, stupor, and coma as essentially diverse conditions, readily to be diagnosed in the sick-room. These states are, however, simply the outcome of different degrees of completeness in the suspension of the functions of the cerebral cortex, and are not separated by any fixed lines. Nor is the unconsciousness of anæsthesia an isolated thing: it is simply a suspension of cerebral function in which a known chemical agent is the cause of the paralysis. Although in the sick-room every grade can be found between light and heavy slumber, between heavy sleep and stupor, and between stupor and coma, yet for discussion we may arbitrarily separate them: *sleep*, that condition of unconsciousness in which the subject is readily aroused, and when aroused is easily kept awake by ordinary external stimulations or by his will-power; *stupor*, that condition in which the subject is aroused with great difficulty, and when left to himself relapses into unconsciousness; *coma*, that state in which it is impossible by external irritation to restore consciousness.

It seems necessary to say here a few words in regard to the immediate causes of lapses of consciousness, since certain authorities claim that they are due to changes in the circulation. No proof of this has, however, ever been

given. It is true that during sleep there is more or less pronounced cerebral anæmia, which on awaking is replaced by turgescence of the cerebral vessels. It is a universal law that cessation of functional activity is immediately followed by lessening in the amount of blood in the part. I conceive, therefore, that the sleep or cessation of functional activity is the cause of the bloodlessness, and not the bloodlessness the cause of the sleep. Insomnia may be connected either with excessive anæmia or with excessive congestion of the cerebral cortex. The best explanation of sleep, then, is that when exhausted by effort the cortical brain-cells pass into a condition of functional inactivity, during which their power of further effort is recuperated. Because consciousness is the expression of functional activity in these cells, therefore when these cells do not exercise their function there is unconsciousness—*i. e.* sleep.

In treating of sleep and its disorders I shall divide the subject into three parts: first, abnormal wakefulness; second, abnormal somnolence, or morbid sleep; third, accidents or groups of symptoms which occur during sleep, and which are not elsewhere spoken of in this book.

Abnormal Wakefulness.—In *simple insomnia* the form of the sleeplessness varies. In some instances the subject is simply unable, when bedtime comes, to go to sleep. In other cases he goes to sleep readily, but in the course of two or three hours wakes, and is unable to slumber again. The latter form of insomnia, in my experience, is not commonly the precursor of severe mental affection, but is often obstinate.

Insomnia may be prodromic of various diseases of the brain. It is very common in the insanities. It is also present not rarely in such general organic brain diseases as general paralysis of the insane, but is seldom a symptom of tumor or other focal brain-lesion. It may be produced by various diseases of organs other than the cerebrum. It may exist, however, in its most aggravated form without other evidences of cerebral disturbance, and in some cases cerebral exhaustion, and even more severe mental symptoms, are without doubt produced by the loss of sleep. The diagnosis of the cause of an insomnia is to be made by exclusion. If other symptoms of cerebral disease are wanting, the condition of the heart and kidneys should be carefully examined, because latent disease of these organs occasionally has sleeplessness for its chief manifestation. When no disease of the brain or other portions of the organism can be made out, the diagnosis of simple or functional insomnia must be settled upon.

The treatment of insomnia requires much tact, and at best is often very unsuccessful. The foundation of it consists in the removal of the condition which is the cause of the insomnia. If the wakefulness be lithæmic, antigout treatment must be instituted; if it be due to a local or general neurasthenia, this must be combated.

In rare cases of active determination of blood to the head local abstraction of blood may be required. More commonly insomnia seems to be connected with exhaustion; at least it is not infrequent to find that food taken at bedtime, or when the patient wakes sleepless in the middle of the night, has

a very beneficial effect. This food usually acts best when it is hot and easily digestible. Bouillon thickened with some nutritive starchy material, oyster soup, milk punch warm, have often been found serviceable. Alcohol in the form of whiskey or brandy, taken with a little hot water, is often efficient. It may well be that in these cases the good is achieved by stimulating the stomach and drawing excitement, nervous and arterial, from the brain. Certainly midnight wakefulness may sometimes be overcome by a single glass of hot water taken in the middle of the night.

In some patients massage taken just before the time of sleep has a distinct quieting influence, whilst upon others it acts as an excitant. A procedure which I have seen act very happily in insomnia with active congestion of the brain is to allow the patient to sit in a bath of very hot water and have a cold douche on the head from three to five minutes. The effects of exercise vary in different individuals, and the amount ordered must be judged of by the result. In neurasthenic insomnia tire usually causes wakeful nights. In most cases of insomnia it is essential that intellectual activity and emotional excitement during the latter third of the day be avoided; that the supper taken be light; that the patient sleep by himself or herself in a well-ventilated apartment; and that no caffeinic drinks be used after the morning meal.

The treatment of insomnia by drugs is always to be avoided as much as possible. In some cases, however, these agents have to be employed, and sometimes it is possible by making a strong nightly influence for a few weeks to break up the habit of insomnia and then gradually to withdraw the remedy. Hypnotic remedies are numerous, and in long-continued insomnia it is better to periodically change them. Sulphonal has seemed to me the least harmful, though by no means the most certain of the class. It should always be given in the form of the powder about an hour before the expected time of sleep. The compressed pill of sulphonal, so much used, very frequently passes through the intestines without change. Chloral still remains the most efficient remedy of the class. Chloralamide has some virtue, but is uncertain. Urethan seems to be even less active, whilst paraldehyde is so disagreeable and irritating to the stomach that it is only to be employed on rare occasions. Except in the case of old people opiates are to be avoided for fear of producing a narcotic habit.

Morbid Sleep.—Morbid somnolence may be due to an almost infinite number of causes, including various acute diseases and poisonings. As almost all of these affections have been sufficiently described in this work, it only remains to say a word in regard to the so-called "*Nelavan*," *African hypnosis*, or *African sleeping disease*, an acute, very fatal fever, the most characteristic symptom of which is excessive somnolence. It is endemic on the west coast of Africa, but appears to occur epidemically in some of the West India Islands. It attacks the negroes especially, but has in a number of instances decimated regiments of French troops. In most cases it comes on gradually, but it may begin brusquely. There is at first a slight frontal headache, with a sense of

constriction in the forehead, attended by a mild fever. The vision may at this period be disordered. The gait becomes irregular, and not very infrequently there is a distinct ataxia. Even during the first hours of the headache an intense desire for sleep is manifested. This continually increases until the patient is overpowered by an irresistible somnolence. During the period of sleepiness the strength fails, the spirits are depressed, and there is some fever, but usually neither diarrhœa nor constipation develops, and the forces of the circulation are well maintained. The somnolence when once developed continues to become more and more intense, and the patient gradually sinks into a profound coma, which may pass quietly into death: violent convulsions and sloughing bedsores are liable to develop. There are no pathognomonic post-mortem lesions unless it be swelling of the glands.

Omitting toxæmic somnolence, most of the cases of morbid sleep seem to be referable to one of five groups:

Group 1. Sleep due to reflex irritations.

Group 2. Narcolepsy, or idiopathic sleep of unknown cause.

Group 3. Hysterical and epileptic sleep.

Group 4. Sleep of insanity.

Group 5. Somnolence connected with organic brain disease.

Of the third and fifth of these groups sufficient has already been said. (For Group 4 see article on Mental Disease.) Reflex sleep is very rare, but Dr. Katerbau has recorded a case in which a seventeen-year-old Jewess, who had slept four days and nights, immediately awoke after the passage from the rectum of a knot containing twenty-four round-worms, whilst Dr. Mayer has related a similar case of a boy nine years old.

Narcolepsy.—The cases of morbid sleep which are here grouped together under the name of narcolepsy vary in the intensity of their symptoms from drowsiness to a sleep which ends in death. It is most probable that the cause of the sleep varies, and that several distinct affections are represented in the group, and that some of the recorded cases have also been instances of hysterical or organic disease. The best that can be done at present is to separate the cases into three subgroups, which are not very clearly distinguishable, and indeed are probably closely connected by intermediate cases. In the first of these groups the subject passes many hours in what seems to be the ordinary slumber. In some cases the sleep comes on daily, in others at longer intervals. In some instances there is a perpetual drowsiness, in others the patient when awake is not sleepy.

The second class of cases comprises those in which the paroxysms of sleep come on at irregular intervals and continue for days, as in a Jewess who shortly after her marriage fell into a prolonged sleep which ever afterward recurred periodically. The average length of the sleeping period was five and a half days, the longest time that she had ever slept being seven days. The intervals of wakefulness lasted from two to twenty days, during which time she did not sleep at all or had only a very little restless slumber.

A third class of cases is that in which the sleep comes on without apparent

cause, and becomes more and more profound until the patient dies. These cases as recorded seem to have been due to brain congestion, and some have yielded to very free venesection. Other cases have been instances of cerebral organic disease, but there remain cases like that reported by Dr. S. Weir Mitchell, in which death after a prolonged seemingly causeless sleep has resulted, and in which a most careful post-mortem examination has failed to detect any lesion.

Accidents of Sleep.—*Sense-shock*, so called, occurs in hysterical women and overworked men, usually whilst passing from sleep to waking. A sensation like an aura rises from the feet—or, more rarely, from the hands—and passes upward to the head, where it disappears in the sense of a blow or shock or of a bursting in the head. Not rarely at the time of the explosion the patient hears a loud noise or sees a vivid flash of light or perceives a strong odor. In some cases two or even more of these sensory manifestations are present together. The paroxysm may occur during the daytime. These attacks have no serious significance, and there is no special treatment.

Night Palsy consists simply of a feeling of numbness in one or more extremities of the body when the sleeper awakes. The most common seat is one arm, but the symptom may be hemiplegic or may affect the whole body. I have seen it in hysterical women, especially after the climacteric. Dr. S. Weir Mitchell speaks of it as occurring in locomotor ataxia. It is certainly not indicative of failure of the circulation, and seems indeed to have no especial significance.

Somnambulism.—Somnambulism is defined by Dr. H. Barth[1] to be a dream with exaltation of the memory and of the automatic activity of the nerve-centres, combined with absence of consciousness and spontaneous will. It is common for a sleeper to give evidence of his thoughts by movements and muttered words: a step beyond this and the dreamer acts. Every grade between the slightest dream-movement and the most active sleep-walking exists; but whenever a dreamer rises from his couch he may be said to be a somnambulist.

If the somnambulist be approached, his eyes will be found to be closed, or, if open, they, with the rest of the face, are impassive and without expression, paying no attention to the brightest lights, and appearing to have no power of sight in them; yet obstacles are avoided, narrow places passed through, feats of balancing performed, and numerous complicated movements made so perfectly that the bystander can hardly persuade himself that the sleeper is not awake. When seized hold of, the somnambulist usually resists with vigor. Left to himself, after wandering for a greater or less length of time he returns to his bed, covers himself up, and sinks into the quiet forgetfulness of normal sleep.

In the milder forms of somnambulism it is sometimes possible to turn the thoughts of the sleeper by speaking to him, and in obedience to a firm command he will return to his bed without waking. Acts the most difficult and

[1] *Du Sommeil non-naturel*, Paris, 1880.

complicated are often performed by the somnambulist, and even murder has been done in obedience to the impulse of the dream.

The so-called *night-terrors* of childhood, although frequently spoken of as a distinct affection, are, in truth, only a form of somnambulism, or, in rare cases, epileptoid seizures. Nothing is more common than for a young child to go in the night to its parent's bed, trembling with terror or weeping bitterly, with the statement that it has had a bad dream. Such a dream may be so vivid as completely to enchain the attention, and if at the same time there be outward manifestations of the overpowering emotions from which the child is suffering, a paroxysm of night-terror results. Very frequently during the paroxysm the child shows terror of some one object—a cat, a dog, a white elephant, a monster of some kind, is indicated by its incoherent cries. In a large majority of cases night-terrors are of no more serious import than an attack of somnambulism. They often depend upon gastric irritation or too much emotional excitement during the day. In a few recorded cases the cause of the attacks has been intestinal worms. Those rare night-terrors which are due to serious disease can only be distinguished by their tendency to continually recur and by their concomitant symptoms.

I have seen one or two cases of night-terrors occurring in adults which by their frequency and severity absolutely destroyed the usefulness of life, and were not removed by any of the innumerable treatments instituted by various physicians. It is stated that the habit of somnambulism can sometimes be broken up by suddenly awaking the patient with a shock.

The general treatment must be that of neurasthenia and hysteria. In the case of night-terrors of children special care should be taken to remove intestinal worms, glandular swellings, or any other possible source of local irritation. The use of stimulating foods and of caffeinic drinks must be avoided, and only light suppers should be allowed.

Correlated Disorders of Memory and Consciousness.

All functional acts are accompanied by, or dependent upon, a nutritive disturbance. It matters not whether the functional act be connected with thought, consciousness, or secretion, the generation of nerve-force by the ganglionic cell and its transmission by nerve-fibre are accompanied by nutritive changes in these bodies. A nutritve act, although temporary, has a distinct tendency to impress permanently the part implicated; and this tendency is especially pronounced in nervous tissue. All nervous tissue is, therefore, liable to be permanently affected by its own functional actions. This, it must be remembered, applies equally to normal and to pathological activities. Thus, the child in learning to walk by repeated efforts trains the lower nerve-centres until, in response to appropriate stimuli, a definite series of nervous discharges and transmissions occur independently of the will, and walking becomes automatic. This, in short, is the history of all training, mental and physical. All nervous tissues therefore, have memory—*i. e.* the faculty of being permanently

impressed by temporarily acting stimuli, the thing remembered being, in fact, the functional excitement.

The recognition of the universality of memory in nerve-tissues is of great importance in the consideration and treatment of disease. Thus, an epileptic fit is produced by a peripheral irritation. If that peripheral irritation be at once removed, the fit does not recur and the patient is cured. If, however, the irritation be not soon taken away, but produces a series of convulsions, the fits may continue after the removal of the irritation, simply because of the permanent impress which has been made upon those cells in the brain-cortex, whose discharge of nerve-force is the immediate cause of the epileptic paroxysm. The nutrition of the cells has been so altered that at irregular intervals they fill up and discharge nerve-force.

Owing to this power of memory a physical habit may become so permanently engrafted upon the nervous system that the patient is unable to control it. An example of this is seen in the so-called habit choreas: movements at first controllable, mere bad habits, become at last fixed, not to be altered by any power. The hysterical woman who gives way to hysterical nervous impulses thereby strengthens their hold upon the system, so that in time she may lose all power of control over the lower nerve-centres. Moral habits are formed in obedience to the same law. Self-control, enforced at first by discipline, may become at last in the child an integral function of the nervous centre by a method parallel to that by which an accidental epilepsy is converted into a permanent disease. In the prognosis and treatment of disease, as well as in the training of the young, the full recognition of the power of habit—*i. e.* of unconscious memory—is a matter of vital importance.

What is true of the lower nerve-centres and fibres is true of the upper ones. Intellectual acts or thoughts and perceptions tend to stamp themselves upon the centres connected with them, and when the function of the nerve-cell is connected with consciousness the changes which occur in the nutrition give origin to conscious memory—*i. e.* to memory in the usual sense of the term.

The methods of ordinary mental action seem to indicate either that special ganglionic cells are set apart for special forms of memory, or else that the single ganglionic cell is capable of distinct acts of memory. Thus, one individual will remember one class of facts with great ease, to the exclusion of other matters, whilst the second person may readily remember those affairs which the first naturally forgets. Disease sometimes dissects out, as it were, the different forms of memory, isolating one from the other. It is well known that in the loss of memory which accompanies senile changes of the brain or is a prominent symptom in the first stage of general paresis the power of remembering recent events may be lost, although the recollection of affairs which happened in childhood days is far more vivid than in the normal condition of the individual. Under these circumstances it may be considered that the ganglionic cells have lost their capability of receiving impressions, but not that of recognizing impressions which were made long before. The separation of different forms of memory is, however, distinct from this. Thus in a case

of dementia recently under my care memory for ordinary events was almost entirely lost, and yet a joke or a ludicrous story would be remembered in all its details without apparent effort. It is well established that one form of memory—namely, that connected with language—has in most individuals a definite brain-location; and it may be that each variety of memory has its own territory.

In considering the disorders of memory I shall omit the discussion of disturbances of specialized forms of memory, because the most important of these, aphasia, will be elsewhere fully elucidated by Professor Osler. (See page 701.)

Failure of Memory is a frequent symptom, which, when not due to obvious acute disease, is a strong indication of an organic affection of the brain, although a slight degree of it may be produced by simple brain-exhaustion. In some cases careful examination is needed to detect it. Under such circumstances the physician must question the patient as to the small events of the last twenty-four hours, and not be misled by that vividness of recollection of the long past which sometimes causes the sufferer to declare that his memory is even stronger than normal. It is evident that what is first lost is not the power of recalling impressions already made, but of receiving or taking new impressions. Old impressions come readily into the scope of consciousness, but passing events leave no stamp upon the brain-cells. In doubtful cases of general paralysis of the insane failure of memory is of special value in enabling us to distinguish the organic insanity from functional mental disturbances which may simulate it. According to my own experience, failure of memory which is not accompanied by paralysis for the time being of all the functions of the mind, as in insanity, is of serious import in proportion to its completeness.

True Exaltation of Memory—*i. e.* exaggeration of the power of receiving new impressions or acquiring new facts—is a rare phenomenon, which must be sharply distinguished from the peculiar exaggeration of recollection spoken of in the next paragraph. It is sometimes present in the insomnia due to exaltation of the cerebral cortex, when it is an extremely alarming symptom. Cases are also on record in which it has preceded an attack of apoplexy or even of general paralysis.

As has already been stated, a memory is possessed by all varieties of ganglionic nerve-cells, but that intellectual function to which the name is usually restricted is so closely related with consciousness that we can scarcely conceive of its existence without consciousness; nevertheless, the connection of memory with dreaming shows that it is a separate function from consciousness.

There are a good many reasons for beliving that the impressions of all events with which an individual has been connected are indelibly recorded upon his brain-tissue, although he may not be able to bring such impressions into conscious perception. At the approach of death or under the stimulation of disease at a time when consciousness is wanting persons will frequently speak in foreign tongues, recite passages of prose or poetry long since forgotten, or give detailed accounts of events that occurred in their earliest child-

hood, and of which they have in their normal condition not the slightest remembrance. It would therefore appear that two distinct functions or acts are involved in conscious memory—one the preservation of the records, the other the dragging out of such records into the light of consciousness and their recognition by the personality of the man. In certain diseases when consciousness is obliterated the connection between the stored records of the cerebral cortex and the automatic speech-centres is so close that the latter act in obedience to the records, and the unconscious patient speaks in an unknown tongue or relates occurrences of which he has no conscious memory.

When the link that binds consciousness to memory is broken by disease consciousness may exist without memory. Under these circumstances consciousness is isolated from the past, although the past may still be connected with the present by an automatic unconscious memory. This is illustrated by the famous case of the French soldier, who, as the result of a wound in the head, was subject to attacks lasting many hours in which he had no sensitiveness of any part, although if put in the position of marching or writing or smoking, etc. he would go through the whole complicated series of acts necessary for the performance of these acts, all of the time evidently unconscious of what he was doing and changing from one performance to another as he was taken hold of and put into a new position.

The sense of *personal identity* is dependent upon the existence of memory and consciousness. The unbroken chain of events recorded from an indefinite past correlated with the consciousness of the present gives the realization of the unity of the present with the past. This sense of personal identity is destroyed by a complete loss of memory, which loss may be abrupt and be unaccompanied by impairment of consciousness or of rationality. I have seen this association of symptoms continue for several days after a sunstroke, so that the patient, who had been brought by ambulance into the hospital, was unable, after he had recovered his mental faculties and was perfectly rational, to give any clue to his personality which could lead to his identification.

Double Personality, the condition in which the subject feels as if he were two distinct personalities, the one alternating continually with the other, has no connection with loss of personal identity nor yet with double consciousness. Its explanation is very difficult: it is occasionally seen as the result of hasheesh or other poisonings, and also in insanity, in which affection it may become the basis of a delusion, as in the case of a patient of my own who was overwhelmed by the constant doubt whether he was himself or his own double.

Double Consciousness, so called—*periodical failure of memory*, or *periodic amnesia*—is a disorder of memory which also involves all the intellectual functions and the character of the individual. In a typical case there is, first, an abrupt loss of memory at the beginning of each paroxysm for everything that has happened during paroxysms not of the same series; second, a change in the personal character of the individual, the disposition, the habits of thought, and even the intellectual powers, being altered. As illustrating this condition may be mentioned the remarkable case recorded by Dr. Azam, in which the

change between the two conditions always was preceded by a profound sleep lasting three or four minutes, during which time a complete alteration in the character of the girl occurred. In state No. 1 she was usually more or less depressed, very quiet, modest, retiring in character, and of only moderate mental power. During state No. 2 she was gay, vivacious, without any evidences of mental aberration, but with her intellectual faculties much more developed than during her normal condition, whilst her moral state was such that she became in one of her abnormal periods pregnant—a condition which overwhelmed her with surprise and shame when she was finally made to understand her condition during a No. 1 state, at which time she had no recollection when or where pregnancy was produced.

I have seen double consciousness caused by a blow upon the head, and subsequently removed by the raising of the depressed fracture—a fact that indicates (what is, indeed, otherwise very apparent) that the condition allies itself very closely to the automatism of epilepsy, and also to the changes in character, mental and psychical, seen in insanity. Epileptiform automatism may, indeed, be considered to be a form of double consciousness.

I have seen melancholia come and go in transitory attacks of a few hours or days, during which the habits of thought, the intellectual powers of the patient, were absolutely abnormal, and Dr. David Skae has put on record a case of a man who lived for many years a twofold life, being sane only on alternate days. On his melancholy days he neither ate, slept, nor walked, but sat incessantly turning the leaves of the Bible and complaining piteously of his misery. At such periods he had no remembrance of the days in which he was well, and could not be made to recognize the existence of well days. On the well day he denied that he had any cause of complaint, believed that he had been well the previous day, transacted business, and was entirely free from delusions or despondency.[1]

[1] Double consciousness must not be confounded with the rare mental condition known by the Germans as double perception or Doppeltwahrneluning, in which ideas received through one sense become to the man reduplicated over and over again through the same or another sense. Thus, in a certain case whenever the man read to himself he would plainly hear each word repeated as though a chorus of fifty or sixty female voices were speaking to him, and when he ceased to read he would hear the last words read after him. This reading after him disappeared as soon as he spoke aloud, and was prevented by his reading aloud. Evidently this condition relates itself to the hallucinations of insanity.

ORGANIC DISEASES OF THE BRAIN.

By WILLIAM OSLER.

I. AFFECTIONS OF THE MENINGES.

Diseases of the Dura Mater.

Pachymeningitis Externa.—Inflammation of the external layer of the dura, which forms the periosteum of the bones of the skull, usually follows injury, such as fracture, and is accompanied with effusion of blood. Extension of inflammation, as from erysipelas of the scalp, is extremely rare: more commonly it follows caries of the bone, as in syphilis or in middle-ear disease. In these cases pus may collect in considerable quantities between the dura and the skull, and compress the brain in a remarkable manner. The purulent infiltration may extend between the layers of the dura or it may reach the inner layer, causing a suppurative dura-arachnitis.

The **symptoms** are not at all definite, and even when the disease is extensive the only complaint may be of headache. When the exudation is large, compression symptoms are present—coma with or without paralysis. In cases of caries or in otitis media, if an external purulent pachymeningitis be suspected, the trephine should be used. In the syphilitic cases there may be a small sinus communicating with the exterior.

Pachymeningitis Interna.—The suppurative form is rare, and is, as a rule, associated with purulent leptomeningitis. Occasionally there is a pseudo-membranous formation, an instance of which I have seen in pneumonia.

Hæmorrhagic Pachymeningitis.—This condition, which is also known as *hæmatoma* of the dura mater, is not common in general medical practice, but is by no means infrequent in asylums and almshouses: thus, Wigglesworth found 42 examples in a series of 400 unselected post-mortems in asylum work. The affection is most common in males at the middle period of life, in persons addicted to alcohol, and in lunatics. It has been met with also in the specific fevers and in profound anæmia. Cases have followed injury to the head. In infants it is occasionally met with, and is said to have followed whooping cough.

The **morbid anatomy** and **pathology** have been much discussed. Virchow, to whom we are indebted for the first accurate description, regarded it as a result of inflammation of the dura, and held that a delicate vascular membrane preceded the clot. In a majority of the cases the exudation is bilateral and extends over the dura of both hemispheres, and in very extensive cases the membrane extends into the fossæ at the base of the skull. Three conditions

are seen in internal pachymeningitis: First, subdural membranes, often of extreme delicacy, with large wide-meshed vessels. There may be no hæmorrhagic exudation whatever, or the membrane may be simply stained with the blood-pigment. Second, there are instances of simple subdural hæmorrhage in which no membrane is seen. This occurred in 15 of Wigglesworth's 42 cases. It is possible that the hæmorrhage may have destroyed all traces of the membrane. Third, there may be a combination of the two vascular membranes and blood-clot. In some cases a series of laminated clots exists, forming layers of from two to three millimetres in thickness, between which cysts may form. It is stated that the hæmorrhage occurs first, and that the vascular sheet arises from the organization of the blood-clot. This does not appear probable when one considers that the inner surface of the dura may be everywhere covered with a delicate, highly vascular membrane, without a trace of staining and without any clot. The hæmorrhage is thought by Hugenin to proceed from the vessels of the pia mater, but the study of very early specimens is convincing, I think, in favor of the view that the blood comes from the wide meshwork of vessels in the new-formed subdural membranes. The condition is usually found with atrophy of the convolutions, and it is stated that this perhaps accounts for its frequent occurrence in the insane; but there must be factors other than atrophy, or we should find it more frequently in phthisis and cachectic states, in which the cortical wasting is almost as marked as in insanity.

The **symptoms** are indefinite. Extensive hæmatoma may exist without any indications during life. Headache, somnolence, contracted pupils, and optic neuritis have been described. In unilateral disease, when extensive, hemiplegia may occur. There are instances in which the symptoms set in abruptly like an apoplectic attack, producing loss of consciousness and sometimes compression.

The **diagnosis** is always doubtful.

Diseases of the Pia Mater.

Acute meningitis occurs under a variety of conditions. The exudation is beneath the arachnoid, and both membranes are involved, hence the term pia-arachnitis. A common designation now is leptomeningitis. The exudate is either purulent or consists of a sero-fibrinous fluid with flakes of lymph.

Various forms of acute meningitis are recognized, such as (1) tuberculous meningitis; (2) simple meningitis; (3) leptomeningitis infantum; and epidemic cerebro-spinal meningitis, which is elsewhere considered.

(1) Tuberculous Meningitis (Acute Hydrocephalus).

This occurs as a secondary affection of the meninges of the brain and cord, a sequel, as a rule, to a local tuberculous disease in other parts, particularly in the lungs. It is often only a part of an acute general tuberculosis. It is most frequent in children, particularly between the ages of two and five. In some instances blows on the head, falls, and overwork have preceded the onset. In

almost every instance there is coexistent tuberculous disease in other parts, caseous foci in the lungs or in the bronchial glands (particularly in children), tuberculous pleurisy, or bone disease. The symptoms may develop during convalescence from measles, whooping cough, or scarlet fever.

Morbid Anatomy.—The membranes at the base are most involved, hence the term basilar meningitis. The appearance varies greatly in different cases, depending a good deal on the amount of inflammatory exudate existing with the tubercles. As a rule, the Sylvian fissures and the interpeduncular space are most involved. There may be only slight turbidity and matting of the membranes, which are moist and infiltrated. In other instances the entire base from the olfactory bulbs to the medulla is covered with a fibrino-purulent exudate, which may extend to the lateral surfaces of the hemispheres, but is very rarely seen upon the cortex. It often extends to the spinal meninges. The tubercles may stand out with great distinctness as grayish-white nodules. In some instances they are small and difficult to find. The amount of exudate does not bear any definite proportion to the number of tubercles. They should be sought for along the branches of the middle cerebral artery, and more particularly those entering the perforated spaces. When the arteries are carefully withdrawn and spread upon a glass plate upon a black background, the tubercles are seen as small nodular enlargements of the vessels. Histologically, the new growth develops in the perivascular sheaths, the vessels are narrowed, and thrombosis not infrequently occurs. The lateral ventricles are, as a rule, dilated and contain an excess of turbid fluid. The ependyma is sometimes swollen, softened, and covered with exudate. The septum lucidum and fornix are usually softened, and tubercles are sometimes found on the choroidal plexuses and in the velum. The contiguous cerebral substance is found infiltrated and œdematous. There may be, particularly about the central ganglia, foci of softening. The condition is, properly speaking, a meningo-encephalitis. Probably in a majority of cases the meninges of the cervical cord are involved. Sometimes the brunt of the affection falls upon the spinal meninges, and the exudation, quite insignificant at the base of the brain, may be extensive throughout the cord.

There are cases in which the coarse tubercles are met with, and the meningitis has been excited by their presence. As mentioned, tubercles usually exist in other organs, either a miliary tuberculosis, in which the liver and spleen are most involved, or careful examination will show the presence of caseous foci in the lymph-glands.

Symptoms.—In very many cases the patient has been in failing health, or is the subject of a recognized tuberculous lesion, or has had a recent operation upon some tuberculous affection, or is convalescent from measles or whooping cough. In some cases the symptoms have followed shortly after a fall. The child may show marked alteration in the disposition and becomes peevish, irritable, and fretful, sleeps badly, complains of headache, and for two or three weeks may display various manifestations of ill health. Then the symptoms pointing to the disease set in, either suddenly with a convulsion or more

commonly with headache, fever, and vomiting. The pain is sometimes very intense, and may cause the child to give short, sharp cries, the so-called "hydrocephalic cry." Nocturnal delirium is present. The vomiting is without apparent cause and independent of the taking of food. The fever gradually rises, reaching 102° or 103° F. The pulse is at first rapid; subsequently it becomes slow. There may be twitching of the muscles or sudden startings, and the child may wake up from sleep in great terror. The pupils are usually contracted. These are the chief symptoms characterizing the early stage, or, as it is sometimes called, the stage of irritation. In the second period of the disease these irritative symptoms subside; the bowels become constipated; the child no longer complains of headache, but is dull and listless, with more or less delirium; the vomiting ceases; the abdomen becomes retracted and boat-shaped (carinated); the pulse becomes slow and irregular; sighing respiration is common; and the pupils vary in size, being often dilated; there may be strabismus, and in some instances optic neuritis. General convulsions may occur; more commonly there is retraction of the head and tenderness in the nape of the neck on pressure. A blotchy erythema about the chest and abdomen may occur. The temperature ranges from 100° to 102.5° F. When the finger-nail is drawn across the skin a red line quickly appears, the so-called *tache cérébrale*, which has, however, no diagnostic significance. In the final period, or stage of paralysis, the child can no longer be roused, and gradually sinks into a condition of coma. Convulsions not infrequently occur, or there are spasmodic contractions of the muscles of the back and neck, or there are irregular movements in the limbs on one side. The pupils again become dilated; the eyeballs may be rolled, so that the corneæ are only covered in part by the upper eyelid. Optic neuritis and paralysis of the ocular muscles may occur, and tubercles may in some instances be seen in the choroid. The pulse becomes rapid, diarrhœa may develop, and the child sinks into a typhoid state, with low delirium, dry tongue, and involuntary discharges of urine and fæces. The duration varies from ten days to three or four weeks. There are cases which run a rapid course, setting in with great violence and proving fatal within a week. This occurs more commonly in adults, and the convexity of the brain is often more involved. There are other instances much more chronic, in which the meningitis is limited, and the symptoms are rather those of cerebral tumor, sometimes with pronounced psychical disturbance.

Certain symptoms require a more special description. The temperature is usually elevated, but there are instances in which it does not rise above 100° throughout the entire disease. In other instances the daily oscillations are very great. Toward the close the temperature usually falls, and may sink as low as 93° or 94°. An ante-mortem elevation may occur, the fever rising as high as 110°. The pulse is often rapid at the onset, then becomes irregular and slow, and toward the close again becomes rapid. The respirations are often irregular and sighing, and in the second and third week the Cheyne-Stokes type may be very marked. The ocular symptoms are important. Nar-

rowing of the pupils is the rule in the early stage. Toward the close they are dilated and irregular. Conjugate deviation of the eyes sometimes occurs. Paralysis of the third nerve is common. Optic neuritis is rarely intense, and is not a very common symptom. Tubercles in the choroid are rare, and are less frequently seen during life than in the post-mortem room. Litten found them in 39 of 52 necropsies in tuberculous meningitis. Of 26 cases examined clinically by Garlick they were present in only 1, and Heinzel examined 41 cases with negative results. Of motor symptoms the convulsions have already been mentioned. Tremor and athetoid movements are occasionally seen; more rarely there is a tonic contraction of one limb. Hemiplegia may follow involvement of the cortical branches of the middle cerebral artery or is due to softening of the internal capsule. Monoplegias are not uncommon, particularly of the face, which may occur with aphasia. Brachial monoplegia may exist with it. In the more chronic cases, in which the symptoms persist for months, there may be characteristic Jacksonian epilepsy.

The **prognosis** is, as a rule, very grave. It is doubtful whether recovery ever occurs.

(2) Simple Meningitis.

In contrast to the tuberculous form the exudation is more apt to be upon the cortex, and is less lymphoid and more purulent in character. A primary meningitis of this description occurs as a manifestation of the poison of cerebrospinal meningitis, sporadic cases of which occur from time to time in certain localities in this country, and present great difficulties in diagnosis. The disease is almost always secondary and is met with—

(1) In the acute infectious diseases, such as small-pox, typhoid fever, rheumatic fever, scarlet fever, measles, and pneumonia. In erysipelas, inflammation of the meninges may arise either by direct extension, which is rare, or by infection through the blood. Pneumonia is the only acute disease which is frequently followed by meningitis. In 100 autopsies in this disease at the Montreal General Hospital meningitis was present in 8 cases, and I saw several charcteristic examples at the Philadelphia Hospital. Acute meningitis is not uncommon in septic processes. In ulcerative endocarditis its frequency may be gathered from my statistics—29 examples in 209 cases. It is very rare in typhoid fever. No case occurred in my 64 autopsies, and it was present in only 11 of the 2000 Munich sections.

(2) Injury and disease of the cranial bones are very common causes, particularly caries of the petrous portion of the temporal bone. Here the disease passes through the thin wall of the tympanum or extends from the mastoid cells, and is, in a majority of instances, associated with thrombosis of the dural sinuses, a condition which will be considered later. Extension from disease of the nose is very rare. The majority of instances of injury exciting meningitis cause fracture, though the possibility of its following trauma alone without an open wound must be acknowledged.

(3) Certain constitutional conditions, such as gout and Bright's disease, are

occasionally complicated with meningitis. In gout it is extremely rare. In Bright's disease cases occasionally occur, and are usually mistaken for uraemic poisoning. They are sometimes associated with inflammation of the pericardium and of the pleura. The exudation may be chiefly basilar.

(4) Among doubtful causes which are mentioned are sunstroke and excessive study. Syphilis rarely induces acute meningitis. Occasionally the disease extends from abscess of the brain.

Morbid Anatomy.—The lesions are practically identical with those described in cerebro-spinal fever. The exudate is usually purulent and as a rule cortical, particularly in the cases following the specific fevers. In the meningitis of Bright's disease and of cachectic states the basilar meninges may be chiefly involved. In the form secondary to pneumonia the exudate may be extremely abundant, completely covering the convolutions. In the simple forms of meningitis the ventricles rarely present the distension and softening of the walls so frequent in the tuberculous variety. In many instances the condition is a meningo-encephalitis, and the cortical portions of the brain are infiltrated, œdematous, and sometimes present small abscesses. The spinal meninges are often affected.

Symptoms.—Many of the cases present a clinical history similar to that already described in the tuberculous form. The secondary affection occurring in the specific fevers is very difficult to recognize, as almost identical symptoms may be caused by the poisons of the fevers without the existence of positive inflammation. For example, in cases of so-called cerebral pneumonia in which, from the outset, brain symptoms are marked (the preliminary excitement, headache, delirium, and then gradual depression, sinking into stupor and coma), unless the basilar meninges are involved, causing local palsies of the nerves—which is not usual—there is no single feature which may not be present as a result of extreme congestion. So also in typhoid fever, the cerebro-spinal manifestations may lead to a positive diagnosis of meningeal inflammation, and the twitchings, spasms, retraction of the neck, and the gradually deepening coma very frequently lead to error in diagnosis. It was from a consideration of these cases that Stokes remarked, "There is no single nervous symptom which may not and does not occur independently of any appreciable lesion of the brain, nerves, or spinal cord."

The onset is more apt to be sudden than in the tuberculous form. Occasionally the disease sets in with a chill. Headache of a severe, continuous character is the most common symptom. In the fevers, however, the patient may make no complaint. Delirium is early, and often bears some ratio to the height of the fever. Sometimes the patient is maniacal. Convulsions are much less common in simple than in tuberculous meningitis. Rigidity, spasm, and twitching of the muscles are frequent symptoms. Stiffness and contraction of the muscles of the neck are common when the inflammation extends to the meninges of the cervical cord. Vomiting occurs in the early stages. Constipation is usually present. Important symptoms are due to involvement of the cranial nerves; thus, optic neuritis may develop, but it is not common in

the meningitis of the cortex. Much more frequently the third nerves are involved, causing strabismus and ptosis. The facial nerve may be attacked, causing paresis of the face on one side, and a lesion of the fifth may be followed by disturbances of sensation; and in one of my cases, in which the Gasserian ganglion was infiltrated with pus, the cornea ulcerated. The pupils vary: they may first be contracted or unequal; later they become dilated and react very slowly to light. The pulse is rapid, sometimes irregular, and in cases in which there is much exudation and compression of the brain it may be slow. The temperature range varies, and in the forms following pneumonia may be very high. In other instances, as in the form secondary to otitis media, the variations are greater. In non-tuberculous meningitis in children and in the disease occurring in cachectic individuals the fever may be very slight.

From what has been already said it is evident that the diagnosis of purulent meningitis is extremely uncertain. It may be stated, indeed, that unless the nerves at the base are involved, causing paresis of the ocular or other muscles, and optic neuritis, there are no positive criteria by which the disease can be distinguished from the so-called cerebral form of the specific fevers. It has been a common experience of every pathologist to have cases sent down from the wards with the explicit diagnosis of meningitis, cerebral or cerebrospinal, when the section showed typhoid lesions or a local patch of pneumonia. In typhoid fever we may be in doubt for days until the abdominal symptoms become plainly manifest. The cases secondary to bone disease, to otitis media, and those occurring in pyæmic processes are less likely to escape recognition.

(3) Leptomeningitis Infantum.

While a majority of the cases of meningitis in children are tuberculous, there is a form affecting infants under two years of age which has very striking anatomical and clinical peculiarities. The disease may appear shortly after birth, and is particularly prone to affect debilitated, cachectic children. Occasionally it follows traumatism, and sometimes is associated with the specific fevers. Anatomically, the inflammation is confined chiefly to the base and to the posterior part, particularly about the cerebellum; hence it has been termed *posterior meningitis*, or, from the fact that the foramen of Magendie is closed, leading to an acute, often purulent hydrocephalus, the condition has been termed *occlusive meningitis*. The exudation may be very abundant at the base, infiltrating the membranes and covering the nerves with a thick, purulent exudate. In many instances the most striking features are in the ventricles. The posterior and descending cornua of the lateral ventricles may be enormously distended with a greenish, purulent fluid, and the ependyma thickened and infiltrated. The choroid plexuses and the velum may be covered with a thick grayish-white exudate, and the ependyma of the third ventricle may be similarly involved. In some cases the aqueduct of Sylvius and the fourth ventricle are greatly enlarged, and the ependyma thickened and infiltrated with a grayish pus. In one instance which I saw the basilar meninges were but

slightly involved, while there was a condition of purulent ependymitis in the posterior part of the lateral ventricles and in the fourth ventricle.

Fever, vomiting, convulsions, and rigidity are present in this as in other forms. The most striking feature is the holding back of the head—cervical opisthotonos—which may be the only important manifestation. Under this title the affection has been described by Gee and Barlow.[1] The child may remain for weeks in a condition of extreme weakness, with slight irregular fever, without convulsions or rigidity, but with this strong tonic contraction of the cervical muscles. In cases which have lasted for some time the head has enlarged, and a few have recovered or have terminated in chronic hydrocephalus.

Treatment of Meningitis.—Absolute quiet should be enjoined. An ice-bag may be applied to the head, and if the subject be young and full-blooded, and particularly if under these circumstances there be maniacal delirium, local or general bloodletting may be practised. Saline purges may be employed to relieve the blood-pressure. Bromides, chloral, sulphonal, or morphine may be required to procure sleep and rest. There are no remedies which influence in any way the course of an acute purulent or tuberculous meningitis. Mercurials are recommended for the purpose, and iodoform inunctions have been used on the scalp in tuberculous cases, but they are of very doubtful efficacy. If counter-irritation be thought necessary, the thermo-cautery lightly applied is the most satisfactory means to employ. In traumatic cases and in disease of the ear the surgeon should be early in attendance, and if symptoms occur which justify interference trephining should be performed.

Chronic Leptomeningitis.

This usually results from the growth of tubercles or gummata in limited regions of the meninges. It sometimes follows trauma.

The **symptoms** are very variable, depending upon the situation of the disease, and in some cases are identical with those of tumor. When in the motor region there may be Jacksonian epilepsy. The leptomeningitis infantum may really be a chronic meningitis. Some of the cases reported by Gee and Barlow lasted for more than a year.

II. AFFECTIONS OF THE BLOOD-VESSELS.

Hyperæmia (Cerebral Congestion).

About no question in cerebral pathology is there more obscurity than in relation to hyperæmia and anæmia, particularly their symptomatology. Any one who reads the report of the discussion which took place recently at the

[1] *St. Bartholomew's Hospital Reports*, 1878.

New York Neurological Society upon the subject of congestion of the brain will be convinced that the extraordinary lack of unanimity can only be correlated with a corresponding absence of all positive and satisfactory knowledge of these conditions. Unquestionably, variations occur in the amount of blood in the cerebral vessels, but how far such changes are associated with a definite group of symptoms is not at all certain. The hyperæmia is usually described as either active or passive.

Active hyperæmia is stated to follow chilling of the surface, sudden suppression of some customary discharge, excessive brain-work, and sunstroke. Alcohol and amyl nitrite also cause acute hyperæmia of the cerebral vessels.

Passive hyperæmia follows obstruction in the cerebral sinuses and veins, engorgement in the lesser circulation, as in mitral stenosis and emphysema, pressure on the superior vena cava by tumors, and from prolonged straining efforts.

The anatomical changes in congestion of the brain are not at all striking. The organ looks full and the dura is tightly stretched. The sinuses and the cortical veins are full, and often the gray matter has a rosy tint, and on section it is seen that the smaller vessels are distended. Active hyperæmia does not persist after death, as is well seen in the disappearance of the areola of congestion about a pustule on the skin. The most intense engorgement of the vessels is met with in death during the early stages of the specific fevers and in the cases due to venous obstruction.

There are no characteristic or constant symptoms of cerebral hyperæmia. In the passive form it may exist in the most extreme grade and without the slightest disturbance of function. In other instances, as in pressure on the superior cava, there may be torpor, but rarely coma. The headache and delirium of the early stage and of fevers are often attributed to congestion of the brain, but it is more likely they are due to the agents which excite the pyrexia. The dizziness, throbbing, and unpleasant sensations described in aortic insufficiency and in hypertrophy of the heart may be due to the sudden overfilling of the cerebral vessels during systole.

As a definite clinical affection congestion of the brain is very rare. Personally I have no knowledge of the cases described by some authors setting in with fever, delirium, and insomnia; still less of the apoplectiform, convulsive, and comatose forms. Perhaps the most definite cases are those met with in persons of a full habit, who are subject at times to headache, flushing of the face, throbbing of the carotids—symptoms which may be relieved promptly by an attack of epistaxis or which yield to a brisk mercurial purge.

Cerebral Anæmia.

The anæmia may be confined to local areas in the brain, as in narrowing of vessels by endarteritis or occlusion by emboli. It may be limited to the brain itself, as in cases of ligature of both carotids, or in diminished blood-supply, as in extreme aortic stenosis, or it may follow the sudden dilatation of a vascular territory, as in rapid distension of the intestinal vessels. The cere-

bral anæmia may be part of a general bloodlessness due to hæmorrhage or is part of an anæmia, primary or secondary.

The brain in anæmia is pale; only the large veins are full; the small vessels over the dura are empty and the membranes are moist; and there is an unusual amount of cerebro-spinal fluid. On section the gray and white matter looks very pale, and the cut surfaces moist and show very few puncta vasculosa.

The consequence of cerebral anæmia when suddenly produced is well seen in a fainting fit, in which loss of consciousness follows the sudden sinking of the arterial pressure in the cerebral vessels. When it results from hæmorrhage the patient complains of drowsiness, giddiness, a feeling of faintness, flashes of light, and noises in the ear; the respiration becomes hurried; the skin is cool and covered with sweat; and gradually, if the hæmorrhage continue, consciousness is lost and death occurs with convulsions. In the more chronic forms of brain anæmia the patient may be subject to fainting spells, and in some instances headache and rambling delirium. In the anæmia of wasting disease or of starvation there is gradually induced a condition of irritable weakness, in which all mental effort is difficult and the slightest irritation is followed by undue excitement. The patient complains of giddiness, noises in the ear, and there is finally developed the delirium of inanition, characterized by marked hallucinations.

An interesting group of symptoms is met with in the prolonged malnutrition of young children, associated usually with diarrhœa. The pupils may be narrow or unequal; the head is thrown back and the child is in a semi-comatose state, but with the eyes open; convulsions may occur and the fontanelles are usually depressed. The body is usually cool, the pulse feeble and rapid, and the respirations normal. It was to this condition that Marshall Hall applied the term "spurious hydrocephalus," and it is also spoken of as the hydrocephaloid (hydrencephaloid) condition. The cases are not infrequently mistaken for tuberculous meningitis.

The treatment of cerebral anæmia is that of the conditions with which it is associated. The suddenly-developed form leads to syncope or fainting, for which, as a rule, the recumbent posture, the dashing of cold water upon the face, superficial friction, and the inhalations of ammonia suffice to restore consciousness. If the syncope persist, a tight bandage can be applied round the legs or the abdominal aorta compressed in order to take advantage of the collateral fluxion.

Œdema of the Brain.

This is often only a complication of cerebral anæmia. An increase in the subarachnoid fluid is common in all atrophic states of the brain. In extreme passive hyperæmia there may be a congestive œdema, in which the brain-substance not only contains an increased amount of blood, but is unusually moist. The most extreme œdema is met with as a local process about tumors and abscesses. A very intense infiltration, localized or general, is met with

sometimes in chronic Bright's disease, and to it Traube referred certain uræmic manifestations.

When a sequence of atrophy, the fluid is chiefly within and beneath the membranes, and the amount of fluid in the ventricles is usually increased. In anæmic states and in death from cachexia the brain-substance is pale, moist, and glistening.

The **symptoms** are not well defined, and are chiefly those of the associated anæmia. As mentioned, Traube thought that uræmia was due to cerebral œdema consequent upon the hyperæmia and high arterial tension—a view which has not received general acceptance. On the other hand, of late years cases have been reported of localized convulsions and of paralysis in Bright's disease in which, after death, no lesions other than œdema have been found.

Cerebral Hæmorrhage.

Cerebral hæmorrhage, the common cause of apoplexy, is almost invariably the result of rupture of an artery. It may be from the central vessels which pass at once into the substance of the brain, from the large branches of the circle of Willis, or from the cortical group which is distributed upon the surface of the convolutions. In a majority of the cases the hæmorrhage is from the central branches, particularly from those which pass in at the anterior perforated spaces. The largest of these vessels passing to the third division of the lenticular nucleus and the hinder part of the internal capsule is so frequently involved that it has been called by Charcot *the artery of cerebral hæmorrhage.* The extravasation may be into the substance of the brain, into the membranes, or into the cerebral ventricles.

Etiology.—The important factors are those leading to degeneration of the blood-vessels. The natural tendency to arterial degenerations as years advance makes hæmorrhage much more common after the fiftieth year. It is, however, not unknown in early life, and in children may be due to rupture of an aneurism or to local degeneration. It occasionally is caused by the paroxysms of whooping cough. Cerebral apoplexy is not unknown in the fœtus. As will be mentioned, the meningeal hæmorrhage is a very frequent and important event in protracted labor, but hæmorrhage into the substance of the brain may itself cause death in the fœtus. Men are more frequently attacked than women—an association doubtless due to the greater liability in the former to arterial disease. Heredity is believed to play an important part, and the apoplectic build or habitus is still spoken of, by which is meant a stout, plethoric frame with a short neck and a congested condition of the superficial vessels. The influence appears to be exerted through the arteries, as there are families in which they degenerate early, usually in association with renal changes. The three special factors in inducing arterio-sclerosis—namely, the abuse of alcohol, syphilis, and prolonged muscular exertion—are important antecedents in a large number of cases of cerebral hæmorrhage. In adults hypertrophy of the left ventricle and sclerosis of the kidneys are almost constant concomitants of hæmorrhage into the brain. The endocarditis following

rheumatism and other fevers may indirectly lead to apoplexy. The cases are not very infrequent in young persons. Emboli are carried off from the valves and lead to softening or weakening and subsequent aneurismal dilatation of a cerebral vessel, and hæmorrhage may follow rupture of the aneurism.

Hæmorrhage occurs sometimes during the course of the specific fevers; more common still are the cases due to profound alteration in the blood, as in anæmia and leukæmia. Occasionally, too, cerebral hæmorrhage occurs in purpura hæmorrhagica and in scurvy.

The exciting causes are not often evident. The attack may be sudden, without any preliminary symptoms. In many cases the rupture occurs during violent muscular efforts, such as straining at stool, vomiting, or coughing, or in very excited action of the heart during emotion.

Morbid Anatomy.—Lesions are found in the cerebral arteries and comprise the following changes:

(1) A diffuse periarteritis of slow development, which causes weakening of the coats and the formation of small miliary aneurisms. These are present in the great majority of all cases of hæmorrhage in adults, and are almost invariably found if carefully sought for. They occur most frequently on the central arteries, but also on the smaller branches of the cortical vessels. They are often to be seen on section of the brain-substance as small dark bodies from 1 to 3 millimetres in diameter. They may be present in numbers upon the arteries withdrawn from the anterior perforated space. Charcot and Bouchard, who first accurately described them, state that they are most frequent in the central ganglia.

(2) Larger aneurisms on the branches of the circle of Willis, which are by no means uncommon, and will be considered in a separate section.

(3) Endarteritis and periarteritis usually lead to hæmorrhage by the formation of aneurisms, either miliary or coarse. There are cases, however, in which careful examination fails to show anything but a diffuse degeneration of the smaller vessels, and doubtless hæmorrhage may occur without the previous formation of aneurism.

Finally, there are instances of cerebral hæmorrhage in which macroscopically and microscopically the changes in the arteries seem insignificant.

The bleeding may be into the meninges, into the cerebral substance, or into the ventricles. Meningeal hæmorrhage may be outside the dura, or more frequently subdural, and often between the arachnoid and the pia mater. In fracture of the skull causing laceration of the meningeal vessels the blood is usually outside the dura or between it and the arachnoid. In rupture of aneurisms of the larger cerebral vessels the hæmorrhage is usually meningeal and very extensive, and may extend high up on the cortex and on to the cord. Owing to the more frequent presence of aneurism in the middle cerebral vessels, the Sylvian fissures are often found distended with blood. Intracerebral hæmorrhage may burst into the meninges. The meningeal hæmorrhage of infants resulting from injury during labor will be subsequently discussed under the section upon the Cerebral Palsies of Children. More or less ex-

tensive effusion may be found in the meninges in fevers, and occasionally in constitutional diseases.

Intracerebral Hæmorrhage.—The most common form is extravasation in the region of the strio-lenticular artery, about the outer section of the lenticular nucleus. If small in extent, it may be limited to the lenticular body and the internal capsule. In other instances it extends outward to the insula or upward into the centrum ovale or inward to the lateral ventricle. Hæmorrhage into the centrum ovale is not nearly so common, and still less frequent are localized extravasations into the pons, medulla, or cerebellum. The hæmorrhage breaks the tissues, and the clots occupy an irregular cavity and are mixed with brain-substance. The walls are at first irregular and composed of blood-stained and softened cerebral matter.

Ventricular Hæmorrhage.—Primary bleeding into the ventricles is rare. The blood in almost all instances comes from rupture of an extravasation into the ventricle. It is not very infrequent in early life, and may occur during birth. Of 94 cases collected by Edward Sanders, 7 occurred during the first year and 14 under the twentieth year. It occasionally occurs during parturition and in the puerperal state. There is in the McGill University Museum a remarkable instance of this in which both lateral ventricles, the third, the aqueduct of Sylvius, and the fourth ventricle are enormously distended with clots which formed a complete mould in blood of the ventricular system. The blood may be found in one ventricle only; more commonly it reaches the other ventricle, either bursting through the septum or finding its way through the foramen of Monro.

In all instances where the extravasation is at all large the hemisphere on the side involved looks fuller and larger, and the convolutions are flattened, and the dura on the affected side is unusually tense. In time the blood-clot undergoes changes. The hæmoglobin is converted into reddish-brown hæmatoidin and pigment-granules. The rapidity with which these changes proceed varies: as a rule, in cases which prove fatal within a month brownish-yellow remnants of the clot are found with disintegrated brain-tissue, molecular *débris*, and compound granular corpuscles. A limited irritative inflammation occurs about the clot, and, if large, a definite wall is formed, enclosing a cyst with fluid contents. In smaller clots a pigmented scar is left. In meningeal hæmorrhage the effused blood may be gradually absorbed, leaving only a brownish stain. In this form of hæmorrhage in infants, when the extravasation is abundant, wasting of certain of the convolutions may take place, and sometimes cysts form in the meninges. It is possible that certain of the cases of porencephaly are produced in this way.

Secondary Degeneration.—After a lesion of the motor centres or of the pyramidal tract secondary degeneration occurs in the motor path. Thus in a case of hemiplegia, caused, as is often the case, by a hæmorrhage in the neighborhood of the internal capsule, a descending degeneration is seen in the crus, in the anterior part of the pons, in the pyramidal fibres of the medulla on the same side, in the direct fibres of the cord on the same side (column of Turck),

and in the crossed pyramidal fibres of the opposite side of the cord. In permanent cortical lesions the secondary degeneration may be traced through the fibres of the corona radiata and into the internal capsule, and through the course of the pyramidal fibres just mentioned.

Symptoms.—These may be divided into the primary, or those connected with the onset of the attack, and the secondary, or late, symptoms, which develop after the early manifestations have passed away.

Premonitory indications are not common. There may be for some days or even for weeks headache, feelings of numbness and tingling, or even pains in the limbs. Still more rarely there are irregular choreiform movements of the muscles on one side, the so-called prehemiplegic chorea. As a rule, the patient is seized while in ordinary health about the performance of some every-day action, occasionally such as requires exertion or strain. When the hæmorrhage causes sudden and complete loss of consciousness, with relaxation of the limbs, it is known as *apoplexy* or an apoplectic stroke. In other cases the onset is more gradual, and the loss of consciousness does not occur for a few minutes after the patient has fallen or after the paralysis of the limbs is manifest. In an apoplectic attack the patient is seized with giddiness or feelings of faintness, sometimes is sick at the stomach, or has a slight convulsion. In rare instances of large extravasations the patient dies in a few minutes, but instant death is rare in cerebral hæmorrhage. There is deep unconsciousness from which the patient cannot be aroused. The face is injected, sometimes cyanotic or of an ashen-gray hue. The skin is usually moist with perspiration. The pupils vary in size, but as a rule are dilated and inactive. They may, however, be strongly contracted. The respirations are slow, noisy, and accompanied with stertor, which, as Bowles, has shown, is only marked when the patient is on the back, and is owing to the falling of the tongue to the hinder part of the mouth.

The Cheyne-Stokes rhythm may be present. The pulse is usually full, slow, increased in tension, and sometimes irregular and small. The temperature may be normal, but very often falls within an hour after the onset, and may even sink below 95° F. An exception to this is found in hæmorrhage into the pons or medulla, in which within an hour of the onset the temperature may reach 104° or 105° F. The urine and fæces are usually passed involuntarily. Albumin and sugar may be found in the urine very shortly after an attack. Convulsions are not common in an apoplectic seizure due to hæmorrhage.

It may at first be difficult to decide whether the condition is apoplexy associated with hemiplegia, or whether the coma is due to other causes, such as uræmia or opium-poisoning. An indication of the hemiplegia may often be discovered, even in deep coma, by a difference in the tonus of the muscles of the two sides. If the arm or the leg be lifted, it drops "dead" on the affected side, while on the other it falls more slowly. One side may present marked rigidity, and in watching the movements of the facial muscles in the stertorous respiration, if paralysis be present, it will be noticed that the cheek on the

affected side is puffed and blown out in a more marked manner. The head and eyes may turn strongly to one side—conjugate deviation.

The patient may at first not lose consciousness, but be slightly dazed, and in the course of a few hours there is loss of power on one side, and gradual unconsciousness, deepening into a profound coma. This is sometimes termed *ingravescent* apoplexy. The attack may occur during sleep and the patient be found unconscious, or he may wake and find the power lost on one side. Cerebral hæmorrhage is not necessarily accompanied by the symptoms of apoplexy; that is, with loss of consciousness. A small hæmorrhage, particularly in the region of the central arteries, may involve the motor path, causing hemiplegia without any loss of consciousness.

The subsequent course varies greatly. In the severer cases the respirations become more rapid, the pulse feeble, the skin is bathed with sweat, the color of the face becomes ashen-gray or livid, noisy râles are heard in the trachea and larger bronchi, and death may occur within twenty-four hours of the onset. In other instances the patient remains unconscious, and within forty-eight hours there is some febrile reaction and constitutional disturbance, which is associated with inflammatory changes about the hæmorrhage. The patient may die in this reaction, and if consciousness has been regained there may be delirium or recurrence of the coma. At this period also the so-called early rigidity may develop in the paralyzed limbs, and, more important still, trophic disturbances, such as sloughing or the formation of vesicles. The most serious trophic change is the sloughing eschar which develops about the middle of the lumbar region on the paralyzed side, and is different from the eschar of acute myelitis, which develops in the centre of the sacral region. It may appear within forty-eight hours of the onset, and is of very grave significance. Some have regarded the congestion of the lungs so common in apoplexy, and which is sometimes unilateral, as an evidence of a trophic change.

Certain symptoms of cerebral hæmorrhage require more description:

Hemiplegia.—If the hemiplegia involve the motor centres or path, loss of power occurs in the muscles of the opposite side of the body. It is known as complete hemiplegia when it involves face, arm, and leg; partial when it involves only one or other of these parts. It may follow a lesion in the motor cortex, the white fibres of the corona radiata, the internal capsule, the crus, or the pons. Hæmorrhage is perhaps the most common cause of hemiplegia, but it is also produced by embolic and thrombotic softening and by tumors. In a majority of severe cases of hæmorrhage the face, arm, and leg are involved on the opposite side. In other instances the leg and arm may be chiefly involved, or the face and arm.

The face is involved on the same side as the arm and leg. The facial muscles stand in precisely the same relation to the cortical centres as do those of the arm and leg. The fibres of the upper motor segment of the facial nerve coming from the cortex decussate just as do those of the nerves of the limbs. The facial paralysis, however, is partial, involving only the lower

face, sparing the orbicularis oculi and the frontalis muscles. There may be, however, slight difficulty in elevating the eyebrows and in closing the eye on the paralyzed side. The signs of the facial paralysis are usually well marked, and the mouth is drawn toward the healthy side. The tongue may be protruded toward the paralyzed side, owing to the unopposed action of the geniohyoglossus of the sound side. It is to be remembered, however, that the position of the tongue must be taken from the incisor teeth, not from the lips, which are drawn toward the healthy side, so that the tongue even when protruded straight may appear to deviate to the paralyzed side. With hemiplegia on the right side there may be also asphasia.

As a rule the arm is more completely paralyzed than the leg. The loss of power at first may be complete, and then gradually returns in the leg, still remaining in the arm. The muscles associated in symmetrical movements, such as those of the eyes and of the thorax and abdomen, usually escape. It may be noted in the deep stertorous respirations that the chest does not move so freely on the paralyzed side. Broadbent's explanation, the one most satisfactory, is thus clearly given by Frederick Taylor: "It is first to be observed that the parts that are least paralyzed or not paralyzed at all are those which rarely or never act independently of their fellows on the opposite side; whereas the parts that are most paralyzed are much more independent, and may be capable of performing acts that the corresponding muscles on the opposite side are unequal to. As extreme instances may be mentioned the eyes, of which one never moves except in association with the other, their muscles not being affected. In contrast with these are the hands, of which the right may be able to do things the other cannot, and *vice versâ;* and these parts are most affected. Dr. Broadbent's theory supposes that in the case of the muscles most commonly associated the commissural fibres between their nerve-nuclei become functionally active, so that in the event of a lesion preventing one, say a right-side nucleus, from receiving stimuli from the left brain, it may be stimulated from the right brain by impulses passing first to the left-side nucleus, and then by the commissure to the right-side nucleus. On the other hand, if in the case of the less associated muscles the commissure remains functionally inactive, such a transference would not take place, and the right-side nucleus would remain completely cut off from the cortical centres. Another view has been put forward to the effect that fibres for the face, arm, leg, and trunk as they pass through the internal capsule have positions corresponding to those of the motor centres for these parts on the cortex; and that since vessels mostly rupture below the internal capsule, and the pressure would most injure the fibres which were nearest, the arm would suffer more than the leg, and the leg than the trunk."

The face and limbs may be paralyzed on opposite sides, forming what is known as the crossed or alternate hemiplegia. This occurs when the hæmorrhage is in the lower segment of the pons Varolii, involving the facial nerve in its way through the pons after it has left its nucleus; whereas the motor fibres of the arm and leg which are involved in the lesion are above their

decussation in the medulla, so that the paralysis occurs in the face on the same side as the lesion, and in the arm and leg on the opposite side.

Hemianæsthesia is rare in hemiplegia. Slight numbness or tingling may be present, or there is loss of sensation after a day or two, which gradually passes off. According to Dana's study, the anæsthesia of organic cortical lesions was generally incomplete and more pronounced in certain parts than in others; total anæsthesia was either functional or due to subcortical lesions. Sensory disturbances are more common in softening than in hæmorrhage. Disturbance of the special senses is not common. There may be diminution in the acuteness of hearing, taste, and smell.

The eye-symptoms in hemiplegia are important. Hemianopia may occur with hæmorrhage in the occipital lobe or in the fibres of the optic radiation. It is, however, not common in ordinary hemiplegia. The most important ocular symptom is—

Conjugate Deviation.—The head and eyes, as a rule, are turned away from the affected side; thus in a right hemiplegia the eyes and head are turned to the left side; that is to say, the eyes look toward the cerebral lesion. When spasms or convulsions develop, or if the state of early rigidity supervenes, the head and eyes may deviate in the opposite direction; that is to say, the patient looks away from the lesion and toward the convulsed side. This symptom occurs in lesions in different localities of the brain, particularly with cortical lesions. It is also met with in lesions of the internal capsule, and in those of the pons or in the latter situations it has been found that the deviation is just the reverse of that which occurs in other cases, as in paralysis the patient looks away from the lesion and in spasm or convulsion looks toward it.

As a rule, there is no wasting of the paralyzed limbs, and the muscles react well to both the faradic and the galvanic currents. The deep reflexes are increased on the paralyzed side; the superficial reflexes, plantar and cremasteric, are often diminished or absent. The sphincters are not often involved in hemiplegia.

The course of the disease depends upon the situation and extent of the lesion. If slight, the paralysis may disappear completely in a few days or in a few weeks. In severer cases partial recovery gradually takes place, associated with which are the changes which may be grouped as

Secondary Symptoms.—These correspond to the chronic stage, which follows in some weeks or months after the initial lesion. The paralyzed limbs undergo certain changes. The leg, as a rule, recovers sufficient power to enable the patient to walk about, but with a characteristic hemiplegic gait. The loss of power is most marked in the muscles of the foot, so that to prevent the toes from dragging the knee is much flexed and the foot swung round in a half circle. In both arm and leg the condition known as secondary contraction or late rigidity supervenes; the arm is flexed at the elbow and resists all attempts at extension; the wrist is flexed upon the forearm and the fingers upon the hand. The position assumed by the arm and hand is very characteristic. Frequently as this contraction develops there is much pain. In the

leg the contractures are not so extreme. Unlike the contractures of hysteria, the secondary contracture of hemiplegia is not relaxed under chloroform, and is an incurable condition, associated with a descending degeneration of the motor tract. Occasionally after hemiplegia secondary contracture does not occur, but the arm remains more or less flaccid, the leg having regained partial power. This condition is met with most frequently in the hemiplegia of children. The reflexes are greatly increased on the paralyzed side. Atrophy of the muscles is not a marked feature in hemiplegia, but develops in certain instances, due possibly to secondary alterations in the gray matter of the anterior horns. It may, however, follow as a direct result of the cerebral lesion, and from this cause is not very infrequent in the cerebral palsies of children.

Other secondary changes in hemiplegia are post-hemiplegic disorders of movement, either tremor, choreiform movements, or the mobile spasm known as athetosis. These will be more fully considered in the hemiplegia of children, with which they are most commonly associated. Arthropathies may develop early in hemiplegia, but more commonly develop late, and are most frequent in the joints of the arm. They take the form usually of a synovitis, with swelling, redness, and pain.

Diagnosis.—This may be extremely difficult if the patient be seen for the first time in a condition of deep coma, as this may be due to alcohol or opium or to uræmic poisoning. The first thing to be determined, if possible, is the existence of hemiplegia. Even in very deep coma the limbs on the paralyzed side are, as a rule, very flaccid, and drop instantly when lifted, whereas on the other side the muscles retain some tonus. Conjugate deviation of the head and eyes, rigidity on one side, or spasm on one side are suggestive of a hemiplegic lesion. In a majority of these cases it is practically impossible to say at first whether the lesion be due to hæmorrhage, to embolism, or to thrombosis. Stiff arteries, an hypertrophied left ventricle, and a sudden onset, with complete loss of consciousness, are decidedly in favor of hæmorrhage or of embolism, while a more gradual onset in a man with degenerated vessels is more commonly due to thrombosis. The most puzzling cases are those in which large hæmorrhage occurs into the ventricles or into the pons, producing sudden loss of consciousness and complete relaxation. The previous history and the mode of onset may give valuable information. In epilepsy the convulsions have preceded the coma and there have been previous attacks. In alcoholism the odor of the breath, the history of drinking, and the more gradual onset are points to be considered. In opium-poisoning the coma develops slowly and the pupils are strongly contracted. In ventricular hæmorrhage sudden and rapidly deepening coma occurs. There may be no hemiplegia, but the muscles on both sides are equally relaxed, sometimes with a preliminary rigidity or with convulsions. These symptoms may be the very ones to lead astray, as in a case occurring in a puerperal patient, in whom albumin and tube-casts were present, the condition was naturally enough believed to be uræmic. In hæmorrhage into the pons convulsions are frequent, the pupils may be strongly contracted, and conjugate deviation may occur, the

eyes looking away from the lesion, and the pupils may be strongly contracted, so that opium-poisoning may be suggested. The temperature may rise rapidly —a point of considerable diagnostic value.

At first it may be quite impossible to give a definite diagnosis. In emergency cases special care should be taken upon the following points: The head should be examined for injury; the urine should be drawn off at once and tested for albumin and sugar; the limbs should be immediately examined with reference to their degree of relaxation, the presence or absence of rigidity, and the condition of the reflexes. The state of the pupils should be noted and the temperature taken. Serious mistakes are often made in the case of individuals who, as not infrequently happens, are drunk at the time of the apoplectic seizure. The condition may be regarded as due to alcoholism or to uræmia. In pontine hæmorrhage respiration is often disturbed, and may be slow as in opium-poisoning.

Prognosis.—From a limited cortical hæmorrhage the recovery may be complete, particularly if the hæmorrhage occurred after injury. The infantile meningeal hæmorrhage causing the birth-palsies is very frequently followed by idiocy and hemi- or diplegia. Large extravasations into the white substance of the hemispheres and into the ventricles and about the base prove rapidly fatal. The hemiplegia following a lesion of the internal capsule is usually persistent and is followed by contractures. If the posterior fibres have been involved, there may be hemianæsthesia, and later hemichorea or athetosis.

The following symptoms in a case of cerebral hæmorrhage are of grave moment: Persistence or deepening of the coma during the second or third day, rise in temperature within the first twenty-four hours after the initial fall. After the reaction on the second or third day, with which there is usually moderate fever, a gradual fall on the third or fourth day, with a return of consciousness, is a favorable indication. The early formation of a sloughing bedsore is very unfavorable. The presence in the urine in large quantities of albumin and sugar is an unfavorable symptom. With the return of consciousness and the improvement in the general condition the question is anxiously asked by the friends as to the persistence of the paralysis. In adults, if the hemiplegia has been complete, involving the face, arm, and leg, and if it persist for a week or ten days, there is little hope that it will entirely disappear. Slight paralysis of the arm and face without profound loss of consciousness may be recovered from completely, but complete hemiplegia which persists for a month usually leaves permanent disability. The leg improves as a rule, and the patient is able to walk about and power gradually returns in the face, but the hand rarely regains control of the finer movements. If complete motor aphasia has occurred, the chances are against full re-establishment of the power of speech. The late rigidity with contractures is a hopeless condition.

Embolism and Thrombosis.

(1) Embolism.—The most common cause of embolism is a fresh warty endocarditis or a recurring vegetative inflammation on sclerosed valves. Sometimes fragments are carried off from segments involved in an ulcerative process. The mitral endocarditis is by far the most common source; less frequently portions of clot in the appendix of the auricle or small white thrombi are responsible for it. Portions of thrombi from an aneurism or from atheromatous patches on the aorta, or thrombi from the territory of the pulmonary veins, may also block the branches of the circle of Willis. In the puerperal condition cerebral embolism is not infrequent, occurring sometimes in women with heart disease, but in many cases the heart is uninvolved, and the condition is thought to be associated with the development of heart-clots owing to an increased coagulability of the blood. Practically, a large proportion of all cases of embolism occur in chronic valvular disease, particularly in those cases of recurring endocarditis so commonly present on the sclerotic segments. It is much less common in the acute endocarditis of rheumatism, chorea, and the acute fevers. The emboli pass most frequently to the left middle cerebral artery, owing to the fact that the left carotid is more in the direct course of the blood-current than the innominate. The posterior cerebral and the vertebral arteries are less often involved. A large embolus may lodge at the bifurcation of the basilar artery. Embolism of the cerebellar vessels is rare.

The statement is usually made that embolism of the cerebral arteries is more frequent in women, owing to the more common occurrence of mitral stenosis in them, but statistics seem to indicate that cases are quite as frequent in men, if not more common; thus Newton Pitt's recent statistics of 79 cases at Guy's Hospital give 44 cases in males and 35 in females.

(2) Thrombosis.—Clotting of blood during life in the cerebral vessels may be due to—

(*a*) The presence of an embolus. About a fragment of clot or a vegetation which blocks a cerebral artery the blood coagulates usually as far back as the first large branch. The embolus may be completely surrounded by recent coagulum.

(*b*) Local disease of the cerebral arteries, either a simple or a syphilitic endarteritis. In elderly persons with advanced atheromatous changes in the larger branches of the circle of Willis it is not very uncommon to find adherent thrombi. The most advanced atheroma may exist without a trace of separation of the fibrin, and in all probability other factors, such as debility and changes in the constitution of the blood, are necessary. The syphilitic endarteritis is a much more common cause of thrombosis. The growth of tubercles in the vessels more rarely causes thrombosis. The blood may clot in aneurisms, both miliary and coarse. Ligature of the carotid has in a few instances caused the formation of thrombi in the arteries, and, as a rule, of course under these circumstances the collateral circulation is readily established. In certain blood-conditions there is a tendency to clotting in the cerebral vessels,

as in marasmus from any cause, phthisis, chlorosis, and in the puerperal state.

Anatomical Changes.—The immediate effect of blocking of a cerebral artery is degeneration and softening of the vascular territory supplied by it. The affected district is rarely in a condition of deep hæmorrhagic infarction, as in embolism of the arteries of the spleen or kidneys. More commonly the change may not be very striking, and the affected area may look only a little paler and slightly softer than normal. Gradually the consistence of the parts lessens, owing to the infiltration of serum, and the nerve-fibres become degenerated and fatty, and the neuroglia swollen and œdematous. The hæmoglobin undergoes a gradual transformation, and the color, which is red at first, changes to yellow. Microscopically, disintegrated nerve-fibres, fatty and molecular débris, pigment-grains, and compound granular corpuscles are present. Much stress was formerly laid upon the red, yellow, and white softening, but they are varieties of one and the same process. The *red softening* is met with chiefly in the gray matter of the cortex and of the ganglia. It may show punctiform hæmorrhages (capillary apoplexy), and the appearance may be almost hæmorrhagic. The *white softening* is most common in the centrum ovale, and its most typical forms occur in the neighborhood of tumors and abscesses and in septic processes. *Yellow softening* is usually an advanced stage of the red. There is a variety of yellow softening, the *plaque jaune*, which is common in elderly persons. The spots are from 1 to 2 cm. in diameter, with cleanly-cut edges, and the softened area represented either by a turbid yellow material, or in the advanced stages a small excavation filled with fluid and crossed by fine trabeculæ. A dozen or more of these patches may be met with on different convolutions. They probably result from fatty or hyaline change in the smaller cortical arteries.

Inflammatory changes occur about the softened areas, and when the embolus is derived from an infected focus, as in ulcerative endocarditis, there may be suppuration. The final changes vary greatly. It is surprising for how long a period red and yellow softening may remain unchanged. Months after the attack the involved area may be only slightly depressed, flattened, somewhat softer than normal, and of a yellowish color. Finally the degenerated and dead tissue-elements are removed, and in a small area replaced by new growth of connective tissue. In larger regions the peripheral portion of the softened area becomes condensed, while the degenerated elements are absorbed and a cyst is gradually formed, sometimes crossed in different directions by connective-tissue trabeculæ.

Softening occurs in all parts of the brain, more particularly on the cortex and in the central ganglia, in which the vessels are terminal arteries. The extent of the softening depends upon the position of the embolus and the possibility of establishing a collateral circulation; thus an embolus blocking the middle cerebral at its origin involves both the central arteries passing into the anterior perforated space and the cortical branches. Softening in the corpus striatum and the internal capsule in such a case is inevitable, and as a rule in

part, at any rate, of the territory supplied by the middle cerebral. The extent of this varies very much, as the freedom of anastomosis between the cortical branches appears to differ. There are instances of embolism of the middle cerebral artery in which the softening has only involved the territory of the central branches, in which case the blood must have reached the cortical area through the anterior and posterior cerebrals. When, as is perhaps most often the case, the middle cerebral is blocked beyond the region of the central arteries, one or other of its branches is most involved. The embolus may lodge in a vessel passing to the third frontal convolution, or in the artery of the ascending frontal or ascending parietal gyrus, or it may block the branch passing to the supramarginal and angular convolutions, or enter the lowest division distributed to the upper convolutions of the temporo-sphenoidal lobe. These are practically terminal arteries, and when involved in embolism or thrombosis softening follows in part, at least, of the territory supplied by them, producing in this way some of the most accurately focalizing lesions which we meet.

Symptoms.—The most extensive softening may occur without causing any symptoms, as when the occlusion involves arteries passing to the silent regions, as they are termed. In elderly persons it is quite common to meet with multiple areas of the *plaques jaunes* which have not apparently caused any symptoms. In many instances the symptoms are identical with those produced in hæmorrhage, and transient or permanent hemiplegia is produced with or without loss of consciousness. There are certain peculiarities associated with the attacks of embolism and thrombosis respectively.

In *embolism* premonitory symptoms are rare; the onset, as a rule, is sudden, without any headache, numbness, or tingling. The patient is the subject of heart disease, or there exists some of the conditions already mentioned as favoring embolism. When the embolus blocks the left middle cerebral artery, aphasia is usually associated with the hemiplegia.

In *thrombosis* premonitory symptoms are usually present and the onset is more gradual. The patient has complained of headache, vertigo, numbness or tingling in the fingers, transient weakness on one side or in the arm or leg. The speech may have been embarrassed for some days, or the patient has loss of memory and is incoherent. The paralysis may begin in one arm and extend slowly. Abrupt loss of consciousness is much less common than in embolism, and still less so than in cerebral hæmorrhage; thus, with thrombosis due to syphilitic disease the hemiplegia may come on without the slightest disturbance of consciousness. There are instances, however, of extensive involvement due to syphilis in which the patient becomes somnolent and is unconscious for days or even weeks. Convulsions may occur with embolism, rarely with thrombosis.

The general symptoms in thrombosis and embolism are, as a rule, not nearly so striking as in cerebral hæmorrhage, and the profound apoplectic condition with stertorous breathing is not so often seen. The focal symptoms are practically the same, and the hemiplegia has the primary and secondary

characteristics described under Hæmorrhage. The following are the effects of blocking of particular vessels:

(*a*) Vertebral. The left branch is usually plugged, and results in an acute bulbar paralysis from involvement of the nuclei in the medulla. It may be unilateral and associated with hemiplegia. More commonly there is with it

(*b*) Occlusion of the basilar artery, which may cause sudden death from involvement of the respiratory centres. In complete occlusion of this vessel there may be bilateral paralysis from involvement of both motor paths, and bulbar symptoms. The temperature rises rapidly, and there may be hyperpyrexia. Death occurs as a rule within a few days.

(*c*) The posterior cerebral artery supplies the occipital lobe on its inner face and the greater part of the temporo-sphenoidal. Localized areas of softening may exist without symptoms. Occlusion of the branch passing to the cuneus may be followed by hemianopia, and hemianæsthesia may be caused by involvement of the posterior part of the internal capsule.

(*d*) Internal carotid. The symptoms are very variable. In a majority of the cases the vessel may be ligated without any risk. Sometimes transient hemiplegia follows. In rare cases the condition is persistent and death has occurred. These variations depend upon the anastomoses in the circle of Willis, which if large and free readily permit of the collateral circulation, but when the anterior and posterior communicating vessels are very small or are absent, the paralysis may persist. When the internal carotid is blocked within the skull, and particularly by the formation of a thrombus, the results are much more serious, as the process is apt to spread into the branches. Hemiplegia, coma, and early death usually follow.

(*e*) Middle cerebral. This is the artery most commonly involved, and, as already mentioned, when plugged before the central arteries are given off, permanent hemiplegia may follow from softening of the internal capsule. Blocking of the branches beyond this point may be followed by hemiplegia, which is more likely to be transient, involving chiefly the arm and face, and, if on the left side, associated with aphasia. As already mentioned, the individual branches passing to the third frontal, the ascending parietal, the supramarginal and angular gyri, and to the temporal gyri may be plugged.

(*f*) The anterior cerebral. Cortical softening in the district supplied by this vessel is rare, as the branches from the middle cerebral are usually able to effect a collateral circulation. Softening of the orbital lobule and the olfactory bulb may occur. Hebetude and dulness of intellect may exist with obstruction of this vessel.

Treatment of Cerebral Hæmorrhage, Embolism, and Thrombosis.— In cerebral hæmorrhage the patient should be placed with the head high, and, if stertor be present, turned on the paralyzed side. Bowles, who has written a most suggestive work on the subject, calls attention to the great importance of position in an apoplectic seizure, holding that the stertor arises largely from the tongue falling back in the supine position of the body, thus offering a serious impediment to respiration. In the lateral position, also, the mucus

and sputa drain away more readily. In a majority of instances the pulse-tension will be found high, and measures should be taken to reduce it. In part this increased tension may be due to the suffocative symptoms associated with the stertor, and Bowles states that the pulse-tension is lowered with the relief of the stertor by proper posture. The most rapid and satisfactory method of reducing the tension when very high is venesection, which is indicated in the case of middle-aged men with arterio-sclerosis, high tension, hypertrophied left ventricle, and a ringing aortic second sound. With a small pulse of low tension and signs of cardiac weakness bleeding is contraindicated, and in the cases of apoplexy due to embolism and thrombosis venesection would probably do more harm than good, by favoring the tendency to clotting. Recently, on experimental grounds, Horsley and Spencer have recommended the practice formerly employed empirically of compression of the carotid, particularly in the ingravescent form of apoplexy. In suitable cases they would advise even the passing of a ligature around the vessel. An ice-bag may be placed upon the head. It is not at all likely that sinapisms to the feet or blisters on the back of the neck are of the slightest benefit. The bowels should be freely opened, either by calomel or by croton oil placed on the tongue. Stimulants are not necessary unless the pulse becomes feeble with signs of collapse, when ammonia and brandy may be administered. Especial care should be taken to avoid bed-sores, and if hot bottles are used to the feet, it should be remembered that in this condition burns are more readily caused than in health. In the fever of reaction aconite may be cautiously used.

The treatment of softening from thrombosis or embolism is unsatisfactory. Venesection, as it lowers the tension and promotes clotting, should not be employed. If the pulse be feeble and irregular, alcohol and small doses of digitalis, with ammonia and ether, may be used. The bowels should be kept open, but it is not well to purge actively as in cerebral hæmorrhage. In the thrombosis following syphilitic arteritis we see very satisfactory results from treatment. In such cases, met with most frequently in men between the ages of twenty and forty, the hemiplegia may set in without any loss of consciousness. Iodide of potassium should be given freely in from thirty- to sixty-grain doses three times a day; and, if the infection be recent, mercury may also be employed.

Not much can be done for the residual hemiplegia of cerebral hæmorrhage or embolism. The paralyzed limbs may be rubbed once or twice a day to maintain the nutrition of the muscles and to prevent, if possible, contractures. Electricity is probably of no special benefit, and it is certainly not comparable in value to frictions and systematic massage. In complete hemiplegia which persists for more than a few weeks the chances of full recovery are slight. Power returns in the leg sufficient, as a rule, to enable the patient to get about. The movements of the arm at the shoulder-joint are regained, but the finer movements of the hand are permanently lost. In permanent hemiplegia in persons above the middle period of life mental weakness is apt to supervene, and the patients often become emotional and irritable. When con-

tractures develop the friends should be plainly told that the condition is past all relief, and that medicines and electricity will do no good, and that nothing remains but to look after the general health and comfort of the patient.

Aneurism of the Cerebral Arteries.

Miliary aneurisms are not here included, but only the coarse aneurisms of the larger branches.

The condition is by no means uncommon. I have reported 12 cases met with in 800 autopsies at the Montreal General Hospital. This is a much larger proportion than in Newton Pitt's statistics from Guy's Hospital, in which there were only 19 cases in 9000 autopsies. Males are more frequently affected than females. Of my 12 cases, 7 were in males. The condition may be present in early life. One of my cases was a lad of six, and Pitt describes one at the same age. The chief causes are endarteritis, simple or syphilitic, leading to weakness of the wall and dilatation, and more frequently embolism. As pointed out some years ago by Church, the aneurisms are usually found associated with endocarditis. In his recent study Pitt concludes that it is exceptional to find cerebral aneurism unassociated with fungating endarteritis. The dilatation follows the secondary changes in the coats of the vessels at the site of the embolus.

The middle cerebral arteries are most frequently involved. The distribution in my 12 cases was as follows: Internal carotid, 1; middle cerebral, 5; basilar, 3; anterior communicating, 3. Of 154 cases which make up the statistics of Lebert, Durand, and Bartholow, the middle cerebral was involved in 44, the basilar in 41, internal carotid in 23, anterior cerebral in 14, posterior communicating in 8, anterior communicating in 8, vertebral in 7, posterior cerebral in 6, inferior cerebellar in 3 (Gowers). The size varies from that of a pea to that of a walnut. The aneurism is most frequently sacculated, and communicates with the lumen of the vessel by an orifice smaller than the circumference of the sac.

A cerebral aneurism may attain considerable size and cause no symptoms. In a majority of the cases the first intimation is rupture with fatal apoplexy. Symptoms are most frequently caused by aneurism of the internal carotid, which may compress the optic nerve or the chiasma, causing optic neuritis. The third nerve may also be involved. When large there may be irritative and pressure symptoms at the base. In a case reported by Weir Mitchell and Dercum an aneurism compressed the chiasma and produced bilateral temporal hemianopia. Occasionally a murmur may be audible on auscultation of the skull, but it is to be borne in mind that in a great majority of instances in which a murmur can be heard over the temporal region, even at a distance from the skull, it indicates simply an ordinary systolic brain-murmur which is of no special significance. Aneurism of the vertebral or of the basilar artery may involve the nerves from the fifth to the twelfth, and on the latter artery it may compress the third nerves and crura.

The diagnosis is, as a rule, impossible. When symptoms exist they are those of tumor.

Thrombosis of the Cerebral Sinuses.

The condition may be primary or secondary, and the clot may occur in the sinuses alone or in the cortical veins as well.

Primary Thrombosis, which is rare, occurs—

(1) In children, particularly during the first six months of life, following diarrhœa; in older children after any exhausting disease. In my experience it has not been very common in children, and the only records among my notes relate to two instances of meningitis, both with thrombosis in the cortical veins as well. Gowers believes that infantile hemiplegia is not infrequently caused by thrombosis of the cortical veins.

(2) In connection with chlorosis and anæmia, an extremely interesting association to which Brayton Ball has called special attention, having collected 9 cases from the literature.

(3) In the terminal stages of cancer, phthisis, and other diseases causing cachexia. In hospital and general practice these are the most common cases, and the clot formed under these circumstances is usually spoken of as "marantic thrombus."

Secondary Thrombosis is very much more common, and is due to extension of inflammation to the sinus-wall in disease of the internal ear, in fracture, in suppurative disease outside the skull, more particularly erysipelas. The lateral sinus is most commonly involved. The thrombus may be small and mural, or large, filling the entire sinus and extending into the jugular vein. Newton Pitt states that of 56 cases in which aural disease caused death with cerebral lesions, in 22 thrombosis existed in the lateral sinus. In more than one-half of the cases the thrombus was suppurating. The inflammation arises usually from necrosis of the posterior wall of the tympanum, rarely from disease of the mastoid cells. The most extensive sinus thrombosis may follow erysipelas, rarely from extension directly through the bone into the longitudinal sinus, more commonly from extension along the nerves.

Symptoms.—In cases of prolonged cachexia in which death has taken place slowly a thrombus is sometimes accidentally found. In other instances there is mental dulness, headache, and gradual torpor, deepening to coma, without any localizing symptoms. In involvement of the longitudinal sinus there has been occasional œdema of the forehead, and distension of the veins has occasionally been noticed, and sometimes epistaxis. In children the fontanelle becomes prominent, and there may be exophthalmus. Convulsions occur in some cases, and there may be vomiting. In the chlorosis cases the head-symptoms have, as a rule, been marked; thus the patient under Ball's care was heavy and lethargic, the pupils were dilated, and there was double choked disk. There was also slight paresis of the left side. In the cases to which he refers, reported by Andrew, Church, Tuckwell, Owen, and Wilks, headache, vomiting, and delirium were present. In Powell's case, with similar

symptoms, there was paresis of the left side. Bristow's case, an anæmic girl aged nineteen, had convulsions, drowsiness, and vomiting; tenderness and swelling developed in the position of the right internal jugular vein, and a few days later on the opposite side. The diagnosis was rendered positive by the occurrence of phlebitis in the veins of the right leg; swelling also occurred in the left leg in Brayton Ball's case.

The diagnosis of primary thrombosis can sometimes be made, particularly in chlorosis cases, in which the thrombi are multiple. In infants and in the forms due to cachexia the symptoms are more doubtful. In thrombosis of the cavernous sinuses there may be œdema of the eyelids and marked prominence of the eyeballs.

The symptoms of secondary thrombi are those of secondary septicæmia. As already mentioned, the condition most frequently follows extension from disease of the middle ear, and involves the lateral sinus. The frequency of this accident may be gathered from Pitt's Guy's Hospital statistics, already mentioned. Headache, chills, and fever are the most constant symptoms. Earache is of course very commonly present, and of other symptoms vomiting, coma, delirium, and convulsions occur. Sometimes there is great pain at the back of the head and the neck is stiff. When the thrombus extends into the internal jugular vein, there may be local fulness in the lateral region of the neck or abscess-formation along the course of the vein. Optic neuritis may be present. The duration after the first onset of the symptoms may be from ten days to eight or ten weeks. As a rule, the patient passes into a typhoid condition, with dry tongue, rapid pulse, and all the symptoms of septicæmia. In three-fourths of the cases death follows from pulmonary pyæmia. Pitt's deductions from his cases of lateral sinus thrombosis are important: 1, the disease more often spreads from the posterior wall of the middle ear than from the mastoid cells; 2, the otorrhœa is generally of some standing, but not always; 3, the onset is sudden, the chief symptoms being pyrexia, rigors, pain in the occipital region and in the neck, associated with a septicæmic condition; 4, well-marked optic neuritis may be present; 5, the appearance of acute local pulmonary mischief or of distant suppuration is almost conclusive of thrombosis; 6, the average duration is about three weeks, and death is generally from pulmonary pyæmia.

The treatment of these cases is most unsatisfactory, as the dangers of pyæmia are extremely great. Pitt recommends that the internal jugular vein be ligatured in the neck, the lateral sinus opened, and the clot scraped out. He gives an interesting case of a boy aged ten with chronic otorrhœa who was admitted with earache, tenderness, and œdema. A week later he had a rigor, and optic neuritis developed on the right side. The mastoid was incised without results. The rigors and pyrexia continuing, two days later the lateral sinus was explored, a mass of foul clot removed, and the jugular vein tied, after which the boy made a satisfactory recovery.

Cerebral Localization.

Our accurate knowledge of the functions of the different portions of the brain dates from the observations of Fritsch and Hitzig. Previous to this time interesting attempts at localization were made by the study of pathological cases, and to Broca and Hughlings Jackson, more than to any others, we owe the stimulus to the clinical study of this question.

In the cerebral convolutions there are areas concerned with the muscular movements—motor centres; with sensation—sensory centres; with the special senses of sight, hearing, smell, and touch. There are also psychical centres about the situation of which we as yet know very little.

Motor Centres.—The area for the representation of movements is in the Rolandic region, and comprises the two ascending convolutions, the hinder part of the three frontal convolutions, and a part of the parietal lobule. (See Fig. 45.) Weak electrical currents in this region produce muscular movements in the opposite side of the body. The centres presiding over the different groups of muscles are thus classified:

Fig. 45.

Lateral Surface of Brain of Monkey (Horsley and Schäfer).

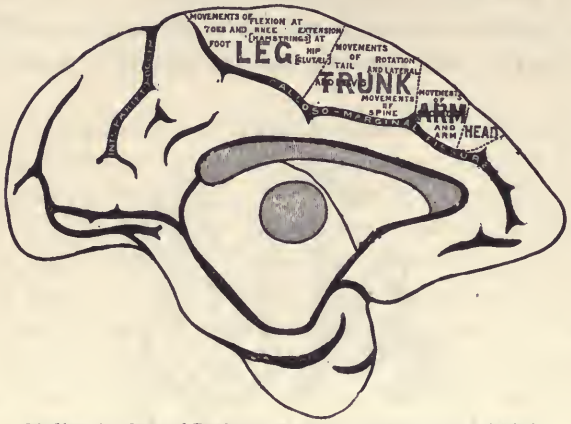
(*a*) The centres for the trunk-muscles are situated just within the longitudinal fissure in the marginal gyrus, in the region sometimes spoken of as the paracentral lobule (Schäfer). (See Fig. 46.)

(*b*) The leg-centres are situated at the upper part of the Rolandic region. The representation of movements of the different portions of the leg is as follows: most anteriorly, the hip; next in order, the knee and ankle; then the big toe, the centre for which surrounds the upper end of the fissure of Rolando; and still farther back centres for the small toes.

(*c*) The arm-centres correspond to about the middle two-fourths of the motor area. The studies of Horsley and Beevor have shown that the different segments of the limb are represented in the following order from above downward: shoulder, elbow, wrist, fingers, the index finger, and last of all the thumb.

(*d*) The centres for the face, tongue, pharynx, and larynx are situated in the lowest portion of the Rolandic region, next to the fissure of Sylvius. From behind forward we have here the following centres: (1) opening of the mouth,

FIG. 46.



Median Surface of Brain of Monkey (Horsley and Schäfer).

around the lower end of the fissure of Rolando; (2) movements of mastication; and (3) contraction of the vocal cords. Anterior to this, in the posterior part of the third left frontal convolution, there is the area concerned with the motor mechanism of speech. In front of the præcentral sulcus are centres for the representation of movements for the turning of the head and eyes to the opposite side.

The determination of these various motor areas has been worked out accurately in animals, and has been established in the case of man partly by careful clinical observation and partly by the direct application of electricity to different regions of the cortex cerebri during operation. The various areas for the representation of movements in the cortex must not be regarded as accurately limited and defined, but as blending one with another.

Uniting these cortical motor centres and the gray matter of the spinal cord are the fibres of the pyramidal or motor tract, which enter the white matter of the hemispheres, the corona radiata, and gradually converge to what is known as the internal capsule, which lies between the thalamus and the two divisions of the corpus striatum. The position of the fibres from the various centres has been pretty carefully determined and represented in the annexed figure. (See Fig. 47.) The fibres from the centres of the face, tongue, eyes, and head occupy the most anterior position, just at the knee, as it is called, of the capsule; the fibres from the arm-centres lie close to these; while the fibres from the leg-centres occupy a position in the middle of the posterior part. After leaving the internal capsule the motor fibres pass into the crus, occupying the lower and medial position. Passing through the pons, they enter the medulla, of which they form the anterior or pyramidal tract, which then decussates, a large portion of the fibres passing to the opposite side of the spinal cord, form-

ing the crossed pyramidal tract, a smaller number of the fibres descending in the anterior column of the same side, forming the direct pyramidal tract or Turck's column. Ultimately the fibres enter the gray matter of the spinal cord and join the plexus of the protoplasmic processes, uniting in this way with the large nerve-cells of the anterior horns.

Lesions of the Motor Centres and Cerebral Motor Path.—The integrity of the fibres of the motor tract depends upon the vitality of the cortical ganglion-

FIG. 47.

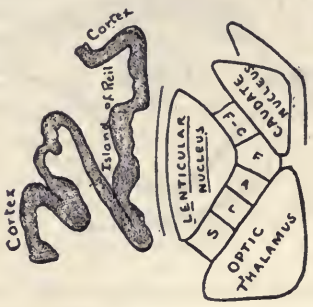


Diagram of Horizontal Section through the Basal Ganglia and Internal Capsule (left side), showing the position of the chief tracts in the internal capsule. The region of the capsule marked by the letters L A F is occupied by motor fibres; L corresponds to the leg-fibres, A to the arm-fibres, F to the face fibres (including fibres to face muscles, and tongue). The region F-C contains the fronto-cerebellar tract (intellectual tract). The region marked S contains the general sensory tract from the opposite side and the fibres from the optic and olfactory nerves of the opposite side, sometimes called the "sensory crossway" (Herter).

cells. If the cells from which they arise are destroyed, the fibres degenerate throughout their length; that is, to the beginning of the lower or spino-muscular motor path. This process, known as secondary or Wallerian degeneration, is a very common event in disease of the brain involving the centres or the pyramidal tract.

The various lesions may be grouped, as Hughlings Jackson suggests, into negative or positive, or, as they are now more commonly termed, destructive and irritative. A negative or destructive lesion anywhere in the motor path results in loss of function in the parts—that is, paralysis; while a positive or irritating lesion causes perversion of the function—*i. e.* abnormal muscular contraction.

(1) DESTRUCTIVE LESIONS.—These cause paralysis, with secondary degeneration and certain characters which distinguish the lesions of the upper or cerebro-spinal tract. Thus the paralysis is accompanied by a condition of spasm shown in an exaggeration of the reflexes and an increase in the muscle-tension. How this is brought about is not yet accurately known, but the explanation usually offered is that under normal circumstances the upper motor centres constantly exert a restraining influence upon the lower (spinal) centres. When this influence is abolished on account of disease in the pyramidal tract, these lower centres take on increased activity, which is manifested by an exaggeration of the reflexes. As the segments of the motor path are

separate for nutritional purposes, the muscles neither undergo degenerative atrophy nor present the reaction of degeneration.

As the motor centres of the cortex are separated more or less from each other, a localized lesion may cause limited paralysis confined to one limb or to one side of the face—the cerebral monoplegias. Where the pyramidal fibres run in a compact bundle, as in the internal capsule, a destructive lesion is more apt to cause paralysis of all the muscles on one side of the body; that is, hemiplegia.

(2) Irritative Lesions.—Our knowledge of such lesions is confined for the most part to those acting on the cortical motor centres, and we know a number of processes which have as their result abnormal muscular contractions. These have as their type the localized convulsive seizures classed as Jacksonian or cortical epilepsy, which are characterized by the convulsion beginning in a single muscle or group of muscles and involving other muscles in a definite order, depending upon the position of their representation in the cortex—for instance, such a convulsion beginning in the muscles of the face next involves those of the arm and hand, and then the leg. The convulsion is usually accompanied by sensory phenomena and followed by a weakness of the muscles involved.

A majority of lesions of the motor cortex are both destructive and irritative—*i. e.* they destroy the nerve-cells of a certain centre, and either by their growth or presence throw into abnormal activity those of the surrounding centres.

Sensory Centres.—Our knowledge of the exact position of the areas for representation of the sensory impressions is still defective. Ferrier places it in the hippocampal convolution, but the experiments of Schäfer suggest that the gyrus fornicatus is also concerned in sensory impressions. As the tactile and muscular senses play such an important rôle in all muscular movements, and are sometimes disturbed in lesions of the motor cortex, it seems not unlikely that their centres are associated with those of motion. Horsley has suggested that they are localized in the motor cortex, and that two of the chief layers of cells in this region may possibly subserve their functions. Dana's study of a large collection of cases indicates that anæsthesia is very frequently associated with lesions of the motor cortex, more particularly in the posterior half of the motor area.

Centres for the Special Senses.—As already mentioned, the cortical representation of the sense of sight is in the occipital lobe, more particularly in the cuneus, unilateral destruction of which is followed by hemianopia. The relation of the angular gyrus, which Ferrier believes is concerned with vision, is still undetermined, and it seems probable, so far as man is concerned, that the visual area is in the occipital lobe. The cortical centre for hearing has not yet been fully determined, though it seems to bear very close relation to the temporal lobe, as lesion of the posterior part of the first left temporal convolution is followed by the phenomenon known as word-deafness, and bilateral destruction of these parts in the monkey produces, as shown by Ferrier, complete

deafness. The centre for smell has been placed by Ferrier in the temporo-sphenoidal lobe and in the uncinate gyrus. The centre for taste has not yet been accurately localized. The parts of the brain which subserve the higher psychical functions are believed to be in the frontal lobes. This opinion is based upon the greater development of these lobes in man and the frequent association of mental impairment when they are diseased.

The following is the summary of the functions and the effects of lesions in other regions of the brain:

Centrum Ovale.—The white substance situated between the gray cortex and the basal ganglia contains (1) the projection system of fibres, which unites the cerebral cortex with the other ganglionic masses and with the spinal cord; (2) the commissural fibres, which join corresponding portions of the hemispheres; and (3) the association tracts, which unite adjacent convolutions.

Lesions of the fibres of the projection system cut off communication with the cortical centres, the effect of which naturally depends upon the portion involved; thus lesion of the fibres of the motor path causes paralysis, which is practically the same as if the centre itself was destroyed. Subcortical lesions involving only a limited number of the projection fibres of the motor path may cause monoplegias. Lesions in the white matter, as the fibres converge to the internal capsule, are more likely to be followed by hemiplegia. Involvement of the white fibres of the optic radiation in the occipital lobe may cause hemianopia and word-blindness, and of the fibres of the temporal lobe, word-deafness. Sensory disturbances are rare from lesion in the centrum ovale proper, but hemianæsthesia is caused by destruction of the fibres near the hinder part of the internal capsule. Interruption of the association tracts between the auditory and visual centres and Broca's convolution may cause forms of disturbance of speech, and a lesion interrupting the fibres from Broca's centre causes motor aphasia.

There is much uncertainty in the diagnosis of lesions of the centrum ovale, and there may be extensive disease, particularly in the prefrontal region, without special symptoms.

Internal Capsule.—As already stated, this important tract of white matter lying between the thalamus and the two divisions of the corpus striatum contains the pyramidal fibres, the sensory fibres, and those of the special senses. The diagram already given shows the position of the motor fibres. Briefly stated, lesions of the posterior part of the hinder limb of the internal capsule cause hemianæsthesia and hemianopia, and there have been instances in which the special senses of hearing, taste, and smell have been involved. In association with lesions of the hinder part of the internal capsule and the contiguous portion of the optic thalamus, choreiform and athetoid movements have been described.

So far as we know, lesions confined to the caudate and lenticular nuclei and of the optic thalamus produce no definite symptoms, unless, as is so often the case, the internal capsule be simultaneously involved.

The corpora quadrigemina are rarely diseased alone. Lesions of the

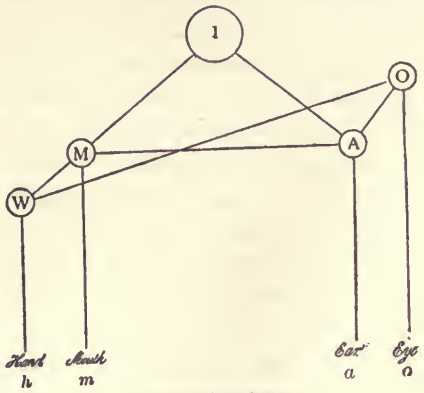
anterior pair result in blindness. Pupillary symptoms are common, and there is usually paralysis of the oculo-motor nerve. Involvement of these parts by tumors is very apt to be followed by hydrocephalus.

In the crus cerebri the motor and sensory fibres are collected in a very small space, and a lesion may cause hemiplegia of both motion and sensation. The third nerve is frequently involved in lesions of the crus, causing paralysis of the muscles of the eye on the same side with hemiplegia on the opposite side. Lesions of the pons in the lower part may cause paralysis of the leg and arm on the opposite side, and, involving the nucleus or fibres of the facial nerve, cause paralysis of the same side of the face. This is known as crossed hemiplegia. In the upper part of the pons the lesion produces the ordinary type of hemiplegia. Extensive lesions of the pons involve both pyramidal tracts, causing loss of power in both sides of the body.

Aphasia.

The central apparatus concerned with speech is made up of receptive, perceptive, and emissive centres in the cerebral cortex, the disturbances of which are considered under the term "aphasia." Disturbance of the centres which preside over the peripheral speech-mechanism, the muscles of phonation and

Fig. 48.



Lichtheim's Schema.

articulation, produces the condition known as *anarthria*, as in the gradual loss of the power of speech in bulbar paralysis.

Articulate language is gradually acquired by imitation; thus in teaching a child to say the word "bell" the sound of the word as uttered enters the afferent path, reaching the auditory perceptive centre, from which the impulse is sent to the motor or emissive centre presiding over the nuclei in the medulla, from which the muscles of articulation are set in action. The arc in Lichtheim's schema (see Fig. 48) is *a*A, M*m*. In this way the child gradually acquires word-memories which are stored at the centre A, and motor memories—that is to say, the memories of the co-ordinating muscular movements necessary to utter the word—which are stored at the letter M. So also when

shown the bell, visual memories are acquired of its size and shape, which are conveyed through the optic nerve to the visual perceptive centre along *o*O. In the auditory perceptive centres is also stored the sound of the bell when struck. The memory picture of the shape of the bell or its sound when struck, of the appearance of the word when written, the motor memories of the movement required to write the word, are distinct from each other, may be separately disturbed, and yet are intimately connected and together form what is termed the *word-image*. In addition to this, the child gradually acquires ideas as to use of the bell—intellectual concepts, the centre of which is represented at I in the diagram. In volitional speech, as in uttering the word "bell," the path would be represented in I, M *m*; in writing the path would be represented in I, M, W, *h*. The various "memories" are, as a rule, centred or stored in the left half of the brain.

Aphasia in the widest sense of the word may be taken to embrace disturbances either at (*a*) the sensory perceptive centres of hearing and sight and in the blind of touch; (*b*) of the emissive or motor centres of speech and writing; or (*c*) of the psychical centres through which we gather rational conceptions of what is said or written, and by which we express voluntarily our ideas in language.

Two chief forms of aphasia are recognized—the sensory, in which the psychical and sensory perceptive centres are disturbed; and the motor, in which the emissive for speech and writing are involved.

Sensory Aphasia.—Loss of the power to recognize the nature and characteristics of objects is known as apraxia, which is thus clearly and accurately defined by Starr: "It is a fundamental position involved in the accepted theory of cerebral localization that memories are the residua of perceptions, and are therefore localizable in the regions of the brain concerned in perception. It follows that these memories forming the idea of an object or an action, being distinct from one another, may be lost by disease of the brain having a limited extent, and that the character of the memories lost will depend on the location of the disease. Now, cases have been recorded in which persons acted as if they no longer possessed such object memories, for they failed to recognize things formerly familiar. A fork, a cane, a pen may be taken up and looked at by such a person, and yet held or used in a manner which clearly shows that it awakens no idea of its use. And this symptom, for which at first the term 'blindness of the mind' was used, is found to extend to other senses than that of sight. Thus the tick of a watch, the sound of a bell, a melody of music, may fail to arouse the idea which it formerly awakened, and the patient then has deafness of mind; or an odor or taste no longer calls up the notion of the thing smelt or tasted; and thus it is found that each or all of the sensory organs, when called into play, may fail to arouse an intelligent perception of the object exciting them. For this general symptom of inability to recognize the use or import of an object the term *apraxia* is now employed. And since apraxia is a symptom which is very frequently associated with aphasia, and which, in fact, may lie at the

basis of aphasia, it should always be looked for in a patient. To test for apraxia it is only necessary to present various objects to a person in various ways, and notice whether he gives evidence of recognition. Have him watched by his friends, and they will be able to tell whether he still chooses his articles of food at the table intelligently—whether he still knows how to put on his clothes, to use various toilet articles, to sew or knit or embroider if the patient is a lady, to admire pictures, or flowers, or perfumes, as before the illness began. The patient may or may not be able to name these objects: that, at present, is not the question. But is it evident that the object awakens an idea in the mind?" There are instances in which apraxia may be the only symptom. Thus, a young man in the secondary stage of syphilis was seized while at his office-work with a convulsion. A day or two subsequently, when I saw him, there was no paralysis and no motor aphasia, but he had completely lost the memory-pictures of faces and places. The street in which he lived was quite unfamiliar to him, and he did not know his way to the office at which he worked. He also did not recognize for some time his parents or brothers. As a rule, apraxia is associated with varieties of sensory and motor aphasia. The patient may be able to read, but the words arouse no intelligent ideas in his mind. While blind to memory-pictures aroused by sight, the perceptions may be stimulated by touch; and there have been patients unable to read by sight who, on tracing the letters by touch, named them correctly. Mind-blindness and mind-deafness are the common and important forms of apraxia.

Mind-blindness, which is the equivalent of visual amnesia, may be functional and transitory or associated with organic disease, often with mental disturbance. The cases collected by Starr indicate that the lesion exists in the left hemisphere in right-handed persons, and in the right hemisphere in left-handed persons. The disease usually involves the angular and supramarginal gyri or the tracts proceeding from them. In a remarkable case reported by Macewen the patient, after an injury to the head, had suffered with headache and melancholia, but there was no paralysis. He was psychically blind, and, though he could see everything perfectly well and could read letters, objects conveyed no intelligent impression. A man before his eyes was recognized as some object, but not as a man until the sounds of the voice led to the recognition through the auditory centres. The skull was trephined over the angular gyrus, and the inner table was found to be depressed and a portion had been driven into the brain in this region. The patient recovered.

Word-blindness may occur alone or with motor aphasia. The patient is no longer able to recall the appearances of words, and does not recognize them on a printed or written page. The patient may be able to pronounce the letters and can often write correctly, but he cannot read understandingly what he has written. It is rare, however, for the patient to be able to write with any degree of facility. There are instances in which the patient, unable to read, has yet been able to do mathematical problems and to recognize play-cards. The lesion in cases of word-blindness is, in a majority of cases, in the angular

and supramarginal gyri on the left side. It is commonly associated with hemianopia, and not infrequently with mind-blindness.

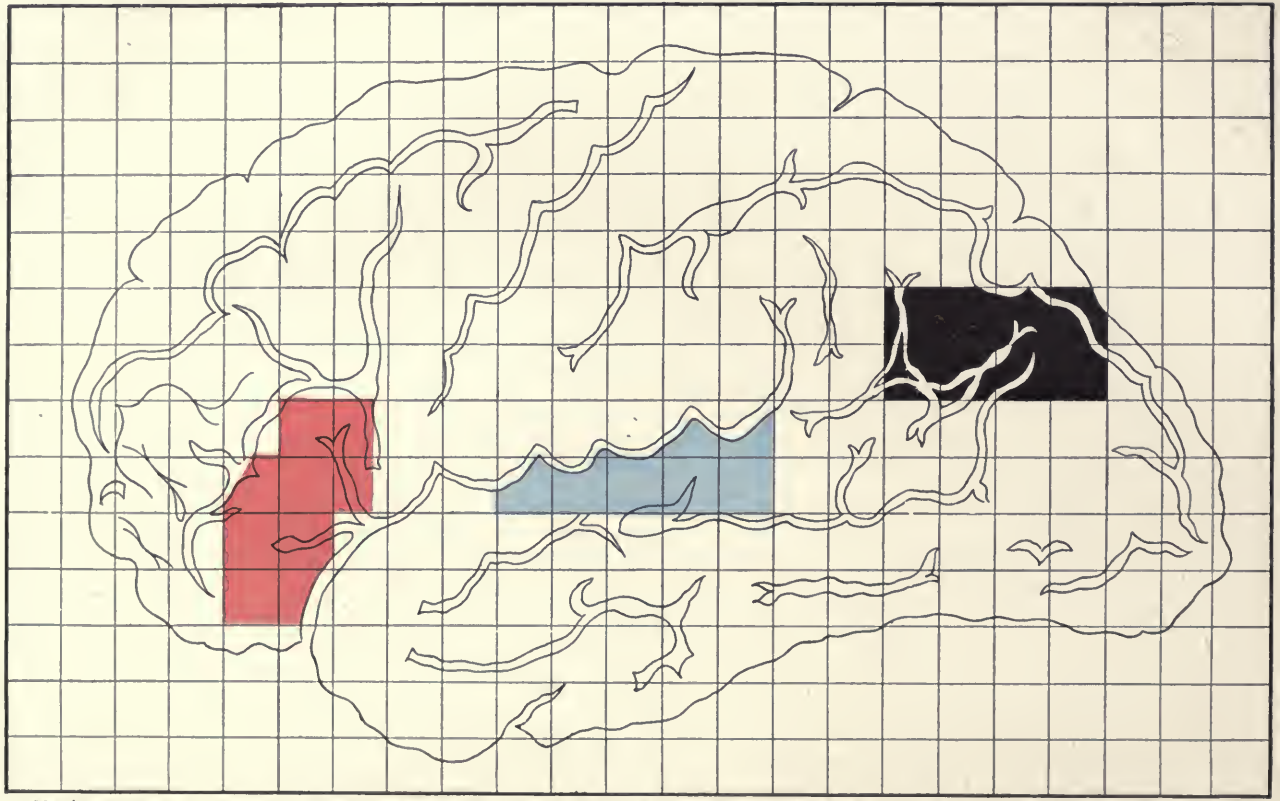
Mind-deafness, or auditory amnesia, is a condition in which sounds, though heard and perceived as such, awaken no intelligent conceptions. A person who knows nothing of French has mind-deafness so far as the French language is concerned, and though he recognizes the words as words when spoken, and can repeat them, they awaken no auditory memories. The musical faculties may be lost in aphasics, who may become note-deaf and unable to appreciate melodies or to read music. This may occur without the existence of motor aphasia; and, on the other hand, there are cases on record in which with motor aphasia for ordinary speech the patient could sing and follow tunes correctly. Word-deafness is a condition in which the patient no longer understands spoken language. The memory of the sound of the word is lost, and can neither be recalled nor recognized when heard. It is usually associated with other varieties of aphasia, though there are cases in which the patient has been able to read and write and speak. The lesion in word-deafness has been accurately defined in a number of cases to be in the posterior portion of the first and second temporal convolutions on the left side (Plate II.).

Motor or *ataxic aphasia* is a condition in which the memory of the efforts necessary to pronounce words is lost, owing to disturbance in the emissive centres. This is the variety long ago recognized by Broca, the lesion of which was localized by him in the third left frontal convolution. In pure cases the patient is able to read (not aloud), and understands perfectly what is said. He may not be able to utter a single word; more commonly he can say one or two words, such as "no," "yes," and he not infrequently is able to repeat words. When shown an object, though not able to name it, he may evidently recognize what it is. If told the name he may be able to repeat it. A man knowing the French and German languages may lose the power of expressing his thoughts in them while retaining his mother-tongue, or, if completely aphasic, may recover one before the other. As the third left frontal convolution is in close contact with the centres for the face and arm, these are not uncommonly involved, with the production of a partial or, in some instances, a complete, right-sided hemiplegia. *Alexia*, or inability to read, occurs with motor aphasia and also with word-blindness.

As a rule, in motor aphasia there is also inability to write—*agraphia*. When there is right brachial monoplegia it is difficult to test the capability, but there are instances of motor aphasia without paralysis in which the power of voluntary writing is lost. The condition varies very much; thus a patient may not be able to write voluntarily or from dictation, and yet may copy perfectly. It is still a question whether there is a special writing centre. It has been placed by some writers at the base of the second frontal convolution, but in a recent study Dejerine concludes that it is not separated from the speech-centre.

There is a form known as *mixed aphasia* or *paraphasia*, in which the patient understands what is said, and speaks even long sentences correctly,

PLATE II.



Maximum Foci of Disease in Seventy-one Cases of Aphasia (Naunyn): *Red*, cortical area for motor aphasia (Broca); *blue*, cortical area for aphasia with word-deafness (Wernicke); *black*, cortical area for alexia or word-blindness.

but constantly tends to misplace words, and does not express his ideas in the proper words. All grades of this may be met with, from a state in which only a word or two is misplaced to an extreme condition in which the patient may talk jargon. In these cases the association-tract is interrupted between the auditory perceptive and the emissive centres; hence it is sometimes known as Wernicke's aphasia of conduction. The lesion is usually in the insula and in the convolutions which unite the frontal and temporal lobes.

Naunyn's figure (Plate II.) gives an accurate representation of the localization of the lesion in the forms of motor and sensory aphasia.

Lichtheim's schema will assist the student in obtaining a rational idea of the varieties of aphasia:

1. In the condition of apraxia or mind-blindness the ideation centres, I, are involved, often with the auditory and visual perceptive centres, A and O.

2. A lesion at A, the centre for the auditory memories of words (first left temporal gyrus), is associated with word-deafness.

3. A lesion at O, the centre for visual memories (angular and supramarginal gyri), causes word-blindness.

4. Interruption of the tracts uniting A M and O M causes the conduction aphasia of Wernicke—paraphasia.

5. Destruction of the centre M (Broca's convolution) causes pure motor aphasia, in which the patient cannot express thoughts in speech.

A lesion at M usually destroys also the power of writing, but, as stated, it is believed by many that the centre for writing, W, is distinct from that of speech. In this case a lesion at M, which would destroy the power of voluntary speech, might leave open the connections between O W and A W, by which the patient could copy or write from dictation.

The following tests should be applied in each case of aphasia: (1) The power of recognizing the nature, uses, and relations of objects—*i. e.* whether apraxia be present or not; (2) the power to recall the names of familiar objects seen, smelled, or tasted, or of a sound when heard or of an object touched; (3) the power to understand spoken words; (4) the capability of understanding printed or written language; (5) the power of appreciating and understanding musical tunes; (6) the power of voluntary speech—in this it is to be noted particularly whether he misplaces words or not; (7) the power of reading aloud and of understanding what he reads; (8) the power to write voluntarily and of reading what he has written; (9) the power to copy; (10) the power to write at dictation; and (11) the power of repeating words.

Treatment.—In the young aphasia associated with hemiplegia from whatever cause is usually transitory, and they quickly learn to talk, probably by education of the centres of the opposite side of the brain. In adults the condition is much less hopeful, particularly in cases of complete motor aphasia with right hemiplegia. Sometimes the recovery is rapid; in others partial recovery occurs and the patient is able to talk, but he misplaces words. If motor aphasia has persisted for several months without improvement, the con-

dition is generally hopeless and the patient may remain speechless, though capable of understanding everything that is said. The education of an aphasic demands the utmost patience, and when the patients are emotional and irritable the attempts are often futile. Detached letters should be at first used, with which small words of one syllable may be constructed, and progress made slowly. The most distressing cases are those of permanent aphasia with right hemiplegia. When the mental condition is good, the patient may with great care be taught to write with the left hand, and so have a medium of communication. Too often the utmost care and pains prove fruitless in these cases.

Cerebral Palsies of Children.

In children palsies due to cerebral disease occur with a frequency almost equal to those of spinal origin.

1. Hemiplegia.—The disease appears to be somewhat more frequent in girls; thus in my series of 135 cases, comprising cases from the Infirmary for Diseases of the Nervous System, Philadelphia, from the Elwyn Institution for Feeble-minded Children, and from my clinic at the Johns Hopkins Hospital, 75 were girls. In a large majority of cases the disease sets in during the first or second year; thus of the total number of cases, 95 were under two years of age. Above the fifth year the disease is rare—there were only 10 cases in my series. Neither alcoholism nor syphilis in the parents plays a rôle in the affection. Injury with the forceps in instrumental delivery is an occasional cause, though not so important as in the spastic diplegia and paraplegia of children. Falls and punctured wounds are occasionally causes, and in one instance in the Elwyn Institution the hemiplegia followed ligation of the common carotid. A certain number of cases set in during the height of or follow an infectious fever. In my series cases are mentioned after scarlet fever, measles, whooping cough, and vaccinia. There are cases in the literature mentioned also after mumps. In some of these instances the paralysis followed the initial convulsion; in others it was not until the fever had subsided that fits came on, and with them paralysis. In the whooping-cough cases the hemiplegia may follow a prolonged spasm of coughing, but in the three cases in my series it occurred with convulsions during the disease.

In a large majority of the cases the disease sets in with severe convulsions, often without any premonition, and in children who have previously been robust and healthy. The importance of convulsions in this affection may be gathered from the combined statistics (those of Wallenberg, Gaudard, Gowers, Sachs, and my own), numbering 428 cases, of which nearly one-half set in with convulsions.

Morbid Anatomy.—The nature of the primary lesion is, in a majority of the cases, unknown. The autopsies which have been reported have almost invariably been late, years after the onset of the hemiplegia. In an analysis which I made of 90 autopsies from the literature the lesions could be grouped as follows:

(*a*) In 16 cases there was embolism, thrombosis, or hæmorrhage. In 7 of these a Sylvian artery was occluded; in 9 there was hæmorrhage. It is interesting to note that in 10 of these cases the disease set in in children over six years of age.

(*b*) Atrophy and sclerosis, which are the common conditions in the majority of the cases. The wasting is either of groups of convolutions, an entire lobe, or one hemisphere. The meninges may look normal, though more commonly they are tightly adherent and the brain-substance tears. The convolutions are shrunken, firm, and hard. In some instances there is a remarkable unilateral atrophy, in which the brain-tissue is a mere shell over the greatly dilated ventricle. Thus, in one of my cases the atrophied hemisphere weighed 169 grammes and the normal 553 grammes.

(*c*) Porencephalus, which was present in 24 of the 90 autopsies. This term was applied by Heschl to a loss of substance in the form of cavities or cysts at the surface of the brain, communicating with the arachnoid space, and in some cases passing deeply into the hemisphere, reaching even to the ventricle. Of 103 cases of porencephalus analyzed by Audry, hemiplegia was present in 68.

Cortical sclerosis and porencephalus are then the most important anatomical conditions, but the nature of the causative processes is still doubtful. Gowers suggests that thrombosis of the superficial veins and sinuses is a frequent cause, but, though this has been found in some cases, they are few in number; and it is to be remembered that in a considerable proportion of all cases the disease sets in in healthy children. Strümpell has suggested that the primary condition is an inflammation of the gray matter in the motor cortex analogous to the inflammation of the gray cornua in the spinal cord—polioencephalitis. Practically, it is better to acknowledge our ignorance of the initial lesion, and there is no section in cerebral pathology more in need of careful anatomical work than this group of cases.

Symptoms.—(*a*) *Of Onset.*—As already mentioned, the disease sets in with partial or general convulsions and loss of consciousness. The convulsions may recur for several days, during which time the child is unconscious. Slight fever is usually present. Occasionally the hemiplegia develops suddenly in an apparently healthy child, without spasms or loss of consciousness; in other instances the paralysis sets in gradually. The child has usually begun to walk, and the hemiplegia is quickly noted as soon as the child recovers consciousness. The face is not always involved. With right hemiplegia aphasia is not uncommon, and occurred in 16 cases of my series, a smaller number than in the series of Wallenberg, Gaudard, and Sachs.

(*b*) *Residual Symptoms.*—In some instances the paralysis disappears almost completely, leaving scarcely a trace. In one of the Elwyn cases the mental defect and a slightly impaired development of the affected side alone remained of an infantile hemiplegia which had persisted for some time. The leg, as a rule, recovers rapidly and more fully than the arm. In a majority of cases there is a characteristic hemiplegic gait. The paralysis is most marked in the

arm, which is wasted, the forearm is flexed at right angles, the hand strongly flexed at the wrist, and the fingers contracted. Motion may be completely lost in the forearm and hand, but is usually retained to some extent in the shoulder muscles. Late rigidity is almost constant, and was the symptom which suggested the name *hemiplegia spastica cerebralis* to Heine, the orthopædic surgeon who first described these cases. The limbs, however, may be quite relaxed—the *hemiplégie flasque*. The atrophy may be striking, and in this disease we see admirable illustrations of the atrophy following a cerebral lesion. As a rule sensation is not disturbed. The reflexes are usually increased.

Mental Defects.—One of the most serious consequences of infantile hemiplegia is failure in mental development, in consequence of which children drift into the institutions for feeble-minded children. There may be idiocy, which is most common when the hemiplegia has existed from birth or has come on at a very early period; imbecility, which may increase with the development and persistence of epilepsy; and a condition of feeble-mindedness, a retarded rather than arrested mental development.

Epilepsy is one of the most common and distressing of the residual symptoms of infantile hemiplegia. Of the cases in my series, 41 were subject to convulsive seizures. In other cases there is only *petit mal*. The convulsions may begin in, and be confined to, the affected side without loss of consciousness—true Jacksonian epilepsy—or there are general convulsions, usually beginning in the paralyzed limbs.

Post-hemiplegic movements.—It was in cases of this kind that Weir Mitchell first described the post-hemiplegic movements. They are extremely common, and were present in 34 of my series. There may be only post-hemiplegic tremor, in which the arm or the leg vibrates gently; more commonly the movements are inco-ordinate and choreiform; or, lastly, there may be athetosis. In this condition, which was described by Hammond, there is a remarkable spasm in the paralyzed limbs, chiefly in the fingers and toes, and in rare instances in the muscles of the face. The muscular movements are involuntary, often rhythmical, and in the hand the motions of adduction and abduction and of supination and pronation may follow each other in orderly sequence. The fingers are frequently hyperextended and spread far apart. The movements are usually increased by emotion, and in some cases persist during sleep. Athetosis is very much more frequent in hemiplegia of children than in adults. In the latter it may be combined with hemianæsthesia, in which case the lesion is usually not cortical, but basic, in the neighborhood of the thalamus opticus.

2. Spastic Diplegia.—A condition dating, as a rule, from birth, in which there is paralysis with spasm of the extremities.

As stated in a previous section, infantile hemiplegia occurs usually during the first two years of life. On the other hand, the instances in which both sides are involved very commonly date from birth, and constitute the most serious of all forms of so-called birth-palsies. In some instances the arms are so

slightly affected that there may be a doubt whether the case should be regarded as one of diplegia or paraplegia. The relation of abnormal parturition to the disease is the most important point in its etiology. In very many of the cases the patients have been born in first labors or have been instances of instrumental delivery. In feet presentation too there is the possibility of laceration and tearing of the cerebro-spinal membranes. Asphyxia or convulsions have been present in a very considerable number, and it is very common to hear the statement made in these cases that the child at birth was a "blue baby."

Morbid Anatomy.—The birth-palsies which ultimately induce the spastic diplegias and paraplegias appear to result more frequently from meningeal hæmorrhage, in which the cerebral cortex is damaged, leading ultimately to sclerosis or atrophy. The frequency of meningeal apoplexy in the new-born has been demonstrated by the studies of Litzmann and Sarah J. McNutt. The hæmorrhage has been found thickest over the motor region. Clinically, these cases present the symptoms of asphyxia or convulsions, the manifestations present in a great majority of instances of birth-palsies. It seems not unreasonable to conclude in cases which recover and subsequently present signs of motor disturbance that a similar though less extensive lesion has existed. There are instances, however, which are probably due to fœtal meningo-encephalitis. The anatomical condition in spastic diplegia in 16 cases which I collected from the literature was either a diffuse atrophy or porencephalus.

Symptoms.—As stated above, the child has usually been resuscitated with difficulty or has had convulsions. For some months nothing abnormal may be noticed; then, at an age when the child should begin to walk it is found not to use the limbs readily, and the mother may say that she finds in dressing the child difficulty in moving the arms and legs. The child sits up with difficulty, or may be quite unable to do so, at the age of two years, and very often the head is not well supported, but tends to fall forward. The rigidity is most marked in the legs, and it was this symptom which gave several names to the disease, such as *spastic rigidity of the new-born*, *essential contraction*, and *tonic contraction of the extremities*. When made to stand up, the child either rests upon its toes or upon the inner surfaces of the feet, with the knees close together, or, if the adductor spasm be very great, the legs may be crossed. The stiffness of the arms is rarely so marked, and is sometimes scarcely noticeable. Irregular movements of the arms are not infrequent, and the child has difficulty in grasping objects. The spasm and weakness may be more pronounced on one side. Convulsive seizures are not uncommon, and the mental condition is, as a rule, defective.

Associated with spastic diplegia there is in certain instances the remarkable condition known as *bilateral athetosis*, in which there is a combination of spasm, more or less marked, with extraordinary bizarre movements of the muscles. The affection usually dates from infancy. A majority of the cases are unable to walk. The head is turned from side to side, the mouth is drawn and distorted, and there are irregular movements of the facial muscles. In making any voluntary effort, or even spontaneously, there are extraordinary

movements of the limbs, and more particularly of the arms, somewhat like, though very much more exaggerated than, ordinary post-hemiplegic athetosis. These vary extremely, and may produce complete disability. In other instances, even when very marked, the patient may be able to feed himself. The reflexes are increased. Many of the cases are feeble-minded or idiotic: 2 of the 4 cases which I have described were intelligent. Audry, who has published a monograph on the subject, has collected over one hundred cases. There have been three autopsies. In Kurella's case there were pachymeningitis and bilateral lesions of the motor area. Dejerine's patient had atrophy of the convolutions on both sides. In my case the brain, macroscopically, showed no changes.

3. Spastic Paraplegia.—The *paraplegia cerebralis spastica* of Heine is a common affection, due in many cases to conditions similar to those found in spastic diplegia. All grades of the disease are met with, from the pure spastic paraplegia with perfect use of the arm to the most extreme bilateral spasm. Evidence of the cerebral origin of the disease is based upon the frequent coexistence of idiocy, imbecility, and nystagmus, and the existence of cases of spastic diplegia in which the paraplegic symptoms are identical. The patients frequently present a typical cross-legged progression. The mental condition is often better than in the cases of spastic diplegia. The primary lesion in these cases is in all probability hæmorrhage during delivery. In the case of Förster the post-mortem showed a moderate grade of cortical sclerosis with slight dilatation of the ventricles. In the case of Sachs there was meningo-encephalitis with atrophy and descending degeneration of both lateral columns.

The **diagnosis** of spastic diplegia and spastic paraplegia is usually easy, but there is a condition known as pseudo-paralytic rigidity which occurs particularly in rickets and in chronic diarrhœa, the differences between which may be thus tabulated:

Pseudo-paralytic Rigidity.	*Spastic Paralysis; Di- and Paraplegia.*
Follows a prolonged illness. Is often associated with rickets, laryngismus stridulus, and the so-called hydrocephaloid state.	Usually exists from birth. History of difficult labor, of asphyxia neonatorum, or of convulsions.
Begins in hands as carpo-pedal spasm; often confined to hands and arms.	Arms rarely involved without legs, and not in such a marked degree.
Spasms painful, and attempts at extension cause pain.	Usually painless.
Intermittent and of transient duration.	Variable in intensity, but continuous.

Treatment of Cerebral Palsies.—The possibility of injury to the brain must be borne in mind in cases of protracted labor. Probably a long-continued compression of the head entails greater risks than the application of forceps. At the onset of a case of infantile hemiplegia the physician, as a rule, thinks he is dealing with a case of ordinary convulsions, perhaps more severe than usual. These should be checked as quickly as possible by the administration of chloroform, and subsequently by the use of bromides. Very little can be done for the paralysis itself, which as a rule improves

gradually, particularly in the leg. Complete recovery is, however, rare. The main indications are to favor the natural tendency to improve by maintaining the general nutrition of the child, to lessen the rigidity and contractures by massage and passive movements, and to correct deformities by mechanical or surgical measures. The aphasia usually disappears. The epilepsy is an obstinate feature which usually persists in spite of all remedies. Prolonged periods of quiescence are, however, not infrequent. The feeble-mindedness is the most distressing symptom, and in too many instances is irreparable. In others, with patient training and care, a fair measure of intelligence and self-reliance may be reached. Of late operative measures have been advised, but when we consider the anatomical condition, which is almost invariably sclerosis, chronic meningo-encephalitis, or porencephalus, it is evident that not very much can be looked for. Certainly the recent review by Starr of the subject with special reference to this point cannot be regarded as at all encouraging.

Insular Sclerosis (Sclerose en Plaque).

Definition.—A chronic affection in which certain localized areas in the brain and cord, or more rarely in the brain or cord alone, are replaced by connective tissue. The cerebro-spinal form is the common type.

Etiology.—In a few exceptional instances two or three members of a family have been affected. In a majority of the cases the symptoms do not appear until adult life. The disease is, however, not uncommon in children, and Prichard states that there are more than fifty cases on record in which it began before the fifteenth year. Cold and exposure, mental shock, and injury have been referred to as possible causes. More important are the infectious diseases, and many cases have followed scarlet fever, measles, and diphtheria. The precise relation is, however, not known, though it may be that the areas of localized induration follow the focal myelitis which has been met with as a post-febrile disorder. In very many cases the disease sets in insidiously without any recognizable cause. The disease is most common in males.

Morbid Anatomy.—The sclerotic areas are widely distributed throughout the brain and cord, usually in the white substance. They vary in size from 2 to 20 or more millimetres; they are reddish-gray in color, somewhat translucent, and cut with firmness. The cortex of the brain may look quite natural. The plaques are most abundant toward the basal ganglia. In the cord they may be seen externally as grayish-white patches, usually having a greater vertical than transverse extent. There are instances in which the sclerosis is more diffuse, involving in certain regions the whole thickness of the cord. The foci of disease are found in the cranial nerves, and in some instances in the peripheral nerves. Histologically, the diseased areas are seen to present a dense plexus of new-formed connective tissue which to a great extent has replaced the nerve-fibres. A peculiar feature is that while the medullary sheaths of the nerves undergo atrophy, many of the axis-cylinders persist in

a remarkable manner, and do not disappear until late in the disease. The blood-vessels in the neighborhood of the patches are, as a rule, either sclerotic or present fatty changes. The primary cause of the sclerosis is by no means clear.

Symptoms.—In many cases the clinical history is most distinctive, but atypical forms are by no means uncommon. Charcot describes two modes of onset: in one the progress is insidious, characterized by headache, vertigo, tired feelings in the muscles, and the gradual development of inco-ordination and tremor; in the other form the disease sets in more abruptly, sometimes with a convulsion or apoplectic seizure, following which the symptoms of tremor and weakness develop. The most important symptoms are the following:

Volitional Tremor.—This is most marked as a rule in the arms. When the hands are at rest on the lap there may be not the slightest movement, but on attempting to pick up an object there are sudden jerkings or an oscillatory tremor. For example, in taking a glass of water the tremor increases to such a degree that by the time the mouth is reached some of the water is spilt, and in drinking the glass rattles against the teeth. When recumbent the head may be perfectly quiet, but on lifting it from the pillow the trembling at once begins. In walking the shaking movement of the head is sometimes very characteristic. In the early stage the volitional or intention tremor may be the only marked symptom. Though present in a majority of the cases, disseminated sclerosis may be present without it. There is more or less inco-ordination, and the gait may be staggering and ataxic. More commonly there is a spastic weakness of the muscles of the legs or an ataxic paraplegia. Romberg's symptom is not usually present.

Speech defects are present in many of the cases. There may be a slight indistinctness, due to tremor of the tongue; more commonly the words are pronounced slowly and separately, and the individual syllables may be accentuated. This constitutes the scanning, *staccato*, or syllabic speech, which is more marked in this than in any other chronic nervous affection.

Eye-symptoms are common: diplopia, strabismus, ptosis, and dilatation of the pupil have been frequently noted. Optic atrophy occasionally occurs. Nystagmus is a very frequent symptom, occurring in considerable proportion of all cases.

Sensory disturbances are not common. The reflexes, as a rule, are increased, particularly when there is a spastic condition of the legs. In the cases in which the posterior columns are involved the reflexes may be abolished. The sphincters are unaffected. Epileptic and apoplectiform seizures occur in some cases. Vertigo is not infrequent. A gradual impairment of the mental power is common, and failing memory, with great complacency and content under increasing helplessness, is stated by Gowers to be somewhat characteristic.

The **diagnosis** is easy in many cases, but there are certain affections which simulate it very closely, and there are remarkable atypical cases. In the form

in which the spinal cord is chiefly involved the volitional tremor may not be a marked feature, and according to the situation of the sclerosed areas the chief symptoms may be those of a posterior spinal or of a lateral sclerosis. In children the disease is frequently confounded with Friedreich's ataxia, and in all probability some of the hereditary cases have belonged really to this latter disease. The ataxia, the nystagmus, and the speech-defects occur in both. In Friedreich's ataxia the tremor is not volitional, and the movements are of a slower more inco-ordinate nature. Curvature of the spine and contractures are very much more common in hereditary ataxia, and the reflexes are usually absent, rarely increased. There are cases of chronic chorea in children which simulate multiple sclerosis.

Anomalous forms of the disease are common. Among the most puzzling cases are those of the *pseudo-sclérose en plaque*, described by Westphal, in which the volitional tremor, scanning speech, and spastic condition were present, but no lesions were found post-mortem. It is possible that some of these were cases of general paresis. Hysteria may simulate multiple sclerosis very closely. In a case regarded as hysteria during life Seguin found disseminated sclerosis.

The **prognosis** is unfavorable. There are long periods in which the progress is apparently arrested. The cases last for years, and finally become bedridden, and are carried off by an intercurrent affection or by invasion of the medulla.

Treatment.—Not one of the host of remedies which have been recommended appears to have any influence on the progress of the sclerosis, but, as in other chronic degenerative lesions of the cerebro-spinal system, practically no treatment is of the slightest service. Electricity may be tried. Attention should be paid to the general health, and iron and arsenic may be used if there be anæmia.

Other Forms of Sclerosis.

(1) Miliary Sclerosis, in which there are small grayish-red spots scattered over the cortex or at the line of junction of the white and gray matter. Sometimes there are multiple nodular projections on the cortex, consisting of firm, indurated tissue. So far as is known, no symptoms are produced by them.

(2) Diffuse Sclerosis, which may involve an entire hemisphere or a single lobe—the *lobar* sclerosis of the French—is a frequent condition in idiots and imbeciles, and cases are common in asylum practice. The cortical sclerosis may be extensive and unilateral, producing the condition known as unilateral atrophy of the brain, in which the lateral ventricle is much dilated. The symptoms in this condition depend very much upon the region affected. There may be considerable involvement without any paralysis or without mental impairment. As a rule, however, there is some degree of imbecility, and, when the motor region is involved, hemiplegia or diplegia.

(3) Tuberous or Hypertrophic Sclerosis is a remarkable form in

which there are upon the convolutions areas of an opaque white color, exceedingly firm and hard, but which do not necessarily disturb the symmetry of the gyri. The color is opaque-white, and on section they are extremely firm and hard.

Inflammation of the Brain, Suppurative Encephalitis, Abscess of Brain.

Etiology.—Abscess of the brain is rarely primary, but follows infection through a wound or extends from inflammation of a neighboring part, or the infection is carried by the blood from some distant part. Cases occur occasionally in which it is very difficult to assign a cause.

The conditions under which we most commonly meet with suppuration of the brain are—

(1) *Trauma*, particularly wounds of the head with fracture of the cranial bones. Of 50 Guy's Hospital cases analyzed by Pitt, 17 were secondary to injury or disease of the cranium. The fractures were all compound. Occasionally it happens that abscess follows uncomplicated bruising without apparent laceration of the skin or skull. In some of these instances the infection is conveyed through an unrecognized fracture at the base of the skull opening into the nose or ear. In other instances suppuration follows local disease, such as periostitis, nasal polypus, or tumor. In all of these cases meningitis is associated with the abscess.

(2) *Extension from Ear Disease.*—In Pitt's series, referred to, of 56 cases, in 18 the suppuration followed otitis media or mastoid disease. Of these cases, 9 occurred on each side. In two-thirds of the cases the abscess was in the temporo-sphenoidal lobe not far from the roof of the tympanum. The mode of origin of the abscess in ear-disease has been much discussed. On this point Pitt remarks: "Where there is healthy brain-tissue between the abscess and the bone, it is probable that the infection has been spread by the veins which empty into the superior petrosal sinus, from the temporo-sphenoidal lobe on the one hand and the tympanum on the other, by means of a septic phlebitis, or more probably by means of the perivascular lymphatics. Other veins run direct from the brain into the dura mater over the petrous bone. It is worthy of note, however, that in not a single case is there a statement that the superior petrosal sinus was thrombosed: it was certainly examined in many of the cases, though possibly not in all. If the mischief had usually spread by the veins, and not by the lymphatics, we should have expected thrombosis to have been occasionally noticed. Frequently the brain adheres to the anterior surface of the petrous bone, over which the dura mater is inflamed, and thence infection spreads by contact to the cerebral tissue."

(3) *Septic Processes.*—It may follow acute periostitis, occasionally abscess of the prostate, suppuration in the liver, and ulcerative endocarditis, in which disease multiple foci of suppuration are not very uncommon.

(4) In association with diseases of the lungs and pleura, more particularly gangrene, bronchiectasis, fœtid bronchitis, and occasionally empyema.

(5) And, lastly, abscess of the brain may follow the specific fevers. Bristowe has called attention to it as a sequence of influenza.

The largest number of cases occur between the twentieth and the fortieth year, and the condition is more frequent in men than in women.

Morbid Anatomy.—The cases following trauma are frequently associated with suppurative meningitis. The abscess may be solitary or multiple. In acute, rapidly-fatal cases after injury the suppuration is not limited, but in long-standing cases the abscess is enclosed in a definite capsule which may have a thickness of from 2 to 5 mm. The appearance of the pus varies with the age of the abscess. In acute cases the pus has a reddish-white appearance and is mixed with the débris of brain-matter. In the solitary encapsulated abscess the pus is usually of a creamy consistence, with a greenish tint and acid reaction, and may have an odor not unlike sulphuretted hydrogen. The brain-tissue about the abscess-cavity is usually infiltrated and œdematous. The size varies from that of a walnut to that of a large orange. Suppuration may occur in any district of the brain. It is most common in the white matter of the hemispheres. In cases following injury and in pyæmia the frontal lobe and the centrum ovale are most frequently involved: in those following otitis media and mastoid disease, the temporo-sphenoidal lobe and the cerebellum. In four-fifths of all cases the abscess is solitary. Various micro-organisms have been described in the pus of cerebral abscess. It may be mentioned, too, that the oïdium albicans and the actinomyces have occasionally been found.

Symptoms.—These vary greatly in character and duration. Following an operation or an injury, the symptoms may be those of an acute meningo-encephalitis, with rigors, fever, headache, vomiting, and delirium. When secondary to otitis, the irritative phenomena may be present for some days, such as restlessness, severe headache, and aggravated earache. Gradually, as the case proceeds, the mental processes become dulled, the patient grows drowsy, and the pulse slow; vomiting may be present, and optic neuritis is occasionally detected. The last symptom is not so frequent as in tumor. Such symptoms developing in a case of chronic otorrhœa are of extreme gravity.

In other instances the course is more chronic: there may be a latent period, with few or no symptoms, continuing for weeks or months, or in some instances prolonged for a year. During this time the patient may have no indication of cerebral disturbance. In other instances there are irritability, drowsiness, vomiting, headache, delirium, and convulsions. The patient may become forgetful or even aphasic, particularly when the disease is on the left side, and occasionally there is facial paralysis or paresis of both face and arm; or, after the persistence for months of indefinite brain symptoms, it is not infrequent to have a sudden outbreak of the most intense symptoms—headache, fever, vomiting, and gradually increasing coma.

Diagnosis.—In the acute cases following injury or operation one is rarely in doubt, particularly when there are severe headache, local tenderness, chills,

irregular fever, and perhaps convulsions. Cerebral symptoms following chronic otorrhœa, more particularly drowsiness, irregular fever, and chills, should always excite suspicion. In these cases it may be extremely difficult to determine whether the suppuration is really within the brain, subdural, or in the sinuses. The difficulties are not lessened by the knowledge that with otitis media and with mastoid suppuration there may be symptoms simulating meningitis, or even abscess, such as headache, fever, and double optic neuritis. In the long-standing cases the symptoms may resemble those of tumor of the brain, but fever is often present, and there may be rigors. It is to be remembered that in cases which last for eight or ten months the severe symptoms develop, just as happens sometimes in tumors, with great rapidity. The localization of the lesion is often uncertain. A large abscess may exist in a frontal lobe without causing any symptoms other than mental dulness. So also in the temporo-sphenoidal lobe, the most common seat, there may be no focalizing symptoms, but on the left side a large abscess is apt to compress the speech-centre and involve the motor areas for the face and arm. In the parieto-occipital region there may be hemianopia, but an abscess of considerable size may be present without any motor symptoms. When the Rolandic region is invaded there may be irritative symptoms, such as convulsions, or, when the centres are destroyed, paralysis. The lateral lobes of the cerebellum may be almost destroyed by abscess without causing special symptoms. When the middle lobe is involved there are inco-ordination and a staggering gait. The localizing symptoms in the basal ganglia and in the pons are very uncertain.

Treatment.—In cases following injury the trephine should be applied and the brain explored. Many recoveries have been reported. The most important group of cases is that which follows ear disease, and the objects to be aimed at in treatment are so clearly laid down by Pitt in his lectures, already referred to, that I give them *in extenso:*

"(*a*) In every case to improve the drainage of the ear by gouging away or trephining the mastoid sufficiently to open up the horizontal cells or antrum, where pus is often found, and to break a hole through the deeper part of the posterior wall of the external meatus, so as to allow no secretions to be retained. The cavity should be rendered sweet and aseptic as soon as possible. In a case of otitis media it is often desirable to carry out this treatment as soon as there is evidence of a fresh accession of severe mischief: should further exploration be necessary later on, the risk of infection from the septic otorrhœa will be very much reduced. It is always desirable that the external ear should be dressed apart from the other openings, if any are made.

"(*b*) To expose the anterior surface of the petrous bone, so as to allow free drainage for any pus or *débris* which may have formed in connection with the dura mater, which is often inflamed or gangrenous. This is best reached at a point half an inch above the anterior margin of the external meatus. Should there be any pus retained, some will often be found in the diplöe of the bone removed, in which case the bone should be broken away to a quarter of an

inch above and just in front of the meatus, so as to expose the most dependent part of the anterior surface.

"(*c*) To drain the abscess from below when possible. Messrs. Horsley, Macewen, Barker, Bergmann, and others have discussed the best methods of attaining this result. In the case of a temporo-sphenoidal abscess the area beneath which the pus will almost universally be found may be said to be bounded anteriorly and posteriorly by curved lines drawn through the temporo-maxillary joint and the middle of the mastoid, running at right angles to the sagittal suture, and to extend from half an inch to two inches above the meatus. The lower part of this area should, therefore, be explored with trocar and canula after breaking the bone away or trephining a fresh hole and opening the dura mater, unless special symptoms indicate that the abscess is higher up. If the attempt to find pus be unsuccessful, the lateral sinus should be exposed and examined half an inch directly behind the meatus; if necessary, the bone may be further broken away and the outer and under part of the cerebellum explored for abscess."

Tumors of the Brain.

New growths occur in the brain at all periods of life, and are frequent in childhood. The maximum number, however, is in the third decade. The chief varieties are as follows:

Tubercle.—This forms a single or multiple growth. A solitary tubercle is not infrequent, particularly in the cerebellum. It may reach the size of a walnut or even of a hen's egg. One-half of the cases occur in the first decade. The frequency is indicated by Starr's statistics of 299 cases of tumor in persons under twenty, of which 152 were tubercle. There may be groups of irregular tuberculous growths adherent to a greatly-thickened pia mater.

Syphiloma.—This is most common in the hemispheres and in the pons, and, like the tuberculous tumor, usually grows from the pia mater or develops on the arteries. Syphilomata are usually small and rarely reach the size of a walnut. They are often multiple. They occur much more frequently in the acquired than in congenital syphilis.

Glioma and Neuro-glioma.—These constitute, in reality, a variety of sarcoma developing from the elements of the neuroglia. They vary very greatly in appearance. Some of them grow slowly, are firm, hard, and difficult to distinguish from an area of sclerosis, altering very slightly the portion of brain involved; others grow more rapidly and are soft and highly vascular. While very many of them consist of a fibrillary network and branching cells, there are others, called neuro-gliomata by Klebs, which have enormous spindle-cells with single large nuclei, and others not unlike the large ganglion-cells. Some of these large spindle-cells undergo a remarkable vitreous transformation. The slow growth of certain gliomata is a point of great interest. Hughlings Jackson has reported an instance in which the symptoms persisted for more than ten years.

Sarcomata, round- and spindle-celled forms, are not infrequent, and con-

stitute the largest and most diffusely infiltrating of the cerebral tumors. They may develop in the substance of the brain itself, in the membranes, or invade from the bone.

Cancer is usually secondary to disease in other parts, and may be multiple. Primary cancer of the brain is rare. It, as well as the sarcoma growing from the membranes, may perforate the bone and appear externally, forming the so-called "fungus hæmatodes."

Cysts occur in all parts of the brain, particularly about the basal ganglia, where they result from changes in clots. Other forms occur between the membranes and the brain-substance. They may result from the breaking down of a tumor. Porencephalus follows congenital atrophy or hæmorrhage at birth, or may be due to a developmental defect. Dermatoid cysts have occasionally been found. Cystic disease of the choroid plexuses is extremely common, and, unfortunately, the word "hydatid" has been applied to them, but they must not be mistaken for the true hydatid or echinococcus cyst.

Parasitic Cysts are not infrequent, particularly in certain countries. Cysticerci may be numerous throughout the brain. When in the substance they do not attain a large size, but in the ventricles the bladder-like cysts may be as large as a walnut. The echinococcus cysts are most frequent in the hemispheres, and may attain a large size.

Other varieties of growths are met with, as fibrous tumors, which usually develop from the membranes; bony growths, which are not at all infrequent in the falx and tentorium; psammoma and cholesteatoma are occasionally met with; fatty tumors have been found on the corpus callosum; and, lastly, aneurisms, which have been elsewhere considered.

Symptoms.—New growths within the skull may produce symptoms by direct invasion and destruction of the brain-tissue, as in sarcomata, or by local pressure, as in the case of fibromata growing from the dura. Irritative effects are very frequently seen either inducing a meningitis or stimulating in a morbid manner the excitable region of the brain. Mechanical effects of tumors are also very important, acting, in the first place, by increasing materially the intracranial pressure. The effect may be more manifest at a distance than in the immediate vicinity of the growth. At the base of the brain and in the neighborhood of the third ventricle the tumors may interfere greatly with the circulation in the venæ Galeni, causing hydrocephalus. Large tumors may cause by their pressure marked atrophy of the cranial bones or even a condition of softening—craniotabes. The symptoms may be divided into two classes—general and focal.

(1) *General Symptoms.*—The most important of these are the following:

Headache, which may be diffuse or localized in one part. When very localized there may be a tenderness on pressure, and the slightest jar greatly increases the pain. Occasionally the pain radiates in the branches of the cervical nerves or there are severe neuralgic pains in the face. The greatest intensity of the pain may not correspond to the region of the tumor, and there are cases in which the tumor of the cerebellum has been associated with great pain in the

frontal region. Headaches of greater severity and persistence are met with in brain-tumor than in any other condition. In some instances there is only a dull, uneasy sensation, which scarcely amounts to pain except on exertion or on emotion. Though continuous as a rule, there may be paroxysms of much greater intensity which are sometimes nocturnal.

Optic neuritis occurs in four-fifths of all cases, usually double, but sometimes it is found only in one eye. It is independent of the size of the tumor, and in large growths which develop slowly it may not be present. On the other hand, it may occur with very small tumors, more particularly at the base. It is very important to bear in mind that a high grade of optic neuritis may exist for some time without special impairment of vision, so that the condition must be carefully sought for. As a rule, atrophy of the nerve supervenes, with great impairment of vision or total blindness.

Vomiting is a very common symptom, and occurs without nausea and without definite relation to the taking of food. It is more frequent in tumors about the central ganglia and the base. It is a very pronounced symptom in intracranial growths in children.

Convulsions are frequent, and are either general, resembling true epilepsy, or localized, the so-called Jacksonian fits. *Petit mal* is rarely present. Occasionally there is tetanic rigidity of certain groups of muscles.

Mental disturbance may be manifested in the dull, stupid condition of the patient, or he may act in an odd, unnatural manner. Occasionally there are hallucinations and delusions, which may be so marked that the patient is committed to an asylum. Occasionally symptoms resembling hysteria may develop. In the final stages coma is common. Vertigo is a very important symptom, often associated with the vomiting. It is most frequent in tumors at the base and of the cerebellum. It is particularly manifested as the patient rises from the recumbent posture. Fever is present in some rapidly-growing tumors, and the local temperature of the head may increase, and in many instances the thermometer has registered higher on the surface nearest the site of the tumor. A slow pulse is not infrequent, and sometimes there is irregularity. Toward the close Cheyne-Stokes breathing may be present.

(2) *Focal Symptoms.*—(*a*) The motor area. The symptoms may be either irritative or destructive in character. In the lower third of the motor region the irritative effects of a growth may be manifested in spasm localized to the muscles of the face or of the tongue. In the middle region containing the centres for the arm and hand the irritation may cause spasm in the fingers, in the limbs, in the muscles of the wrists, or in those of the shoulder; while in the upper third of the motor area the effects may be manifested in spasm beginning in the toes or the muscles of the foot or in those of the leg. With these there are usually important sensory disturbances, such as numbness and tingling or anæsthesia, which may be felt before the spasm occurs. Convulsive seizures localized in this way to certain groups and extending are known as Jacksonian spasms, and they are strictly comparable to those which may be induced by electrical stimulation of the cortex. In the study of the cases it is

of the utmost importance to determine the region first affected by the numbness, tingling, and spasm. Together, these constitute what Seguin has termed the signal symptom. The spasm is not necessarily accompanied by sensory disturbance. The effects of local irritation in the cortex radiate from the point of origin, involving successive groups of motor cells, and often inducing an orderly sequence of spasms in the muscles which they control; thus, an irritative lesion in the lower third of the motor area causes first spasm of the facial muscles, then, if it increase in intensity, of the centres above this point controlling the arm and hand, and may ultimately reach the centres higher in the convolutions which control the muscles of the leg. Following the spasm there may be anæsthesia and frequent inability to use the muscles which have been convulsed—paresis. In studying the localized convulsions from tumor three special points are to be observed: first, the starting-point of the spasm or of the preliminary sensory symptoms; second, the order of march of the spasm; and third, the condition of the parts after the spasm has passed, whether there be any paresis or anæsthesia.

Destructive lesions in the motor areas cause a paralysis which is often preceded by the localized convulsion. It is frequently monoplegic in type, affecting the face or face and arm together, more rarely the leg alone. The paralysis is usually slow and gradual in its onset. In large growths involving the internal capsule the hemiplegia may be complete and may be accompanied with hemianæsthesia. Tumors of the pons may cause paralysis of the arm and leg on one side and of the face on the opposite.

A not infrequent symptom in tumors situated in the motor area on the left side is asphasia from involvement of the third left frontal convolution. The tactile and muscular senses are also impaired in cortical lesions in the motor area, and should always be carefully tested.

(*b*) Prefrontal area. Tumors in this region may not present any localizing symptoms whatever. The general symptoms are usually well marked, and stupor and gradual impairment of the mental powers are not infrequent. On the orbital surface the olfactory bulb may be destroyed, producing loss of the sense of smell, and in many cases the growth of the tumor backward involves the motor centres and causes spasm or convulsion, or on the left side aphasia.

(*c*) In the parietal lobe tumors may attain some size without causing any local symptoms. Sensory changes have been noted in many cases, particularly paræsthesia and partial anæsthesia on the opposite side of the body. In the lower portion of the parietal region involvement of the angular and supramarginal gyri on the left side causes a form of sensory aphasia in which the patient is unable to recognize written or printed words. Another important localizing symptom in tumors which invade deeply the white matter of the parietal lobe is hemianopia, due to involvement of the visual tract.

(*d*) Tumors of the occipital lobe cause no motor disturbance, but produce hemianopia on the side opposite the lesion. More rarely the irritation of a new growth causes hallucinations of light or of sight, which are often followed by spasms or convulsions. Large growths in the left hemisphere may be

associated with word-blindness and a condition known as mind-blindness. Passing forward, the tumors may invade the internal capsule, causing hemiplegia and hemianæsthesia.

(*e*) In the temporal lobe tumors may reach a large size without causing any symptoms. In the left hemisphere invasion of the first and second gyri is associated with word-deafness, and not infrequently a condition of paraphasia in which words are misplaced. There are a few cases which indicate that involvement of the uncinate convolution and the hippocampus causes disturbance in the sensations of taste and smell.

(*f*) Tumors of the insula cause symptoms which are chiefly indirect; thus, involvement of the arteries as they pass over the convolutions is followed by softening in the motor area and mono- or hemiplegia. Paraphasia, in which words are misplaced, is a frequent symptom, due to interruption of the association tracts uniting the auditory perceptive centres and Broca's convolution. Owing to the close proximity of the internal capsule to the island of Reil hemiplegia is not infrequent from pressure.

(*g*) Basal ganglia. Limited growths in the nuclei of the corpus striatum do not necessarily cause symptoms. Tumors of the thalamus opticus are also, if small, latent in their growth, but when large they involve the fibres of the optic radiation and the internal capsule, causing hemianopia and hemianæsthesia. By far the most important symptoms of tumor in this region are those produced by invasion of the internal capsule which lies between these ganglia. The anterior part of the capsule may be invaded without symptoms. Destruction of the central portion causes hemiplegia, and of the posterior portion hemianæsthesia and hemianopia.

Tumors of the corpora quadrigemina are rarely limited, but involve the crura cerebri as well. Ocular symptoms are most frequent—loss of pupil reflex, nystagmus, and motor-ocular paralysis. There may be involvement of the third nerve as it passes through the crus, causing motor-oculi palsy on one side and hemiplegia on the other. Optic neuritis is an early symptom, and hydrocephalus from pressure very frequently occurs.

(*h*) Tumors of the pons and medulla. The symptoms are chiefly those of involvement of the nerves emerging from this region. In the pons the pyramidal tracts and nerves may be involved separately or together. Of 52 cases analyzed by Mary Putnam Jacobi, there were 13 in which the cranial nerves were involved alone, 13 in which the limbs were affected, and in 26 there were hemiplegia and involvement of the nerves: 22 of the latter had what is known as alternate paralysis; that is to say, involvement of the cranial nerves on one side and of the limbs on the opposite side of the body. A tumor growing in the lower region of the pons involves the sixth nerve, producing internal strabismus; the seventh nerve, causing facial paralysis; and the auditory nerve, causing deafness, sometimes with vertigo. Conjuate deviation of the eyes to the side opposite the facial paralysis also occurs.

Tumors of the medulla involve the cranial nerves alone, or cause a combination of hemiplegia with paralysis of these nerves. Irritative effects in the

territory of the ninth, tenth, and eleventh nerves are usually present, and there may be difficulty in swallowing, irregular action of the heart, irregular respiration, vomiting, and retraction of the head and neck. The unsteadiness of the gait is frequent, and there may be well-marked ataxia. Sensory symptoms, such as numbness and tingling, are present; toward the close convulsions may occur.

(*i*) Cerebellum. Extensive disease may exist in either hemisphere without causing symptoms. When the middle lobe is involved there are very characteristic features, of which the following are the most important:

Vertigo, which is more frequent in disease of the cerebellum than in any other part of the brain. The giddiness may be of a most distressing nature, and the patient may, on attempting to stand, complain of a sense of swimming in the head. It is frequently present with severe headache, and may be associated with nausea and vomiting. The semicircular canals are known to have their central relations in the cerebellum, and the giddiness is probably due to disturbance of the central mechanism of equilibration.

Headache is more common in tumors of the cerebellum than in other regions, and is usually, but not necessarily, occipital.

Cerebellar ataxia is characterized by an irregular staggering gait, the patient reeling to and fro like a drunken man. It is quite unlike, and cannot be confounded with, the gait of locomotor ataxia. When the growth invades the middle peduncle of the cerebellum, the tendency as a rule is to fall to the same side. Sometimes the patient falls forward, at other times backward.

Other less constant but suggestive symptoms are optic neuritis, neuralgic pains in the region of the neck and occiput, nystagmus, pressure symptoms on the medulla, progressive coma due to distension of the lateral ventricles, and lastly bilateral rigidity from pressure on the motor paths.

Diagnosis.—From the general symptoms the existence of a tumor can generally be determined. The combination of severe headache, optic neuritis, and vomiting, particularly if the latter be causeless, are especially significant. Neither in uræmia, lead-poisoning, nor anæmia—conditions in which we sometimes have optic neuritis—is the headache of such an agonizing character as it commonly is in brain-tumor. In these cases, when focal symptoms are absent, for a time doubt may exist, and I have known several instances of chronic Bright's disease in which the headache and the intense neuro-retinitis led at first to the suspicion of brain-tumor. The urinary and the cardio-vascular changes in these cases are, however, always pronounced.

The focal symptoms already referred to are of great value in determining the existence, as well as in defining the location, of the new growth. It must not be forgotten that focal symptoms, such as Jacksonian epilepsy, may occur with general paresis. The diagnosis from abscess of the brain has already been referred to. The syphilitic tumors are the most important of all to diagnose, as medicinal treatment is of such importance. Careful examination should be made for traces of old sores, and the patient should at least receive the benefit of any doubt.

Prognosis.—In a majority of cases this is unfavorable. Gummata are alone amenable to treatment. Tuberculous growths have been known to undergo calcification. The gliomata and fibromata may persist for years. Cases have been reported in which Jacksonian epilepsy has continued for from ten to fourteen years. The more rapidly-growing sarcomata prove fatal, as a rule, within from six to eighteen months. Death may be sudden, particularly in growths near the medulla: more frequently it is due to coma, in consequence of the gradual increase in the intracranial pressure.

Treatment.—(*a*) *Medical.*—Whenever a suspicion of syphilis exists, the iodide of potassium and mercury should be given, more particularly the former, in increasing doses. The results are sometimes most satisfactory. In tuberculous growths the chances of healing are very slight, though there are instances in which the symptoms have yielded. The headache is usually the symptom for which the patient seeks relief, and the ice-bag may be applied, or in the case of occipital headache the Paquelin cautery to the back of the neck. In the syphilitic cases the pain is often relieved promptly with the iodide. Chloral and cannabis Indica may be used, but in the severer forms morphine alone gives relief. The bromides are not of much service in relieving the symptoms of brain-tumor.

(*b*) *Surgical.*—The cases suitable for operation are limited in number. Some tumors are quite inaccessible, and in others which are accessible the invasion of adjacent parts contraindicates removal. The most satisfactory forms are those which grow from the membranes and only compress the brain-substance, as in the case reported by Keen. The impunity with which large sections of the calvarium can be removed and the cortex cerebri exposed warrants the exploratory operation in suitable cases.

Chronic Hydrocephalus.

Diagnosis.—A condition, congenital or acquired, in which there is a great accumulation of fluid in the ventricles of the brain, usually with enlargement of the head. An external hydrocephalus is described in which the fluid is in the arachnoid sac, but this is met with in cases of atrophy of the brain, the so-called *hydrocephalus ex vacuo*. In a few instances a sacculated exudation occurs, forming a meningeal cyst. In cases of extreme enlargement of the lateral ventricles the brain-substance may be so thinned at the cortex that the ventricular and arachnoid spaces communicate. Cases of true hydrocephalus may be divided into two groups—the congenital or infantile and the acquired.

(1) Congenital or Infantile Hydrocephalus.—The condition may develop in the fœtus and the enlarged head may obstruct labor. No reasonable explanation has been offered of its occurrence. Several children in succession have been known to be affected in the same family. Much more frequently nothing abnormal is noted at the time of birth, but gradually the head enlarges.

The **anatomical condition** is very striking. The ventricles, particularly the lateral, are enormously distended. The ependyma is clear, occasionally a

little thickened and granular; the veins are large; the choroidal plexuses are vascular, sometimes sclerotic, but often natural-looking. The third ventricle is enlarged; the aqueduct of Sylvius dilated and funnel-shaped, and the fourth ventricle may be, but is not always, distended. The fluid, which may reach several litres in amount, is limpid and contains traces of albumin and salt, sometimes urea and cholesterin. The cortex cerebri is stretched and thin. Over the Rolandic region there may be a layer of not more than 2 or 3 mm. in thickness, and all trace of sulci and convolutions is obliterated. The basal ganglia are compressed in the floor of the sac. The most striking feature in the appearance of the child is the great enlargement of the skull, which in a child of three or four years of age may reach twenty-five or even thirty inches in circumference, and looks enormous in proportion to the size of the face. The bones of the cranium are extremely thin, the sutures widen, and Wormian bones develop in them. The subcutaneous veins are usually large and well marked. The orbital plates of the frontal bone are depressed, causing exophthalmos, and the eyeballs cannot be completely covered by the lids. The fluctuation wave may sometimes be obtained, and Fisher's brain-murmur is often present. The child learns to walk late, and in extreme cases the legs become feeble and spastic. The reflexes are increased, and occasionally convulsions occur. The mental condition is variable: the child may be bright, but as a rule there is some grade of imbecility and the child learns to talk slowly. Nystagmus frequently develops, and in the congenital cases death usually occurs within the first four or five years. Occasionally the disease is arrested and the patient may reach adult life, as in the case of Cardinal, described by Bright, who lived to the age of twenty-nine, and whose head was translucent when the sun was shining upon it.

The **diagnosis** is rarely difficult. In moderate enlargement the disease may be confounded with the rickety head, which, however, is distinguished by the squarer outline, the flattened vertex, the absence of bulging of the fontanelles, and more particularly by the presence of other rickety manifestations.

(2) Acquired Hydrocephalus.—In the adult, distension of the ventricles is met with most commonly as a result of interference with the circulation in the straight sinus or in the venæ Galeni. In a majority of instances there is œdema at the base. In other instances the foramen of Magendie, by which the ventricles communicate with the spinal meninges, becomes closed, or the foramen of Monro is occluded, or the passage from the third to the fourth ventricle is closed by tumor. A rare cause is meningitis, particularly the epidemic cerebro-spinal form, after which hydrocephalus has been known to develop. There are other instances in which the inflammation is a meningo-ependymitis. In rare instances the hydrocephalus develops in the adults without any observable cause. The skull, as a rule, does not enlarge in the hydrocephalus of adults, though occasionally the sutures may separate and there is some increase in size. In the cases associated with tumor, even when the disease begins early in life, there may be no enlargement of the skull. In the

case of a girl aged sixteen, blind from her third year, the ventricles were enormously distended, owing to the presence of a tumor in the third ventricle. The head was not at all enlarged.

The **symptoms** of acquired hydrocephalus are very variable. Headache, attacks of somnolence, progressive optic neuritis leading to atrophy and blindness, have been frequently present.

The **diagnosis** is rarely possible. Gradually progressing optic neuritis without focalizing symptoms, severe headache, stupor, and attacks of somnolence, are suggestive symptoms. One patient, whose case I described, was unconscious for more than three months.

Treatment.—Medicines are useless in this condition. Gradual compression may be made by means of broad plasters applied so as to cross each other on the vertex, while others are made to encircle the head. Puncture of the distended ventricle has frequently been made, and when pressure symptoms are present this is a rational operation. The aspirator needle may be inserted at the outer angle of the anterior fontanelle. Only a few ounces should be removed at a time: convulsions and acute meningitis have been known to follow. Quincke recommends and has practised in acute as well as chronic hydrocephalus, puncture of the subarachnoid sac beween the third and fourth lumbar vertebræ. The spinal cord cannot of course be injured at this point, and the fluid can be removed more slowly and with much less danger of collapse.

SYPHILIS OF THE NERVOUS SYSTEM.

By HORATIO C. WOOD.

Syphilitic Disease of the Brain.

Etiology.—Cerebral syphilis is usually a late phenomenon, but may appear within three months after primary infection. I have myself seen it at every period from one to thirty years. It is especially liable to develop when the secondary symptoms have not been severe, and in common with other observers I have repeatedly seen it when both primary and secondary symptoms have been so slight as to escape observation on the part of the victim. Inherited syphilis is less prone to attack the nervous system than is acquired syphilis, but cerebral gummata may develop during intra-uterine life and at any time subsequently; indeed, nervous syphilis may develop after puberty as the first open outbreak of inherited disease.

Pathology.—The cerebral gumma probably always has its origin in the membranes, is usually surrounded by a reddish zone, and does not become so uniformly and completely caseous as the tubercle, from which it is further distinguished by its proneness to cause cerebral softening. In gummatous meningitis the exudation forms an extended, shapeless, gelatinous mass, which is in the majority of cases situated at the base of the brain. Microscopically, the cerebral gumma differs from other similar bodies only in the presence of very large, spider-like cells containing an exaggerated nucleus and a granular protoplasm, which extends into the multiple, branching, rigid prolongations.

Under treatment gummata may disappear completely or may leave behind them cicatrices, imperfect cysts, or even calcareous masses. A gumma may involve a blood-vessel, and, extending along its wall, give rise to a thrombus with secondary softening. A gummatous inflammation commencing in the pia mater may infiltrate a wide extent of the cortex.

Syphilitic atheroma of the cerebral vessels is not rare, and the arteries of the base are especially prone to suffer from a peculiar destructive specific lesion which renders them whitish, opaque, and hard, and finally almost obliterates their lumen.

Symptomatology.—Although *acute* or fulminating *syphilitic coma* may develop abruptly in the midst of apparent health, it probably is, in fact, always preceded by headache, vertigo, or other prodrome. The coma produced by the obliteration of the cerebral vessels is usually progressive, the true fulminating coma being commonly the outcome of gummatous inflammation. It may or may not be accompanied by delirium or convulsions. A patient of my own, about thirty years of age, became very drowsy one afternoon, and fell asleep. In a few minutes sleep changed to coma, interrupted an hour or so

later by violent delirium, alternating with furious convulsions. The coma may, however, be accompanied with complete muscular relaxation or in rare cases by local or hemiplegic paralysis. The pulse-rate may fall below the norm or may become rapid; the arterial tension may be high or low, and the pulse-wave large or small.

It is of vital importance to recognize that the symptoms of syphilitic coma are the same as those of congestion and inflammation of the brain from other causes, and that the first treatment in a serious case should be directed not so much to the specific disease as to the brain congestion which it has provoked.

The symptoms of *chronic brain syphilis* are so protean, so varying, that it is almost impossible to reduce them to any order. Possibly, the most dangerous cases are those in which the symptoms are least severe and so elusive that they fail to cause alarm. Malaise, a little brain failure, a succession of causeless headaches,—these may for a time be all the outcome. After a greater or less continuance of these prodromes epileptic attacks usually develop, with a hemiplegia or a monoplegia which is almost invariably incomplete and usually progressive; very frequently diplopia is manifested before the epilepsy, and on careful examination is found to be due to weakness of some of the ocular muscles. Not rarely oculo-motor palsy is an early and pronounced symptom, and a marked paralytic squint is very common. There is almost always distinct failure of the general health and progressive intellectual deterioration, as shown by loss of memory, failure of the power to fix the attention, mental bewilderment, morbid somnolence, perhaps aphasia, and toward the end of life not rarely dementia. If the case convalesce, the amelioration is gradual, the patient travelling slowly up the road he has come down. If the case end fatally, it is usually by a gradual sinking into complete paralysis, or the patient is carried off by an acute inflammatory exacerbation, or a very violent epileptic fit may produce a sudden fatal asphyxia. Death from brain-softening around the tumor is not infrequent, but a fatal apoplectic hæmorrhage is rare.

It is almost impossible satisfactorily to reduce to any order or types the various forms of cerebral syphilis. Besides those cases which resemble dementia paralytica, Heubner makes two types: (1) psychical disturbances, with epilepsy, incomplete paralysis (seldom of the cranial nerves), and a final comatose condition, usually of short duration; and (2) genuine apoplectic attacks with succeeding hemiplegia, in connection with peculiar somnolent conditions occurring in often-repeated episodes; frequently phenomena of unilateral irritation, and generally at the same time paralyses of the cerebral nerves.

The only conformity of meningeal syphilis, as I have seen it, with these types is in the fact that when epilepsy is pronounced the basal cranial nerves are not usually paralyzed; and it seems necessary to add two other types of disease—namely,

(3) Psychical disturbance without complete epileptic convulsions, associated with palsy of the basal nerves and often with partial hemiplegia.

(4) Paraplegia associated with ocular or other symptoms indicative of lesions at the base of the brain.

In nature, however, there are no distinct varieties of cerebral syphilis, all forms grading one into the other, and it is most satisfactory to study the important symptoms separately.

Headache is the most constant, and usually the earliest, of the symptoms of meningeal syphilis, but it may be entirely wanting. It may last for several years without the development of other distinct symptoms, and sometimes disappears when these appear. It has no fixed character, but is usually paroxysmal, and may occur solely in the form of very distinct and very violent paroxysms, accompanied by partial unconsciousness or other marked congestive symptoms. Distinct soreness of the head indicates disease of the skull or its periosteum.

Insomnia is a frequent prodrome of cerebral syphilis, but a peculiar *somnolence* is much more characteristic. The foudroyant coma has already been described: in the second variety of syphilitic stupor the symptoms develop gradually. The patient sits all day long or lies in bed in a state of semi-stupor, indifferent to everything, but capable of being aroused, answering questions slowly, imperfectly, and without complaint, but in an instant dropping off again into his quietude. In other cases the sufferer may still be able to work, but often falls asleep while at his tasks, and especially toward evening has an irresistible desire to slumber, which leads him to pass, it may be, half of his time in sleep. This state of partial sleep may precede that of the more continuous stupor or may pass off when an attack of hemiplegia seems to divert the symptoms. The mental phenomena in the more severe cases of somnolency are peculiar. The patient can be aroused—indeed, in many instances he exists in a state of torpor rather than of sleep; when stirred up he thinks with extreme slowness, and may appear to have a form of aphasia, yet at intervals he may be endowed with a peculiar automatic activity, especially at night. Getting out of bed; wandering aimlessly and seemingly without knowledge of where he is, and unable to find his own bed; passing his excretions in a corner of the room or in some other similar place, not because he is unable to control his bladder and bowels, but because he believes that he is in a proper place for such acts,—he seems a restless nocturnal automaton rather than a man. Apathy and indifference are the characteristics of the somnolent state, yet the patient will sometimes show excessive irritability when aroused, and will at other periods complain bitterly of pain in his head, or will groan as though suffering severely in the midst of his stupor—at a time, too, when he is not able to recognize the seat of the pain. I have seen a man with a vacant, apathetic face, almost complete aphasia, persistent heaviness and stupor, arouse himself when the stir in the ward told him that the attending physician was present, and come forward in a dazed, highly pathetic manner, by signs and broken utterances begging for something to relieve his head. Heubner speaks of cases in which the irritability was such that the patient fought vigorously when aroused—this I have not seen.

After some days of excessive somnolence and progressive deepening of the stupor, or sometimes more rapidly, the victim of cerebral syphilis may pass

into a condition of profound coma, out of which he cannot be aroused, and during which his fæces and urine are either not passed at all or are voided involuntarily. This condition of coma may end in death, but even when the symptom, seem most serious the patient may gradually recover, slowly emerging from coma into stupor, and from stupor into wakefulness and normal life.

Motor paralysis is very frequent in cerebral syphilis. It sometimes develops gradually, but it may appear suddenly, with or without the occurrence of an apoplectic or epileptic fit. When under these circumstances the paralysis is, on the first return to consciousness, complete, it is almost always due to a clot or thrombus.

The characteristic syphilitic palsy is progressive and incomplete. Any portion of the body may be involved, but the syphilitic exudation especially haunts the base of the brain, and a rapidly but not abruptly appearing strabismus, ptosis, dilated pupil, or any other paralytic eye-symptom not readily accounted for in the adult is, in the majority of cases, syphilitic. The specific palsy is often temporary, transient, and shifting. *Sensory palsies* are less frequent than motor palsies, but hemianæsthesia, localized anæsthesia, indeed any form of sensory paralysis, may occur. Special sense-palsies are sometimes present, whilst specific aphasia is common. It may be incomplete, transitory, and paroxysmal, but is more apt to be complete and to have permanency than are motor paralyses. Owing to the tendency of syphilis to produce multiple lesions, a lack of apparent agreement between the palsy and the aphasia is almost characteristic. Thus, Tanowsky found that out of 32 cases of syphilitic aphasia with hemiplegia, in 14 the paralysis was on the left side. Polyuria and true saccharine diabetes occur in cerebral syphilis, probably as the result of vaso-motor disturbances.

Epileptiform convulsions are a most characteristic symptom. A history of intense and protracted headache, followed by an epileptic fit, in an adult should excite the greatest suspicion. My experience is in accord with that of Fournier, that epileptiform convulsions not due to alcoholism or uræmia, and not appearing until after thirty years of age, are in nine cases out of ten specific. The aura is rarely present; the symptoms may be unilateral or even monoplegic, but any variety of epileptiform convulsions may be simulated. Furious attacks of local spasms also occur without loss of consciousness. Then, again, the movements may be continuous and distinctly choreic.

Psychical Symptoms.—Apathy, somnolence, loss of memory, and general mental failure are the most frequent and characteristic mental symptoms of meningeal syphilis; but almost any form of insanity—mania, melancholia, erotic mania, delirium of grandeur, etc.—may be of specific origin. Usually, sooner or later, distinct symptoms of organic lesion appear. Especially common is a loss of mental and physical power similar to that which occurs in dementia paralytica.

Diagnosis.—In the diagnosis of cerebral syphilis too much weight should not be attached to the history of the case, as non-syphilitic, organic brain dis-

ease may occur in persons who have had syphilis, and cerebral gummata may develop in persons who are unconscious of ever having been infected.

The prodromes of fondroyant cerebral syphilis are worthy of the most careful study on account of their diagnostic value and of their habitually being overlooked by the patients themselves. Persistent headache, slight failure of memory, unwonted slowness of speech, general lassitude, and lack of willingness to mental exertion, sleeplessness or excessive somnolence, attacks of momentary giddiness, vertiginous feelings when straining at stool, yelling or in any way disturbing the cerebral circulation, alteration of disposition,—any of these (and *a fortiori* several of them) occurring in a syphilitic subject should be the immediate signal for alarm. Of these varied possible prodromic symptoms the most important and characteristic, according to my experience, are headache and somnolence. Slight and shifting localized weaknesses sometimes precede an acute attack, but are more characteristic of the disease at a later stage. A momentary weakness of one arm; a slight drawing of the face, disappearing in a few hours; a temporary dragging of the toes; a partial aphasia which appears and reappears; a squint which to-morrow leaves no trace,—all or any of these may be due to a non-syphilitic brain tumor, to miliary cerebral aneurisms, or to some other non-specific affection; but in the majority of cases, when these phenomena occur repeatedly in a patient who is not suffering from hysteria, they are the result of syphilis.

In a doubtful case of sudden coma other ordinary causes must be eliminated: a pronounced rise of temperature or a pronounced conjugate deviation of the head and eyes tells strongly against, whilst decided ocular palsy or a partial paralysis of any character argues in favor of, a specific origin.

Headache occurring with palsy or with a history of attack of partial monoplegia or hemiplegia, vertigo, *petit mal*, epileptoid convulsions, disturbances of consciousness, attacks of unilateral or localized spasms, ocular palsies, epileptic forms of attacks occurring after thirty years of age, morbid somnolence,—any of these, even when existing alone, ought to be sufficient to put the practitioner upon his guard. Any apparent causelessness, severity, and persistency of headache should arouse suspicion, to be much increased by a tendency to nocturnal exacerbations or by the occurrence of mental disturbance or of giddiness at the crises of the paroxysms. Not rarely there are very early in these cases curious, almost indefinable, disturbances of cerebral functions which may be easily overlooked, such as temporary and partial failure of memory, word-stumbling, fleeting feelings of numbness or weakness, and alterations of disposition. In the absence of hysteria any indefinite and apparently disconnected series of nerve-accidents is of very urgent import. To use the words of Hughlings-Jackson: "A random association or a random succession of nervous symptoms is very strong warrant for a diagnosis of a syphilitic disease of the nervous system." Cerebral syphilis occurring in an hysterical subject may be readily overlooked until fatal mischief is done.

The *age* of the patient must also be taken into consideration. Apoplexy occurs most frequently in persons over fifty years of age, while congestive

syphilitic attacks are most common before that age. The course of a case for the first six or ten hours after the commencement of the acute paroxysm is sufficiently different in the two affections to be worthy of the closest study. A hemiplegic or embolic apoplexy which is sufficiently severe to keep up pronounced disturbance of consciousness for some hours is almost invariably accompanied by a complete hemiplegia, or more rarely by some other form of complete palsy; whilst in the syphilitic attack the paralysis is often absent, and probably never complete. Unless the clot has been a very large one, the return to consciousness after hemorrhagic apoplexy is usually much more rapid than it ordinarily is in syphilitic cases. Headache after an apoplexy is rare, whilst headache is very frequent after a severe syphilitic congestive attack.

The peculiarities in the symptoms of cerebral syphilis are chiefly due to the fact that the lesions are apt to be multiple or widespread; to be rapidly developed at an age when other organic diseases are rare; and to be situated in the cerebral cortex or at the base of the brain. Hence, multiple local or partial palsies are frequent, whilst the symptoms of the basal chronic meningitis in the non-tubercular adult are, in the majority of cases, the outcome of syphilis. Homonymous hemianopsia is very rare, because the occipital lobes are rarely invaded. Optic neuritis may occur in specific as in other organic brain diseases: it usually develops with moderate, but not excessive, rapidity.

The diagnosis of cerebral syphilis during life is always a matter of inference. When, however, the symptoms disappear under antisyphilitic treatment, for practical purposes the diagnosis may be considered as fixed. The therapeutic test is therefore a matter of the gravest importance. The old belief of syphilographers that tolerance of the iodides warrants the diagnosis of syphilis has been in recent times strongly combated, but I still think that, whilst it is not a positive sign, the tolerance strongly increases the probabilities of specific disease.

Prognosis.—Although death may occur during a syphilitic convulsion, yet the prognosis of an acute attack of cerebral congestion or inflammation due to syphilis is on the whole favorable, although it should be somewhat guarded. In chronic brain syphilis the prognosis is favorable for more or less complete recovery unless the symptoms indicate an absolute destruction of brain tissue. Whenever amendment of the symptoms occurs under antispecific medication, recovery becomes very probable. As, however, unexpected accidents occasionally happen, it is best not to make the prognosis too absolute.

Treatment.—The treatment of cerebral syphilis is best studied under two heads: first, the treatment of the accidents which occur in the course of the disease; second, the general treatment of the disease itself.

In the accidents of cerebral syphilis the treatment should be that which is adapted to the relief of the same symptoms when dependent upon other than specific causes. Thus, in foudroyant coma, if there be pronounced arterial excitement or if the patient's strength be good, venesection should be resorted to at once. I have seen life saved by the abstraction of nearly a quart of blood, whilst in other cases a few ounces suffice. Care must be exercised not

to mistake a simple epileptiform convulsion for a pronounced congestion of the brain, but if there be epileptic status with repeated convulsions, or if there be violent delirious excitement, venesection may be resorted to if the patient's general condition permit. In severe cases the bleeding should be as rapid as possible, and be continued until a distinct impression is made upon the pulse. When the heart's action continues violent after venesection, the hypodermic injection of the tincture of aconite-root (two to four drops) may be given every half hour until physiological effects are manifest. In feeble cases cupping to the back of the neck, stimulating injections, sinapisms to the extremities, cold to the head, croton oil as a derivative, and other classical remedial measures for brain congestion may be used.

In chronic cerebral syphilis remedial measures looking to the relief of symptoms may occasionally be employed with temporary advantage, but are of comparatively little importance.

The first therapeutic question to be decided is usually as to the choice between mercurials and the iodides. Cerebral gummata may develop in persons showing marked evidences of cachexia, but in the great majority of cases cerebral syphilis appears at a time when there is no general breaking down of the tissues or of the general system. The choice between the alternatives should rest upon the existing symptoms, and not upon the time which has elapsed between the primary infection and the outbreak. When cachexia contraindicates the free use of mercurials, or even of iodides, tincture of iron and corrosive sublimate may be given together, as in the following formula:

℞. Hydrarg. chlor. corrosiv., gr. iss;
Tr. ferri chloridi, ʒij;
Glycerinæ, ʒj;
Ol. caryophylli, ♏xviij;
Syrupi, q. s. ad ʒxviij.—M.
Sig. Teaspoonful in water after meals.

The slowness of the action of the iodides may be serious. In two cases I have seen death occur in an epileptic fit in patients who were rapidly improving under iodides. If mercury had been exhibited so soon as these cases came under care, the rapid removal of the lesions would have probably prevented the fatal fits. More and more has it become with me a favorite rule of action in cerebral syphilis, without evidences of cachexia or a history of recent mercurialization, to begin the treatment with mercury in such doses as are necessary to cause slight salivation, and to maintain a mercurial impression just below the line of slight tenderness of the gums for some days or weeks, *pro re nata*.

The method of administering mercury should be suited to the exigencies of the individual case. If mercurials by the mouth are well borne, they should be so administered. If the symptoms are extremely urgent, the mercury may be given both by the mouth and by inunctions. When there

is a tendency to diarrhœa the mercurial inunction should be used alone. I do not think that the oleate is preferable to the old blue ointment: a half drachm to three drachms of either may be used at once. An excellent plan is to give a hot bath late in the afternoon and use the inunction on going to bed, ordering the patient to rub the ointment on Sunday night into the left axilla; Monday night, into the left flank; Tuesday night, into the inside of the left thigh; Wednesday night, into the right axilla; Thursday night, into the right flank; Friday night, into the right thigh; Saturday night, into the region of the umbilicus; after this recommencing with the left axilla. In Europe the mercury is often given hypodermically, but I believe that the dangers of local inflammation overbalance any superiority of the plan; at least my own experience of hypodermic injections of mercury has been singularly unfortunate.

After a prolonged mercurial course iodide of potassium should always be given in order to secure elimination of the mercury as well as to relieve the syphilis. The dose of the iodide must be suited to the individual case. It is usually best to begin with 10 grains three times a day; in the course of two or three days this may be increased to 20 grains. Usually the patient who will tolerate a drachm of iodide a day will also tolerate two drachms a day. A majority of those persons who can take two drachms a day without the production of iodism can take three drachms. It is therefore safe to advance the dose very rapidly after it has been found that a drachm a day causes no inconvenience. Not rarely it seems almost impossible to produce iodism. I have frequently given the iodides up to or even beyond six drachms a day. I do not believe that larger amounts than these are of any especial service, and I am not sure that any advantage is gained by going beyond a daily dose of half an ounce.

The iodide is so soluble that a watery solution, one minim of which represents a grain of the salt, is readily made and is permanent. I have been accustomed to use the following formula, directing the patient to add to a dessert- or tablespoonful of No. 2 and a quarter tumbler of water the desired number of minims of No. 1:

℞. Potassii iodidi,	ʒj;	℞. Syr. sarsap. comp.,	fʒvj.
Aquæ,	fʒj.	S. No. 2.	
M. et ft. sol. S. No. 1.			

I am not sure that the abandonment by the profession of the use of the so-called "Woods" is right. I have seen "Zitmann's decoction" do good after the failure of the other forms of the iodides and mercurials. A fair imitation of the old "Woods" may be obtained by substituting for Number 2 of the formula just given a mixture of equal parts of the compound fluid extract and compound syrup of sarsaparilla.

Spinal Syphilis.

The **pathology** of the acute, explosive form of spinal syphilis, in which the symptoms resemble those of Landry's paralysis, is at present uncertain. It is not known whether the disease is centric or is a peripheral neuritis.

The second form of spinal syphilis is that in which softening of the cord occurs as the result of previous syphilitic disease of the blood-vessels. The third form is that in which syphilitic neoplasms develop. The fourth variety is that in which a gummatous infiltration occurs, commencing in the pia mater and spreading inward, involving the cord even into the gray matter, the first change being usually, if not always, thickening of the walls of the blood-vessels, with dilatation of the perivascular spaces and exudation of minute cells around the vessels. Heubner describes another variety of spinal syphilis in which there is found after death a condensation of the cellular tissues around the cord. This so-called *syphilitic callus* is probably not a primary syphilitic lesion, but the resultant of true gummatous inflammation.

Symptomatology.—Spinal softening and spinal neoplasms due to syphilis produce symptoms similar to those caused by similar lesions not due to syphilis. The symptoms of gummatous spinal meningitis are those of a localized sub-acute meningitis—namely, pain and spasm, with paralysis, affecting some peripheral part corresponding to the seat of the lesion. The pains are sometimes exceedingly severe, furious agonies shooting along the affected nerves or fulgurant crises simulating those of true locomotor ataxia. Often there is aching in the back. When this aching is accompanied by marked soreness on pressure or on jarring, the vertebræ themselves may be considered to be affected. Various paræsthesiæ, marked hyperæsthesia or anæsthesia, girdle pains, tonic spasms, localized tremors, grossly exaggerated reflexes,—such are the symptoms of irritation, which may be followed by complete paralysis with trophic changes.

The symptoms of diffused syphilitic infiltration of the cord vary with the seat of the lesion, simulating now locomotor ataxia, now spastic paraplegia, now chronic myelitis.

Diagnosis.—The recognition of the true nature of the spinal syphilis must depend upon the study of the collocation of the symptoms rather than of the individual symptoms themselves.

The lesions of syphilis are prone to be multiple, and are rarely as strictly confined to individual functional tracts as in sclerosis; consequently, the symptoms of syphilis of the cord are very apt to be mixed. Thus, there will be loss of co-ordination associated with retention of the patellar reflex; or the patellar reflex may be lost at a time when there is marked loss of power in the muscles rather than loss of their co-ordinating function; or an apparent locomotor ataxia will be associated with loss of power over the rectum or bladder; or a case which up to a certain point offers a typical outline of lateral sclerosis suffers from fulgurant pains or from paralysis of the sphincters.

Almost any conceivable mixture or interweaving of spinal symptoms may

occur as the result of syphilis of the cord, so that the most pathognomonic evidence of the existence of the disease is an atypical aggregation of symptoms. Whenever a contradictory mass of phenomena, evidently spinal in origin, present themselves before the practitioner, suspicion should at once be strongly aroused.

Prognosis.—The prognosis in spinal syphilis is less favorable than in syphilis of the brain. Frequently great improvement can be obtained by treatment, and alleged cures are not rare; but even in these so-called "cures" careful examination will usually reveal the existence of some permanent damage.

Treatment.—In the treatment of spinal syphilis the most urgent haste should be made by the free use of the mercurials to break down the gummatous exudation before it shall have produced secondary degeneration in the spinal cord. Only the most distinct cachexia justifies the beginning of the treatment with iodides. Absolute or partial rest should always be enforced during the treatment, whilst the hot and cold douche, massage, muscle-beaters, faradization, and other remedial measures and appliances may be used to keep up the circulation and nutrition of the affected muscles. These palliative measures are, however, of very little importance as contrasted with the antispecific medication. When the vertebræ are involved, immediate treatment by suspension should be resorted to, and the plaster jacket or one of its substitutes should be used.

Syphilis of the Peripheral Nerves.

Syphilitic Affections of the peripheral nerves are rare, but occur in three forms: first, *pressure neuritis*, including those cases in which the nerve-trunk is affected simply by pressure, the alterations not being in any proper sense specific; second, *secondary syphilitic infiltration*, including those cases in which the nerve-trunk is involved in a syphilitic deposit which has commenced in a neighboring organ and has secondarily infiltrated the nerve with gummatous tissue; third, *primary nerve-syphilis*, including those cases in which the lesion is distinctly specific and primary. Of these varieties of nerve-syphilis, only the last seems to need any pathological discussion here.

I have occasionally noted, in cases in which there was evident specific disease of the nerve-centres, a coincident tenderness of nerve-trunks, indicating that the latter were in a condition of inflammation, but have always been very doubtful as to whether such neuritis should be considered as due directly to the specific poison, or whether it were not simply a secondary inflammation propagated along the nerve-trunk irritated by a gumma somewhere in its course. A case published in the *Wien. med. Blätter* for 1886 by Dr. S. Erhmann makes it probable, however, that the syphilitic poison may act like the rheumatic, the alcoholic, the plumbic, and kindred poisons in producing widespread polyneuritis. Further observations seem, however, necessary before this conclusion can be considered as established.

Primary gummatous syphilis of those portions of the peripheral nerves

which lie outside of the bony cavities is extremely rare; the nerve-roots or trunks inside of the vertebræ and cranium are more frequently attacked; usually, however, in these cases neighboring larger gummata exist in the nerve-centres or in the membranes.

The first change is an infiltration of the wall of the blood-vessels in the nerves with minute cells. As the process continues the vessels become more and more enlarged and tortuous, and the infiltration forces itself a way through the trabeculæ of the nerve, whilst the nerve-bundles themselves gradually disappear, and often can be seen in various parts of the preparation undergoing degeneration. When the process is complete the blood-vessels themselves have been destroyed, and the position of the obliterated arteries may be seen, in the syphilitic product, occupied by spindle-form cells and the evidences of fibroid structure. The epineurium, or sheath of the nerve, is usually distended or spread out over the growth, but very rarely is it completely destroyed.

The **symptoms** which are produced by nerve-gumma are almost always intermingled with those which are due to implication of the nerve-centres, since it is extremely rare for nerve-gumma to exist as an early single lesion. Pain, spasm, and even paralysis are not infrequently the outcome of disease of the spinal nerve-roots, but it is very unusual for the disease of the nerve-roots to go so far as to entirely abolish their functions. Thus, pain of a most atrocious character following the distribution of their nerve-trunks is much more frequently seen than is anæsthesia, and very infrequently does a motor nerve suffer sufficiently for the production of distinct trophic changes in its tributary muscles. In my own experience the trigeminus nerve has been more frequently attacked than any other. I have seen the most atrocious *tic douloureux* as the sole symptom of a gummatous syphilis situated within the cranium.

The **treatment** of this form of nervous syphilis should be actively anti-syphilitic.

ORGANIC DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

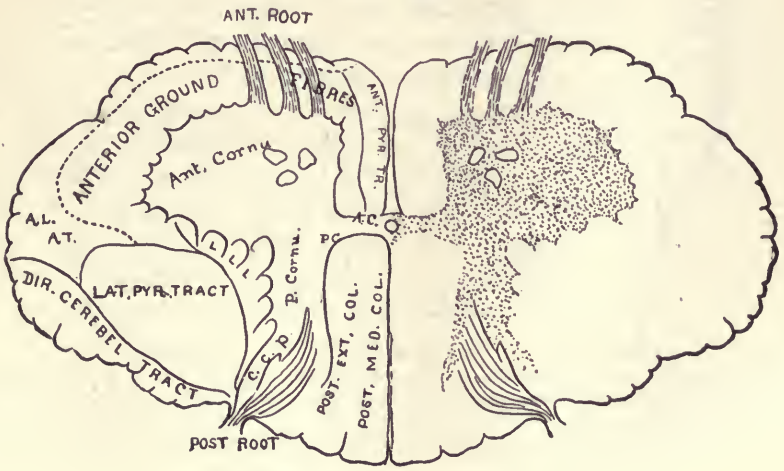
By HORATIO C. WOOD.

Spinal Localization.

When the practice of medicine has to be discussed within limits so narrow as those of the present volumes, detailed anatomical considerations are out of place: the student should be already furnished with anatomical and physiological knowledge. Nevertheless, for the purposes of review it does seem proper here briefly to point out by text and diagrams those anatomical and physiological facts concerning the spinal cord which have special relations to the localization of disease.

Localization of spinal disease is twofold: it relates disease, in the first place, to the anatomico-physiological tracts of the spinal cord; in the second place, to the vertical position of the lesion in the cord. The white matter of the spinal cord is divided into certain so-called longitudinal tracts or columns, which are set forth in the accompanying diagram.

Fig. 49.



Diagrammatic Section of Spinal Cord.

The Posterior Median Column, or the Column of Goll, lies immediately in contact with the posterior fissure of the cord. It is composed chiefly of fibres which enter through the posterior nerve-roots and pass upward. The increase in the size of the column of Goll from below upward does not seem to be sufficient for the accommodation of all of the fibres that enter the column, sup-

posing that these fibres continuously travel upward to the brain; further, the function of the column of Goll still remains in doubt; it is indeed probable that some or possibly all of the fibres escape from the column before reaching its summit, but how or where such escape is made still remains uncertain.

Next to the column of Goll lies the Postero-external Column, the Column of Burdach, or the Posterior Root-zone, chiefly composed of vertical fibres, whose function is at present unknown.

The Direct Cerebellar Tract seems to be chiefly composed of fibres which enter it through the lateral column from the gray substance and pass upward. It seems to have the function of conducting impulses upward, and, according to Flechsig, it probably carries impressions from the trunkal muscles.

Both the lateral pyramidal tract, "crossed cerebral tract," and the anterior pyramidal tract, "direct cerebral tract," or column of Turck, are composed of fibres whose course is downward from the pyramids of the medulla. At the decussation of the pyramids about three-quarters of the fibres cross over to form the lateral tract, whilst the remaining fourth of the fibres enter the cord without decussation and constitute the anterior tract. The fibres of both pyramidal tracts finally pass through the gray matter of the spinal cord into the

FIG. 50.

Diagrams of the Groups of Nerve-cells in the Anterior Cornu: Group I., inner or medial; A, anterior; A.L., antero-lateral; P.L., postero-lateral; I.L.P., intermediate lateral process; P.V.C., posterior vesicular column or tract. The two mid-cervical sections are only a few millimetres apart, and show how the anterior group, separate in the one, may be blended with the antero-lateral group in a neighboring part of the cord (Gowers).

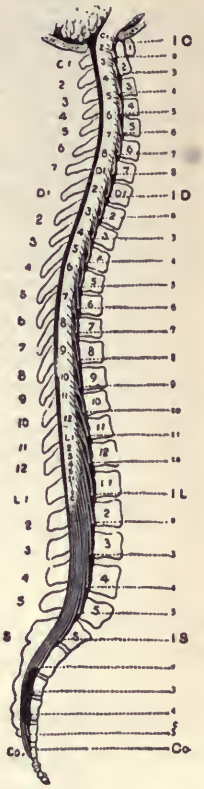
anterior cornua, and, although they have not been traced, almost certainly end in the processes of the motor cells. Their function is to conduct from above downward, and they probably are the pathway by which impulses from the motor regions of the cerebral cortex reach the motor ganglionic cells of the spinal cord, whose answering discharges provoke the final muscular contraction.

The remaining portion of the white matter of the spinal cord is composed,

first, of the so-called Antero-lateral Ascending Tract (A. L. A. T.), which forms the periphery of the anterior portion of the cord; second, the mass of white fibres marked in the diagram as the anterior ground-fibres. The function of these portions of the spinal cord have not been made out. Flechsig also anatomically separates the little patch of white matter between the lateral pyramidal tract and the gray matter, marked in the diagram L.L.L., and known as the lateral limiting layer. The separation of the antero-lateral tract from the so-called anterior ground-fibres does not seem to me at present warranted, either on anatomical or physiological grounds. Indeed, it is doubtful whether there is sufficient continuity, either of structure or of function, for any of the parts of the spinal cord spoken of in this paragraph to be considered as distinct columns or tracts; and I doubt whether any degeneration ever follows the course of these regions.

The gray matter of the spinal cord, besides the numerous conducting fibres which it contains, has situated in it ganglionic cells whose processes are prolonged into nerve-fibres composed solely of the axis-cylinders. The ganglionic cells are arranged in groups which vary in different portions of the cord, and probably in the same portion of the cord in different individuals. The most readily recognized of the groups are the small inner or medial group, situated in the inner anterior angle of the cornua; the large anterior group, placed near the anterior edge of the cornua, in the middle or a little to the outer side of the middle of the margin; the anterior lateral group, situated in the outer extremity of the front of the cornua (the last two groups frequently consolidate); the external or postero-lateral group, which is usually the largest and is extended in the posterior outer angle of the cord. The diagram (Fig. 50), taken from Gowers, shows the general arrangement of these groups in four different portions of the cord.

Fig. 51.



Anatomy of the Spinal Cord (after Gowers): C, cervical; D, dorsal; L, lumbar; S, sacral; Co, coccyx.

The most condensed statement of the facts necessary for the practitioner to know for the purpose of locating vertical lesions of the spinal cord that I am acquainted with, is contained in the table of Professor M. Allan Starr. By means of this table and the diagram of Professor Gowers (Fig. 51), both of which are here reproduced, the vertical position of almost any spinal lesion can be determined. In studying the diagram and table it must be remembered that the cervical cord is divided into eight segments, and that "II. and III. C." in the first column of the table refer to second and third cervical segments; that the dorsal cord is divided into twelve segments, "I. D.," and so on; that the lumbar cord is divided into five segments, and the sacral also into five segments; thus, "III. to V. S." means third to fifth sacral segments.

SEGMENT.	MUSCLES.	REFLEX.	SENSATION.
II. and III. C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infraspinatus.	Pupil. Fourth to seventh cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V. C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. Fifth cervical to first dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. C.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. Fifth to sixth cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. Sixth to eighth cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. Seventh to eighth cervical. Tapping anterior tendons causes flexion of wrist. Palmar; seventh cervical to first dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. C.	Flexors of wrist and fingers. Intrinsic muscles of hand.	. . . . .	Forearm and hand, inner half.
I. D.	Extensors of thumb. Intrinsic hand-muscles. Thenar and hypothenar eminences.	. . . . .	Forearm, inner half. Ulnar distribution to hand.
II. to XII. D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric, fourth to seventh dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal, seventh to eleventh dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.
I. L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric, first to third lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. L.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Striking tendon causes extension of leg.	Outer side of thigh.
III. L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.	. . . . .	Front and inner side of thigh.
IV. L.	Abductors of thigh. Abductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. Fourth to fifth lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.	. . . . .	Back of thigh, back of leg, and outer part of foot.
I. to II. S.	Flexors of ankle. Long flexor of toes. Peronei. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III. to V. S.	Perineal muscles.	Foot reflex. Achilles tendon. Over-extension of foot causes rapid flexion; ankle clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perineum. Genitals.

Hæmatorrhachis (Hæmorrhage into the Spinal Membranes).

Etiology.—By the bursting of aneurisms, tearing of spinal arteries by violence, etc. the spinal cord and its membranes may be involved in extraordinary outpourings of blood; whilst, on the other hand, minute hæmorrhages into the spinal membranes often occur as complications of various constitutional or local disorders and give no sign of their presence. Moderate hæmatorrachis may be produced by injury or acute blood disease; but, in the cases that I have seen, it has been the outcome of syphilitic degeneration of the blood-vessels.

In hæmatorrhachis the dominant symptom is an acute paralysis which takes the form of a paraplegia, abrupt and extremely rapid in its course, but still requiring some minutes or hours for its completion, during which time there is great pain in the back and extremities. The rate of the development of the paralysis varies according to the amount and rapidity of the hæmorrhage. The loss of power is due, not to an immediate lesion of the cord, but to the pressure of the exuded blood upon the cord, and especially upon the motor nerve-roots. Unless the blood be in great amount and thrown out with excessive rapidity, the paralysis grows more and more marked during several hours, and at the same time ascends higher and higher. As the blood creeps up the spinal cord or forces its way downward it tears the membranes away from the cord, presses, stretches, or perhaps tears the posterior as well as the anterior nerve-roots, and produces thereby muscular contractions and spasms with loss of muscular power, as well as shooting, tearing, or burning pains with more or less marked loss of sensibility. The anæsthesia is usually not as complete or as abrupt as in cases of intraspinal apoplexy. Nevertheless, if the clot be a large one the sensory palsy may be complete, and the zone between the anæsthetic and the sensitive portions may be very narrow. Consciousness and intellection are not affected except in rare cases, and then only in the first moments of attack. The bladder and rectum are very commonly paralyzed. Priapism or other evidences of genito-urinary irritation might naturally be expected, but I have never seen them.

The **prognosis** of hæmatorrhachis is very serious, most of the cases ending fatally. There is no specific **treatment**: in a robust subject, seen early, free venesection would be justifiable.

Acute Spinal Meningitis.

Definition.—An acute inflammation of the spinal membranes, not syphilitic.

It is usual to divide acute inflammation of the spinal membranes into acute *spinal pachymeningitis*, or inflammation of the dura mater, and acute *leptomeningitis*, or inflammation of the arachnoid or pia mater. Commonly, however, all of the membranes are affected, and, except as the result of injury or of septic poisoning, it seems doubtful whether acute spinal pachymeningitis exists; and even the septic and traumatic forms of the disease are almost always associated with inflammation of the other membranes. The diagnosis

of *acute pachymeningitis* is said to rest upon pain in the back, increased by movements of the trunk; cutaneous hyperæsthesia; tingling, or numbness in various parts of the surface of the body; paresis or paralysis of the lower extremities in severe cases; along with a history of vertebral disease or injury or of suppurative disease in the neighborhood of the spine.

Etiology.—Acute spinal meningitis, not dependent upon blood-poisoning, is an exceedingly rare disease, but is said to be occasionally produced by severe exposure to heat or cold. It occurs most frequently in young persons of the male sex.

Symptomatology.—Acute spinal meningitis usually commences with a distinct rigor, followed by high fever, arterial excitement, and other constitutional evidences of an active inflammation. At the same time, severe pain, affecting the whole extent of the spine, comes on, rapidly becomes very severe, and finally spreads throughout the body and the limbs. This pain is aggravated by movements of the trunk or by movements of the limbs, but is not usually associated with any distinct tenderness of the spinous processes. Evidences of motor irritation appear usually in a very few hours in the form of violent tonic spasms, most marked at first in the back, but soon involving the extremities. The contractions are usually severe enough to produce opisthotonos or other forced position of the trunk, and to set the limbs in rigid flexion. The pain produced by any attempt at motion gives an appearance of paralysis, but until late in the disorder, when the nerve-roots have been almost destroyed or the cord itself involved, there is little true loss of power. The reflexes are grossly exaggerated; retention of urine and constipation commonly develop early. Hyperæsthesia is in most cases an early symptom, but late in the disease may give way to or coexist with anæsthesic patches. Consciousness and intellection are not affected in the early stages, but there may be delirium or even coma before death.

Acute spinal meningitis may kill in two days, but, if the patient survives a week, is prone to end in recovery, with more or less permanent disablement from contractions and paralyses due to injuries of the spinal nerve-roots. The fatal result may be produced by the rise of temperature, which is often excessive, or may be due to paralysis of respiration or of deglutition.

Diagnosis.—The only disease with which spinal meningitis can readily be confounded is rheumatism. The rapid development of the symptoms, the universality of the pain and spasm, the widespread hyperæsthesia, the exaggeration of the reflexes, and the general severity of the attack, stigmatize the spinal disease.

Prognosis.—The prognosis should always be guarded in proportion to the severity of the symptoms.

Treatment.—The treatment of acute spinal meningitis should be most actively antiphlogistic. Free venesection, general or local; free purgation by means of calomel, followed by the rapid induction of ptyalism by mercurial inunction; the use of very active counter-irritation over the back,—these measures find their justification in the seriousness of the local disease, and in the fact

that inflammations of serous membranes are much more readily influenced by such treatment than are parenchymatous inflammations. Absolute quiet and rest should be enjoined. The food at first should be liquid, non-stimulating, and moderately nutritive, but afterward should be both nutritive and stimulating; and during convalescence the most absolute care must be enjoined to protect from any chilling of the surface or any fatigue of the nervous or muscular system.

Chronic Spinal Meningitis.

Definition.—A chronic inflammation of the spinal membranes.

Chronic spinal meningitis may occur as a local or as a widespread disorder. The most important local form is that originally described by Charcot, and commonly known as *cervical pachymeningitis*, an affection in which the membranes of the cervical spinal cord are found after death enormously thickened, compressing the cord and involving the nerve-roots.[1] Two stages of the disease are recognized: first, that of irritation; second, that of paralysis; but it must be remembered that the separation of these two stages is in fact artificial. Pain in the back of the neck, extending into the head and along the arms, associated with stiffness and muscular weakness of the parts and increased by movement, constitutes the chief symptom of the first stage. Vesicular or other trophic skin lesions due to inflammation of the nerve-roots are often present. The second or paralytic period is characterized by loss of muscular power, with muscular atrophy especially affecting the domain of the ulnar and median nerves, and followed by contractures which extend the hand and the forearm and flex the fingers into a claw-like position. The disease may finally ascend upward and downward, and give rise to widespread symptoms of chronic meningitis. Cervical pachymeningitis is probably, in a majority of cases, specific, and certainly should always be treated with mercury and the iodides until their lack of efficiency is demonstrated. Very severe repeated counter-irritation is also indicated. In the advanced disease, when the nerve-elements have suffered great change, no treatment is of avail.

Generalized chronic spinal meningitis is exceedingly rare, save as the outcome of syphilis, traumatism, or alcoholism. It is to be recognized by the slowness of its course and the existence of symptoms similar to those of acute meningitis, due to implication of the nerve-roots. Pain in the back and limbs, increased by active or passive movements; hyperæsthesia, perhaps associated with spots of anæsthesia; heightening of the reflexes, or in advanced stages loss of the reflexes; muscular contractions, followed by loss of power and wasting of the muscles,—these are the most important of the positive symptoms.

Treatment.—The treatment of chronic spinal meningitis should consist in the long-continued use of counter-irritants; in the administration of the mercurials and iodides; in absolute rest, associated with access to the open

[1] Chronic spinal pachymeningitis and chronic spinal leptomeningitis cannot be separated from one another, either clinically or anatomically.

air; and in the maintenance of health by all hygienic means. In all forms of meningitis opiates, antipyrin, and other analgesic remedies are to be used for the relief of pain.

Concussion of the Spinal Cord.

The spinal cord is liable to have its functions temporarily suspended by mechanical violence, being in this respect entirely similar to the brain. This sudden arrest of function may pass off in from a few minutes to a few hours, and leave behind it no trace of its former presence, or, if it has been accompanied by structural alterations, may be the starting-point for various chronic inflammations of the cord itself or of its membranes. The most absolute rest should of course be enjoined at the time and for some days subsequent to the injury, but there is no specific medicinal treatment. The term concussion of the spine is used by many authors as the name for the state which frequently follows injuries to the back—a condition which will be found treated of in a later section of this book under the heading of the "Remote Effects of Traumatism to the Nervous System."

Anæmia of the Spinal Cord.

Changes in functional activity are always associated with corresponding changes in the blood-supply of the organ, but the changed activity is commonly the cause of the changed vascularity, not the changed vascularity the cause of the changed activity. Nevertheless, the relations between function and local circulation have created a marked tendency in the professional mind to attribute all irregular, not readily explained functional disturbances to suppositious alterations of the local circulation. As an example of this may be cited the hysteroidal disease which is frequently spoken of as *spinal anæmia*, although there is not any proof that the alleged spinal anæmia exists, or that an existing anæmia would be capable of producing the symptoms present in the disease. It is a known physiological fact that excessive general hæmorrhage will occasionally produce partial paraplegia, which is recovered from when the blood-loss is made up; also, that partial paraplegia sometimes immediately follows excessive purgation; and a natural explanation of such paraplegias seems to be lack of food-supply for the motor-centres of the spinal cord. In profound anæmia slight tingling and numbness, with partial loss of muscular power in the legs and arms, is sometimes seen, but if during an anæmia a paralysis becomes complete, it is probably always hysterical or due to organic change.

The loss of functional activity of the spinal cord which follows hæmorrhage is to be treated by rest in bed, as free a diet as the digestive organs will assimilate, and the exhibition of iron, strychnine, and bitter tonics.

Hyperæmia of the Spinal Cord.

The plexus of veins which surround the spinal cord is so large that it is probably possible for the function of the cord to be interfered with by an

excess of blood in the extra-spinal venous circulation. In addition to this, analogy points out that the spinal cord must, like every other organ, be liable to acute hyperæmia with disturbance of function. There does not seem, however, at present to be any way of positively recognizing during life the condition of the circulation in the cord. It is probable that many of the symptoms of the condition known as neurasthenia are really the outcomes of passive congestion, and it is entirely possible that cases reported as instances of ascending paralysis have been really cases of congestion of the cord. I have certainly seen death under such circumstances, and found at the autopsy absolute fulness of the vessels with serous exudation. Again, I have seen cases in which there were rapidly developed, either without apparent cause or after excessive exertion with exposure, numbness, with marked loss of power in the limbs, with lessening of reflex activity, but without the symptoms going on to complete paralysis of motion or reflexes. That excessive functional activity and its consonant excessive congestion may result in pathological change is shown by those cases in which excessive coitus has been immediately followed by hæmorrhage into the cord; and probably in sexual exhaustion local weakness of the blood-vessels in the lumbar cord due to their frequent distension during the excessive coition is an influential factor.

In cases in which the symptoms point toward congestion of the spinal cord, if the patient be robust, it may be necessary to draw blood from the arm. In other cases wet and dry cupping along the vertebral column may be of value. As no evil results can come in these cases from moderate loss of blood, it is better for the physician to err on the side of too free bloodletting. This bloodletting, however, is only to be practised in the first few hours of the attack. Later, absolute rest on the side, *not on the back*, should be prescribed, and ergot should be given in large doses, the extract being preferred, as being the least apt of all the preparations to produce gastric disturbance. Ten grains of it may be administered every two hours, the dose after several days being reduced. In the very beginning of an attack hypodermic injections of the extract of ergot may be advantageously given, so as to make the first impression as rapidly as possible.

Spinal Apoplexy.

Definition.—Hæmorrhage into the spinal cord, not occurring as secondary to inflammatory or blood diseases, producing motor and sensory paralysis below the point of lesion.

Synonyms.—Hæmatomyelia; Intraspinal hæmorrhage.

Etiology.—Hæmorrhage into the spinal cord is not rarely found after death from tetanus, strychnic convulsions, and diseases or accidents which have produced rapid asphyxia. Such hæmorrhages are mere accidents of the case, and are spoken of as *accessory*. They are usually not to be recognized during life. Local inflammations, tumors, or other organic diseases of the intravertebral contents may give rise to spinal hæmorrhages which are spoken of as *secondary*. The name of "spinal apoplexy" should be reserved for

cases in which the hæmorrhage is acute in its attacks, sufficient in its amount to produce distinct symptoms, and not preceded by obvious organic disease, although, as in the case of cerebral apoplexy, the hæmorrhage is in a certain sense secondary, being probably always the result of previous disease of the coats of the blood-vessels.

The symptoms of hæmatomyelia develop with great suddenness, and may be attended with a primary brief loss of consciousness. Sometimes the subject falls at once, but a sharp, pricking pain is often first felt in one or both extremities, followed immediately or in the course of five or ten minutes by loss of power, which becomes complete in a very brief period. This loss of power is associated with disturbance of sensation, and usually the anæsthesia becomes complete almost as rapidly as the palsy. The motor paralysis at once occupies its whole territory, and does not extend upward. Pain in the extremities is very rarely at all violent, and never constant, though aching at the seat of hæmorrhage may be decided. The muscles are relaxed, with their electro-contractility undisturbed. The bladder and rectum become at once implicated. The knee-jerk and the cutaneous reflexes are at first not disturbed or, it may be, are a little increased.

There are cases on record in which the first symptoms have been confined to one foot, and in which several days have elapsed before the complete paralysis of the other foot. The clot, under these circumstances, must have been so exceedingly minute as not to involve by direct pressure, but only by secondary inflammation, the whole structure of the spinal cord. At first the extremities are free from vaso-motor disturbances, but subsequently the vaso-motor tone is often completely lost and the limbs become congested and cold.

Spinal apoplexy is apt to be followed by myelitic degeneration of the cord, under which circumstances the reflexes are extinguished and trophic changes rapidly develop, so that muscular atrophy, loss of electro-contractility, and decubitus are common symptoms to be seen a few days after a spinal hæmorrhage.

The **prognosis** in spinal apoplexy is absolutely grave, and no treatment is of distinct value. After the first hours great care should be taken to prevent the development of bedsores.

Spinal Embolism and Thrombosis.

Embolic and thrombotic arrests of circulation probably do occur in the spinal cord, but except as complications of specific or inflammatory disorders are among the rarest of clinical phenomena. Thrombosis would probably produce symptoms similar to those caused by hæmorrhage into the cord, but its diagnosis during life is impossible. The sudden occurrence in a case of valvular heart disease of paraplegia, followed by symptoms of softening and destruction of the cord (such as complete paralysis of motion and sensation, bedsores, and other trophic changes, paralysis of bladder and intestines), would justify the diagnosis of embolism.

White Softening of the Spinal Cord.

Arrest of circulation in the spinal cord must produce a true necrobiotic softening, in which the nerve-tissue would break down with hyperæmia and discoloration. White softening of the spinal cord does occur, and is thought to be commonly if not invariably the result of a myelitis. It is, however, somewhat difficult to perceive how white softening can represent a stage of inflammation, though it is very conceivable that it may be caused by inflammatory products or inflammatory tissue-changes interfering with the circulation in neighboring territory. Be this as it may, the explanation of Dr. C. L. Dana,[1] supported in one case at least by microscopic examination, that widespread degeneration of blood-vessel walls by interfering with blood-supply causes spinal white softening, has much of plausibility. The symptoms in a case of such softening would be simply those of progressive paralysis of all the spinal functions, without evidences of spinal irritation, and without fever or other constitutional disturbance save exhaustion.

Spinal Abscess.

Abscess of the spinal cord, excluding the minute focal collections of débris sometimes occurring in myelitis, probably exists only as the result of a septic meningitis or meningo-myelitis. Even in this form it is of great rarity, but may be suspected when rapid and severe spinal symptoms develop during a septicæmia.

The **prognosis** is of the gravest character, and there is no known treatment capable of at all modifying the course of the disease.

Spinal Tumors.

Non-malignant tumors of the spinal cord are only to be diagnosed by the symptoms which they produce through pressure upon the spinal cord and the nerve-roots. This pressure may be so extreme that the cord is reduced to a flattened band, and in many cases in which there is no great encroachment upon the spinal canal the transverse meylitis which is set up completely cuts off the function of the cord. The symptoms vary according to the size of the tumor, its position, its rapidity of growth, and its tendency to inflame nerve-roots and spinal tissue. These symptoms can best be studied as "cord-symptoms" and "root-symptoms."

The cord-symptoms are sharply limited—loss of motion and sensation, with heightening of the reflexes, and without trophic changes. If the tumor be of very slight growth, one side of the spinal cord may be pressed down so as to lose its functional power much earlier than the other part; indeed, one function of one-half of the cord may be primarily affected. In this way a sensory or motor paralysis or a sensory and motor monoplegia may result from a tumor.

The root-symptoms are most serious in those cases in which the growth has a tendency to set up inflammatory changes in the nerve-roots. Hence they are much more severe in cancerous than in other spinal tumors; indeed, the con-

[1] *Journ. Nerv. and Mental Diseases*, Sept., 1890.

dition known as spinal anæsthesia dolorosa is almost characteristic of cancer. The root-symptoms consist chiefly of sensory disturbance, loss of power, contractures and atrophy of the muscles, with change of the electro-muscular contractility.

The pain caused by a tumor may be slight or may be atrocious. It varies in character: sometimes it is constant, usually it is more or less paroxysmal; a steady, heavy localized ache deep in the back usually indicates involvement of vertebræ, the nerve-root pains being burning, lancinating, tearing, or "claw-like." Usually the pain follows the course of the nerves, shooting out to their utmost distribution and girdling the body in an agony. Unless the growth be so low down as to affect the cauda equina or be high up in the neck, the pains are felt chiefly, if not exclusively, in the trunk. When the changes in the nerve-roots have progressed far enough, complete anæsthesia may develop and yet the pain be in no way abated—*anæsthesia dolorosa.*

Diagnosis.—The diagnosis of compression of the spinal cord by a tumor rests upon the consentaneous development of sensory and motor paralysis, without trophic changes or loss of reflexes, and the abrupt limitation of this paralysis by a narrow zone of partial palsy. In other words, the connection between the cord and the brain is severed, and an abrupt line of motor and sensory paralysis marks the seat of the separation of conduction. Trophic changes can only occur in such a case as the result of a secondary lesion of the cord. The distinction between a tumor and the development of a transverse myelitis by spinal caries is to be made out by noting the slowness of development of the tumor and the lack of tenderness upon direct or indirect pressure upon the vertebræ. If the growth be very small and only slightly affect the cord, the diagnosis must be one of inference. Transverse myelitis does not produce a pain compared to that caused by an extending spinal tumor, but the cause of a transverse myelitis may often set up inflammation in the sensory nerve-roots, with its consequence—atrocious pain.

There is no known medicinal **treatment** for a non-specific spinal tumor.

Acute Ascending Paralysis.

Definition.—An acute disease, of uncertain pathology, characterized by the rapid spreading of a motor paralysis, commencing in the lower extremities, and in a very short time involving the whole muscular system; without trophic disturbance or alteration of the electro-excitability of the muscles, and with only minor sensory disturbances.

Synonym.—Landry's paralysis.

Etiology.—The causes of acute ascending paralysis are at present very obscure, unless, as seems probable, the disease is due to a micro-organism. The victims are usually persons in middle life, males more commonly than females. In some cases the attack has followed excessive exposure; still more frequently it has come on after one of the infectious diseases. A number of cases are reported in literature resembling acute ascending paralysis in which syphilis has been assigned as the cause, and in some of which recovery has been reached

by antisyphilitic treatment. These cases are, however, probably not instances of ascending paralysis, but of syphilitic myelitis or neuritis.

Symptomatology.—The first manifestation of an attack of Landry's disease is a sensation of numbness and weight in the feet, followed in the course of a few hours by a distinct loss of power. Sometimes this numbness is preceded by malaise, with paræsthesiæ for one or two days; in other cases it appears during apparently full health. The numbness rapidly mounts, whilst the loss of power in the legs becomes more and more perceptible, so that within a few hours, or at most a day, the power of standing is almost or altogether lost. Rapidly the symptoms continue to increase, the trunkal muscles becoming involved one after another, dyspnœa from paralysis of the diaphragm and respiratory muscles coming on, motion failing in the upper extremities, deglutition becoming difficult or impossible, the voice growing feeble and almost inarticulate or being in some cases entirely suppressed, and the patient dying, it may be, within two or three days from respiratory paralysis.

All of the muscles of the body and the extremities are thus paralyzed in this disease; but, probably because death usually occurs before the centres high up in the medulla or pons are reached by the ascending lesion, it is very rare for the muscles of the eyes to be affected, though strabismus and diplopia have been recorded. The brain-functions are not interfered with, intelligence and consciousness being preserved until the last. The sensory symptoms are always slight. There is no severe pain, only formication, numbness, and weariness. There may be a little blunting of the surface, so that a prick of a pin is not felt as readily as normal, but any pressure or distinct contact is recognized. In some cases sensation is somewhat delayed. The sphincters are usually not affected, there being no difficulty of urination or defecation, but the general rule in this respect is sometimes departed from late in the disease. The reflexes suffer with the motor paralysis, the knee-jerk being lost very early in the attack, whilst the cutaneous reflexes, although they may first escape, soon become impaired. In most of the recorded cases there has been no elevation of temperature, yet it may be that a slight initial rise has been in these instances overlooked, and in a few cases which have appeared to be true Landry's paralysis distinct elevation of temperature has been noted. Certainly, however, absence of marked fever is characteristic of the disease, and when there is pronounced febrile reaction the diagnosis must always be considered doubtful.

The muscles preserve intact until the last their normal electro-contractility, and never suffer any decrease in their bulk, whilst the complete freedom from decubitus or other evidences of trophic disturbance shows how completely the trophic centres escape. Since enlargement of the spleen was first noted by Westphal it has been found in a number of cases, and it is probably a constant symptom. Less constant, but probably present in the majority of cases, is enlargement of the lymphatic glands.

Some cases have been reported in which the course of the disease has not been ascending—in which, indeed, the medulla has been the first part of the cord to be attacked, so that speech, deglutition, and respiration have been pri-

marily affected. In the present uncertainty as to the real nature of ascending paralysis it seems doubtful, however, whether these cases should not be considered as instances of acute bulbar myelitis or of some other disease distinct from Landry's paralysis.

Acute ascending paralysis usually runs a rapid course, terminating in death in from forty-eight hours to a week. It is not, however, always fatal, and cases are reported in which several weeks have elapsed before an alleged Landry's paralysis has reached its maximum. The recovery may be brought about in two ways: sometimes the ascending lesion seems to stop at a point below the respiratory centres; in other cases the paralysis fails to be complete, and perhaps after the most alarming respiratory failure power is slowly regained. Occasionally recovery is rapid; usually, however, it is brought about by a slow reversal of the original course of the disease, the muscles first paralyzed being the last to recover function. The recovery may be complete, but literature contains numerous instances in which after a supposed Landry's paralysis chronic spinal lesion with trophic or spastic symptoms have gradually developed.

Pathology.—Various lesions, especially focal myelitic changes, slight meningitis, and alterations in the peripheral nerves, have been described as found in cases of Landry's paralysis. It seems certain, however, that these changes have been accidental and do not belong to the disease, since the concomitant results obtained by various highly skilful observers definitely prove that a typical ascending paralysis (with slight disturbances of sensibility, with immunity of the sphincters, without disturbances of the muscular contractility) may result in death, without producing either in the central nervous system or in the peripheral nerve-trunks any anatomical alterations that can be recognized by our present methods. It is true that sometimes, when no alteration of the spinal cord can be determined by means of the microscope, the existence of excessive venous congestion and increase in the cerebro-spinal liquid suggest that the pathology of the disease is an acute congestion of the spinal cord, but cases are not rare in which no such congestion can be made out.

Under these circumstances two theories of the disease naturally suggest themselves: first, that the lesion is an inflammatory congestion which leaves no trace detectable by our present method of investigation on account of the short time which elapses between the commencement of the disease and death; second, that Landry's paralysis is due to a toxæmia. The first of these theories is not plausible; the second seems probable. This probability is increased by the recent discoveries of the enlargement of the spleen and lymphatic glands, which further suggests that the affection belongs to the infectious diseases and is due to the presence of micro-organisms. In corroboration of this Baumgarten and Curschmann claim that they have found bacteria in the enlarged glands, but Westphal, Kahler and Pick, and others have looked for them in these places without success. Centanni[1] found in a typical case of Landry's paralysis a peculiar bacillus, which existed in moderate numbers in the spinal cord, but in great numbers in the peripheral nerves, where it formed colonies which had

[1] *Ziegler's Beitrage*, 1890.

resulted in structural alterations of the nerve-fibres, not of the nature of neuritis, but of a neuromycosis. This discovery of Centanni has been confirmed by Eisenlohr, who in two cases found a widespread—partially interstitial, partially parenchymatous—alteration of the peripheral nerves extending to the extreme end-filaments of the nerves, caused by the presence of various forms of micrococci; which micrococci also existed to some extent in the spinal cord, where they appeared to have set up an acute myelitic process.

If Landry's paralysis be a bacterial disease, analogy indicates that atypical aberrant cases will occur, and that in prolonged or even in very rapid cases the changes in the spinal cord may go beyond those ordinarily produced, since it is well recognized that the symptoms and the extent of the characteristic local lesions vary greatly in different cases of an infectious disease. May it not be that some of the cases of alleged myelitis have been instances of Landry's paralysis?

Diagnosis.—The combination of rapidly ascending paralysis with little disturbance of sensibility and loss of the reflexes, but without paralysis of the sphincters, trophic changes, or alterations of electro-muscular contractility, makes the recognition of Landry's paralysis usually easy. The enlargement of the spleen and lymphatic glands should always be looked for, and if neither exist, with our present knowledge the true nature of the disease must be considered doubtful. It seems probable that several poisons may induce similar spinal symptoms, and there is much plausibility in the supposition that those cases of Landry's paralysis that follow severe exposure may be of rheumatic origin. Evidently no enlargement of the spleen would be present in a rheumatic case, if such case really could exist.

In any individual case irregularity of the mode of attack should raise a suspicion; and if high fever, pain, exaggerated reflexes, or trophic changes develop, the attack must be considered to be one of organic disease, probably a central myelitis or a neuritis. In a peripheral neuritis there would probably be pain, and certainly tenderness, over the nerve-trunks, without enlargement of the spleen or lymphatics.

Prognosis.—The prognosis in Landry's paralysis is always very grave, but cessation of ascent or failure of the palsy to become complete would in any case give hope of arrest.

Treatment.—There is no known specific treatment. Absolute rest, with careful feeding, should be strictly carried out, and any symptom that may arise be met. I am not aware that the effect of early venesection has been carefully studied, but with the present probabilities of the bacterial nature of the disease local or general bloodletting, and even severe spinal counter-irritation, seem scarcely indicated. The free hypodermic use of extract of ergot for the purpose of diminishing spinal congestion may be justified. When there is any suspicion of rheumatic origin the salicylates should be administered with great freedom.

Acute Myelitis.

Definition.—An acute inflammatory affection, involving the whole thickness of a shorter or longer portion of the cord, characterized by paralysis of motion and of sensation, with trophic changes.

Etiology.—Acute myelitis occurs most frequently between the age of puberty and the fortieth year of life, and more often in men than in women. It may have its origin in traumatism, in compression of the spinal cord, and especially in the implication of the cord in the growth of thin inflamed tissue producing the pressure. It is asserted by authors to be sometimes due to excessive sexual excesses, especially unnatural coitus, and sometimes to be the outcome of excessive bodily exertion. A much more potential and positive cause of the disease is exposure, especially of the overheated body. Thorough wetting, sleeping on the snow or damp earth, etc. have in numerous instances been immediately followed by an acute myelitis. In winter campaigns it has been especially abundant, probably induced by the conjoint effects of violent emotional and physical excitement, with over-exertion and extraordinary exposure.

Acute myelitis has been noted as a complication of various acute exanthemata, diathetic and septic diseases, and it is said to occur with great frequency and severity among syphilitic patients, though the etiological value of syphilis is very questionable.

Pathology.—The macroscopic changes produced in the spinal cord by myelitis consist of alterations in color and consistency. Even whilst still in its membranes the cord feels to the fingers much softer than normal, or even fluid-like, and in extreme cases, when the meninges are opened, the whole inner mass escapes as a pultaceous fluid. If sufficient firmness remain, so that a section can be made, the surface of the section will be reddish, yellowish, or brownish, and seemingly structureless, no distinction existing between the gray and the white matter.

The situation and longitudinal extent of the lesion varies indefinitely, but the dorsal cord is especially prone to suffer. The transverse position of the softening also varies. In severe cases the whole thickness may be completely disorganized, but the gray matter is most universally and overwhelmingly attacked; hence the term *central myelitis*. Very often, instead of a single considerable territory being softened, foci are scattered through the cord (*insular* and *disseminated myelitis*). The variations in color chiefly depend upon the amount of blood in the part; ruptures and necroses of capillaries, and even larger blood-vessels, are inevitable, and hence occurs the exudation of altered blood, giving brownish or reddish tints, and also not infrequently the formation of small blood-clots (*hæmorrhagic myelitis*). The line between sound and diseased tissue is never abrupt, each focal change being surrounded by a zone of diseased tissue shading off into the normal cord. When life is prolonged and the acute disease merges into a chronic condition, the cellular or neurogliar tissue around the foci of inflammation undergoes a hyperplasia which

results in a pronounced sclerosis, and the focal débris becomes surrounded by a dense tissue, or in extreme cases the débris is finally absorbed and the sclerosed tissue more and more condensed until a thick-walled cyst remains.

Under the microscope all the nerve-elements are seen to have changed. The multipolar cells of the gray matter at times show multiplication of their nuclei: more commonly they are bloated, with their process broken, shrunken, irregularly enlarged, or in some way showing marked change of form. The cells become coarsely granular, or, losing all structure, are glass-like in their transparency. Vacuoles—*i. e.* spherical, transparent, seemingly empty, bubble-like spaces—can be seen in many of the cells. Whether they are present during life or are the results of post-mortem change seems uncertain. In all affected parts the nerve-filaments are found altered, swollen, almost broken up into strings of beads, the axis-cylinder being especially prone to increase in size when the structural alteration has gone into complete softening.

The nerve-elements are all more or less completely destroyed. There are left glistening, structureless remains of nerve-cells, bits of axis-cylinders or fatty degenerated sheaths, or altered nerve-filaments mixed with drops and masses of myeline, large granule-cells, altered blood-corpuscles, pigment-granules, and a mass of minute granules of unrecognizable origin. The walls of the blood-vessels are thickened, highly nucleated, and often filled with fatty granules, whilst the connective-tissue framework, if it remain, is swollen and softened by new cells and fatty changes.

The condition of the cord sometimes spoken of as *gray myelitis* represents an attempt at recovery, in which the escaped myelin and other results of disintegration have been absorbed, whilst the connective tissue has been increased and hardened into the beginning of a sclerosis. As already stated, this attempted reparation may result in the formation of a cyst or of a cicatrix, but the nerve-filaments have no power to undergo repair, so that restoration of function is impossible.

The process whose anatomical results have just been described is believed by the great mass of pathologists to be an inflammation, and in accordance with this view three stages are described: first, the stage of hyperæmia and commencing exudation (*red softening*); second, the stage of fatty degeneration and resorption (*yellow softening*); third, the terminal stage (formation of cicatrices or cysts, sclerosis, etc.).

The fact, however, that true suppuration probably never occurs in pure myelitis, but is only found when the meninges are involved, and the great rarity of post-mortems during the stage of simple hyperæmia, have led some writers to deny that myelitis is in truth really an inflammation; indeed, Spitzka affirms that, although authorities describe as anatomical alterations in the first stage capillary congestion and infiltration of the vascular area, the adventitia, and the neuroglia with granule-cells, yet he has never been able to find in literature a recorded case in which these things have actually been seen.

Symptomatology.—The course and symptoms of acute myelitis vary so

much within certain limits that it seems best to analyze them before speaking of the course of the disease.

In severe, rapid cases of myelitis the fever develops very early, and may throughout remain persistently high. Sometimes the febrile reaction occurs in paroxysms, and an excessive rise of temperature just before death is not uncommon. The fever may, however, even in fatal cases of myelitis, be entirely absent, and very commonly the temperature does not rise above 101° F., whilst a primary fever often disappears during the attack. The fever of the myelitis itself must be distinguished from the fever which in the later stages is not rarely produced by septic absorption from sloughing bedsores.

The spinal symptoms are those of irritation and those of paralysis. The symptoms of irritation, both motor and sensory, usually appear early in the attack, and are more or less completely lost within a short time, in some cases to reappear when partial convalescence develops. Twitching of the muscles, tonic or clonic contractures, and exaggeration of the reflexes may be present; in some cases any movement of the limbs produces violent, irregular muscular contractions. The symptoms of sensory irritation may be mild or severe. Tingling, numbness, violent formication, shooting pains, excessive distress during micturition and defecation, have been frequently noted, and even after a complete abolition of sensibility an agonizing anæsthesia dolorosa may remain. Sometimes the pain amounts to an intense agony—a burning girdle of molten iron, a thrusting of superheated needles through the limbs, a dragging or tearing of muscles from the flesh, etc. Pain in the back, with excessive sensitiveness over the spinous processes, especially to hot or cold applications, is not rare. True hyperæsthesia is not common, but very early in an attack a peculiar, diffused, painful vibrating sensation may occur when the part is touched (the *dysæsthesia* of Charcot). True sexual excitement is never present in myelitis, but painful priapism is not rare during the stage of irritation, and may last into the paralytic stage.

The symptoms of irritation usually very rapidly disappear, more or less completely, in those of paralysis, the motor paralysis becoming complete, the muscles being flaccid, and the limbs lying as though dead. The form of the palsy is usually paraplegic, but it follows the seat of the lesion and may become universal. The paralysis is accompanied with loss of the reflexes, the knee-jerk and the cutaneous reflexes disappearing entirely. In some cases in the lower part of the body the paralysis is complete and the reflexes absent, whilst higher up exaggeration of the reflexes shows that the stage of irritation is not yet past. The sphincters are almost always involved, and retention or incontinence of urine is often an early symptom.

The loss of sensation is complete, involving all forms of sensibility. Probably as the result of the involvement of nerve-centres presiding over secretion, the excretions rapidly become abnormal. Thus, even in two days the urine may become highly alkaline, bloody, muco-purulent, and loaded with the crystals of triple phosphates, whilst the perspiration is excessive, irregular, and altered in quality.

Vaso-motor palsy occasionally shows itself at first in a temporary rise of the temperature of the paralyzed limbs, but usually the extremities are cold and may be swollen by a diffuse œdematous exudation.

Muscular atrophy, with loss of faradic irritability and the development of the reactions of degeneration, appears very early. The trophic bedsore, *decubitus acutus*, which in severe cases may be unavoidable, usually attacks the sacro-gluteal region, but occasionally appears in the heels or other portions of the body. The first warning consists of one or several dark-red or violet erythematous patches, variable in extent and irregular in shape. Within twenty-four or forty-eight hours reddish or brownish vesicles or bullæ form in the central portions of the erythema. In rare cases, under careful management, the blebs wither and disappear without further symptoms; usually, however, the elevated epidermis is torn or drops off, leaving a bright-red surface with bluish or violet points or patches, and with swelling and sanguinolent infiltration of the surrounding tissue. Quickly the reddish surface becomes blackened, and a slough of variable extent forms. The whole buttock may thus melt down in the course of a few hours. Sometimes the process is arrested and the slough separates, but oftener the process continues, and, unless the patient die too quickly, the deeper muscles, with the nerve-trunks and arterial branches, are laid bare, and finally the bones themselves appear.

Distinct disturbance of vision is not common in acute myelitis, but contraction of the field of vision, amblyopia, or amaurosis due to optic neuritis have been noted, and in some cases the disturbance of vision has preceded the outbreak of more ordinary symptoms.

Acute myelitis varies indefinitely in the rapidity of its course, but three types may be recognized: the explosive, the acute, and the subacute, it being understood that in nature these grade one into the other.

The *foudroyant* or the *explosive myelitis* (*myelitis centralis*) commences abruptly, with disturbance of sensation, followed in a few minutes or hours by complete anæsthesia, motor paralysis, trophic changes, and abolition of reflexes. It is usually associated with more or less intense fever, delirium, coma, or convulsions. This central myelitis is often associated with hæmorrhage into the cord (*hæmatomyelitis*), when the paralysis becomes complete in a few minutes. In these cases death may occur without violent constitutional symptoms.

The *acute myelitis* runs a very rapid course, with or without fever, the paralysis becoming complete in from one to two weeks. Disturbances of the cerebration are not a necessary symptom of acute myelitis, but usually in rapid cases fever, headache, and delirium are present.

The *subacute myelitis* is that in which several weeks are required for the full development of the paralysis or in which the paralysis never becomes absolutely complete.

The *explosive myelitis* very frequently ends in death from paralytic asphyxia in a very few days, or septic absorption from decomposing urine and from sloughing bedsores brings about the fatal termination in two or three weeks. Acute myelitis usually ends in death from septic fever and exhaustion in a

few weeks or months, or occasionally may terminate in an imperfect recovery. Subacute myelitis may end in death, but very commonly passes into a condition of chronic myelitis, in which mild trophic symptoms and partial paraplegia may exist for years; or it may end in an imperfect recovery with atrophies and paralysis in groups of muscles. In very mild cases recovery may occur after a prolonged convalescence.

Diagnosis.—The difficulties which hang about the diagnosis of myelitis are best discussed by considering the different forms separately. The only diseases with which an explosive myelitis can be confounded are Landry's paralysis and hæmorrhage into the spinal cord. The myelitis, however, is usually distinguished by the existence of decided fever; by the pronounced disturbance of sensation; by the early paralysis of the bladder, and especially by the early coming on of muscular atrophy, with the reactions of degeneration; and by the diffuse œdema, the sloughing bedsores, and other trophic alterations. Hæmorrhagic myelitis so closely resembles hæmatomyelia that no less an authority than Spitzka denies the existence of the latter condition; and when headache, fatal delirium, and other constitutional symptoms are absent, it may not be possible at first to make out the myelitis, which must, however, soon be revealed by the occurrence of trophic changes.

Acute myelitis may be confounded with certain forms of poliomyelitis, but the latter lack the pains and the superficial trophic changes in the skin which occur in myelitis. From a peripheral neuritis the myelitis is to be distinguished by the intensity of its paralytic phenomena, by the rapidity of the development of the muscular atrophy and other trophic changes, and by the absence of tenderness over the nerve-trunks.

In subacute myelitis the trophic changes often occur slowly or are altogether absent. Such a case, however, lacks the nerve-trunk tenderness and the excessive pain of peripheral neuritis.

Treatment.—The treatment usually advised in acute myelitis is founded upon the theory that the lesion is inflammatory and capable of arrest by antiphlogistic measures. Under this view of the case, if the patient be seen in the onset the most active antiphlogistic treatment is justifiable. If it be possible to arrest so serious a local inflammation, the fear of producing a general exhaustion should have little consideration. Unfortunately, however, there seems to be no weighty clinical evidence that the most severe venesection, use of cold, or other antiphlogistic measures, have distinct influence upon the disease. Nevertheless, if the general constitutional condition be good, blood may be drawn from the arm, and active local bloodletting, by means of leeches or dry cups, is usually advocated by authorities. Ergot is commonly employed for the purpose of diminishing congestion, and, although our knowledge of the actual value of the drug is imperfect, its harmlessness and the possibility of usefulness warrant its free administration. It may be given in the beginning hypodermically in the form of the extract, and afterward the extract may be administered in doses of ten to fifteen grains every three hours, until disturb-

ance of the stomach, ergotic coldness of the surface, or the continuing progress of the disease indicates its withdrawal.

The production of diaphoresis by the use of the hot bath or hot pack is especially recommended by Erb in cases in which the premonitory signs of myelitis make their appearance after exposure to cold, etc.; but I cannot believe that these measures really avail anything, though they may in various cases have relieved rheumatic pains and general muscular soreness following exposure, which had been supposed to be precursors of myelitis. During an attack the warm bath, however, does appear to be grateful to patients and to render them more comfortable, and should always be tried. In employing it absolute precautions must be taken that the patient himself make no effort whatever, a sufficient staff of nurses to readily lift him being provided. The temperature of the bath should be in the beginning 90°, to be increased later if it be found advisable. The duration of the bath should at first be about ten minutes, but it should be rapidly increased almost indefinitely, according as it is found to agree with the individual case. The bath may be given once, twice, or three times in the twenty-four hours as seems wisest.

The free use of mercurials has been largely advocated, usually on theoretic grounds. Certainly, grave doubts surround the advisability of mercurialization, and if ptyalism be produced at all it should be done with great caution. There is not the slightest reason for supposing that belladonna, derivation to the intestines, or the production of diuresis by means of the ingestion of large quantities of alkaline waters, as recommended by Erb, are of any service whatever. Of course if excretion fails from want of nerve-influence, care should be exercised to see that the emunctories are kept active. Strychnine has been recommended by high authorities, whilst other practitioners (myself among them) have found it to do injury. If the generally held views concerning the nature of the disease and the action of strychnine be correct, injury rather than good is to be expected from its use.

A question which always requires very careful consideration is as to the use of local applications to the spine. The application to the spine of ice contained in a long thin rubber bag possibly may be of service, and probably is not injurious. Counter-irritation by means of the actual cautery or the blister has been largely practised, and finds much commendation by some writers. The grave danger, however, of precipitating ulcers and widespread gangrene attends the use of remedies of this class, and certainly no counter-irritants should be applied to the skin which is already distinctly anæsthetic or to a part which may be exposed to continuous pressure. Spitzka, on theoretic grounds, believes that counter-irritation applied to the lower legs and feet is of much more service than are the same measures applied to the back. The use of the galvanic current, as occasionally practised, seems to be an outcome of a childish credulity.

From what has been already said it will be seen that the value of drugs in myelitis, save only for the relief of symptoms, must be at present considered problematic.

The nursing during acute spinal inflammation is of the utmost importance. So soon as there is any reason to suspect the commencing of a myelitis absolute rest in bed should be prescribed, and, so far as possible, the patient should be prevented from moving a single muscle of the body, the feeding, the making of the personal toilet, etc. all being done by an attendant. This absolute abstinence from muscular movement applies not only to the precursory stage, but is even more important when the symptoms of convalescence are developing. Under these latter circumstances any muscular activity may produce a relapse. In those fortunate cases in which the patient recovers the avoidance of fatigue should be strictly enjoined for one or two years after the attack. What is true of muscular movements during convalescence is even more true concerning the sexual functions.

Various authorities lay stress upon the influence of the dorsal decubitus in increasing congestion, and consequently inflammation, in the spinal cord, and although it seems to me that this injunction is based upon a supersensitive theorism, it may possibly be correct, and the patient should therefore be kept as much as is convenient upon the side, or, according to some, even upon the face. If the patient can be made comfortable in the ventral position, it has the advantage of removing from pressure those portions of the body most prone to development of gangrenous lesions. Such lesions constitute one of the most serious complications of myelitis, and are therefore to be guarded against by keeping the surfaces perfectly dry, by preventing pressure, and especially by putting the patient on a water-bed, which should be covered with one or more heavy woollen blankets so as to avoid any chilling of the body. If bedsores appear, they must be treated according to the ordinary method, irritating applications being, at least in the early stages, avoided, and antisepsis carried out as thoroughly as may be.

In the very beginning of the case it is necessary to pay the strictest attention to the condition of the bladder, as urinary retention and its consequent cystitis and pyelitis are so frequent and so fatal. It is probably better in most cases to use continuous catheterization, along with washing out of the bladder once in twenty-four hours with an antiseptic solution. It is always best to use the soft, flexible rubber catheter, and in continuous catheterization this is imperative. The catheter may be retained by adhesive strips, but the plan suggested by Spitzka of using a perforated condom fixed to the catheter and then fastened to the inguinal region is preferable. To the catheter should always be attached a soft-rubber tube ending in some sort of urinal. The condition of the bowels must always be closely attended to, mild laxatives being employed, and aided, if necessary, by occasional stimulating injections.

Chronic Myelitis.

Definition.—Chronic inflammation of the spinal cord, occupying more or less thoroughly the whole transverse section of a greater or less length of the cord, and presenting various disturbances of the spinal function.

Etiology.—Chronic myelitis may originate in an acute myelitis or may

be especially chronic from the beginning. The cause of it as an original disease appear to be traumatism, exposures to cold, sexual excess, and syphilis; in a word, those causes which when present in more active form or affecting more susceptible individuals produce acute myelitis. It is also believed by many clinicians that certain diseases of the circulation, as well as propagation of irritation from peripheral nerves and irritating diseases, may produce chronic myelitis. Certainly there seems to be some relation between long-continued and severe hæmorrhoids and the disease.

Pathology.—The macroscopic alterations of the spinal tissue vary in different cases of chronic myelitis, and it is probable that several really diverse affections are confounded under the one name. In the majority of cases the chief alteration is sclerosis or hardening, the substance of the cord being denser and firmer than normal, presenting a smooth section which is often grayish or yellowish gray, constituting one of the conditions which has been called *gray degeneration* of the cord. The membranes usually suffer, showing after death thickening or opacity or other evidences of chronic inflammation. The nerve-roots also are very frequently atrophic.

In the sclerotic tissue the neuroglia is increased in amount and density. It contains an abundance of neuroglia cells, many of them enlarged and furnished with proliferated nuclei and numerous processes (the so-called Dieter's cells), but it is especially composed of wavy, fibrillated bundles of fibres. The nerve-fibres are swollen, often irregularly so, with sheaths and axis-cylinders abundantly and irregularly enlarged, or are atrophied, with destruction at first of the medullary sheath and afterward of the naked axis-cylinder. The ganglia-cells are variously altered, clouded and swollen, or more frequently atrophied, shrunken, indurated, strongly pigmented, or finally changed into irregular, unrecognizable structures. Amongst the nerve-elements can usually be seen fat-granule-cells, corpora amylacea, and pigment-granules, whilst the walls of all the blood-vessels are enormously thickened and the perivascular spaces crowded with cells and exudate.

In one form of chronic myelitis the whole body of the cord is filled with moderately large neuroglia cells, pressing upon and destroying the nerve-elements, but showing little or no tendency to the formation of fibres. I have seen this form of change usually in the cords of persons who have had very distinct syphilitic history.

Symptomatology.—When chronic myelitis develops as a primary affection, it usually comes on very insidiously and with marked fluctuations. Slight sensory disturbances, paræsthesiæ, partial anæsthesiæ, girdle sensation, loss of endurance, especially in walking, and uncertainty of gait may develop so slowly and with so many remissions that the subject scarcely knows when to date the beginning of his disorder. Constipation, loss of sexual power, and vesical weakness may be amongst the earliest symptoms.

In fully-developed chronic myelitis the symptoms resemble those of the acute disease, but the depression of function usually predominates over irritation. Hence violent pains and muscular spasms are not usual, although very

frequently the legs draw up, or attacks of vibratile contractures in the legs occur at night. Nevertheless, although the greatest complaint is usually of loss of power, examination will almost invariably show evidences of irritation of the spinal cord until very late in the disorder. Thus the muscles are usually stiff rather than relaxed, and occasionally when the patient can still walk the spastic gait of lateral sclerosis is present in a moderate degree. The reflexes are, in the early stages of the disease, almost invariably exaggerated. Often this exaggeration is very marked, the slightest touch upon the patella tendon, tickling of the soles of the feet, or even stroking of the thighs, provoking not only local muscular contractions, but also general widespread movement. Ankle clonus may be present. In the later stages of the affection the reflexes may be diminished or even entirely lost, but this rarely happens until the gray matter of the cord is disorganized, so that it is almost invariably associated with atrophy of the muscles or other trophic change which belongs to the last stages of the disease.

The sphincters are prone to be involved, and vesical weakness or paralysis, with retention or dribbling of the urine, is almost universal, and is liable to produce a paralytic cystitis, which in turn may creep up the ureters and involve first the pelvis and then the secreting structure of the kidneys, and end in a fatal renal degeneration.

The **course** of chronic myelitis is very slow and prolonged. The effect upon the general health of the patient is in most cases remarkably slight, and the bodily nutrition may be well preserved at a time when the lower portions of the spinal cord are hopelessly degenerated. Death may be produced by intercurrent diseases, by trophic lesions, by renal contraction, or in some cases by the sheer exhaustion of prolonged suffering and confinement.

Diagnosis.—The diagnosis in chronic myelitis depends upon the rate of development of the case and the universal disturbance of all the functions of the cord. Much more slow in its oncoming than the acute myelitis, it usually lacks the years required for the development of true sclerosis.

Prognosis.—The prognosis of chronic myelitis should always be very guarded, as recovery is rare; but not infrequently an arrest of the disease may be secured.

Treatment.—There is no reason for supposing that drugs have much influence over the progress of chronic myelitis. Nitrate of silver, at one time much used, has never in my experience accomplished anything. The effects of mercurial treatment or of the use of iodide of potassium are scarcely, if at all, better marked, although I think it often useful to administer continuously corrosive sublimate in the dose of one-sixtieth to one-ninetieth of a grain three times a day for its tonic as well as alterative influence.

Counter-irritation is sometimes useful, but to be of any value must be severe. The best form is the application of the actual cautery, made over a considerable extent of the affected cord with such light touches as only to destroy the epidermis. If the part be frozen before applying the cautery, the pain of the application is trifling, whilst the after-pain is usually not so severe

as that of a blister. Frequent light applications of the cautery are of more service than severe applications at longer intervals. The Paquelin cautery has seemed to me the best form, and may be applied every ten days or as often as the part heals. Hot baths or hot packs at short intervals are often of service, and afford an explanation of the reputation that certain thermal mineral waters have acquired.

The hygienic treatment is exceedingly important, and by change of air, careful selection of diet, and all other means the general health should be improved as much as possible. Mental depression, over-exertion, and fatigue are to be sedulously avoided, and as favorable a view of the case as possible should be given to the patient. Rest on the bed or couch is often of the greatest service, and when conjoined with daily use of massage may be maintained for a length of time without endangering the general health or producing muscular relaxation. When circumstances favor it the patient may with great advantage spend a large portion of his time on the bed, couch, or lounge in the open air.

Acute Poliomyelitis.

Definition.—An acute disease dependent upon inflammation or degeneration of the ganglionic cells in the anterior cornua of the spinal cord, characterized by paralysis, with complete relaxation, rapid atrophy, and alterations of the electrical reactions in the affected muscles.

Synonyms.—Acute anterior poliomyelitis; Infantile paralysis; Essential paralysis of childhood; Acute atrophic paralysis.

Etiology.—Acute poliomyelitis is essentially a disease of childhood, although it does occur during adult life. It may appear in the first month of infancy, and about five-sixths of the cases are developed in children under ten years of age. It is indeed often claimed to be of intra-uterine development, but the correctness of this is doubtful. It attacks males more frequently than females. So rarely is it possible to trace hereditary influence that it appears not to occur with abnormal frequency in neuropathic families. As was first shown by Wharton Sinkler, at least in the climate of Philadelphia, it comes on more frequently during the summer than the winter months. The attacks have in so many cases followed immediately upon exposure that it is impossible to escape the conviction that the exposure had been the exciting cause. The same is true of over-exertion, especially over-walking, in very young children. Traumatisms appear occasionally to afford the initial point. Dentition is frequently assigned as a cause, and certainly poliomyelitis has in numerous instances been a secondary result of acute exanthematous diseases or of some local acute inflammation with high fever.

The explanation of the frequency of the disease in childhood, and of the variability of the exciting causes, seems to me not far to seek. The spinal structure involved is trophic in its functions, and during childhood has not only to maintain the nutrition of the muscles already developed, as it does in adult life, but also to preside over growth and development in these muscles.

The functional activity in these parts must therefore be excessive during childhood, and must be attended with a constant hyperæmia and excitement, which make the part liable to be thrown over the line of health by any transient irritation.

Pathology.—The one lesion which has always been found in modern autopsies in cases of essential infantile paralysis has been degeneration of the multiple ganglionic cells in the anterior cornua of the gray matter of the spinal cord. Death is so rare in the early stages of the disease that there are few records of post-mortems occurring in other than the fourth stage. In one case reported by Dr. Drummond, in which death resulted after a few hours of illness, the ganglionic cells were granular and swollen—a condition which probably represented the incipient stage of poliomyelitis. The next change in the cells seems to be an increase in the density of the granulation, with pigmentation: this is followed by disappearance of the processes and shrinking of the bodies of the cells until they become irregular masses whose true nature is scarcely recognizable. Finally, the cells disappear, so that no traces of them are usually found in old cases. The cells are attacked in foci, ranging from a hundredth of an inch to more than an inch in length. All the cells in a focus may be affected, or the destruction may be limited to certain groups in the anterior, posterior, or other part of the focus. The lesion of the cells is so constant, and is physiologically so closely related to the symptoms seen during life, that there can be no doubt as to its being the cause of these symptoms.

Two theories have been and are still to some extent in vogue as to the nature of the lesions in infantile paralysis: one attributes the changes to a primary idiopathic atrophy of the ganglionic cells; the other teaches that the cells are not affected primarily and apart from the other gray matter, but are involved in a limited central and focal myelitis.

It would seem established that in some sections of the spinal cord in recent cases of infantile paralysis the tissue surrounding the cells appears normal, but I do not know of any case in which this condition has prevailed through the whole length of the affected region, and certainly evidences of hyperæmia and myelitic changes in the gray matter about the cells have been very pronounced in most of the early autopsies. In the case reported by Dr. Drummond intense capillary congestion, with minute extravasations of blood and swelling of the neurogliar elements, were evident in the gray matter, and in various cases a little more advanced than these the investigator has found the blood-vessels dilated, with their lymphatic sheaths infiltrated with leucocytes or surrounded by minute extravasations of blood; the neurogliar tissue swollen, granular, containing large round granular cells; the myelin of the nerve-tubes broken; and indeed not rarely such general disintegration as to cause minute patches of red softening. Our present knowledge trends in favor of the theory that not only the motor ganglionic cells, but also the surrounding tissues, suffer in poliomyelitis.

In old cases of poliomyelitis the atrophy of the gray matter is usually

accompanied by changes in the anterior nerve-roots and in the antero-lateral columns of the cord. The normal nerve-tubes are wasted, stripped of their myelin, often without their sheaths, and are surrounded by hyperplastic neurogliar tissue. Often the parts are infiltrated with amyloid corpuscles, and sometimes the original focal lesion is surrounded by embryonic neurogliar cells, looking as though an attempt had been made to isolate it.

It is not probable that these changes are due primarily and directly to the original poliomyelitis, for in no recent cases have lesions of the white columns been observed. It is therefore most probable that these widespread spinal lesions are either trophic or due to a propagation of the inflammation by physiological or anatomical continuity of structure. The microscopic changes seen in the nerve-roots resemble the degenerative atrophy that follows section of the peripheral nerve. Probably in a majority of cases the nerve-trunks themselves undergo change. As was shown by Leyden, this change may consist of a degenerative atrophy or of a neuritis. The discovery of the frequency of nerve-trunkal disease has given rise to the theory that neuritis is the cause of infantile paralysis. There can be no doubt that many of the symptoms of poliomyelitis may be produced by a peripheral neuritis. It is also known that certain metallic poisons, like lead and arsenic, are capable of originating either a neuritis or a poliomyelitis or a combination of the two diseases. It is therefore probable that in some cases of disease, which we call natural because we are unable to discern the cause, the poliomyelitis exists alone; in others neuritis exists by itself; whilst in others, again, both affections are consentaneously developed.

Symptomatology.—The onset of acute poliomyelitis is almost invariably sudden, usually occurring in the midst of apparently robust health; indeed, so rare is any history of a preceding nervous disturbance that such disturbance must be considered as accidental rather than as prodromic. The attack may be without constitutional symptoms, the child perchance waking after a good night's rest paralyzed, or even with apoplectic abruptness developing weakness in the daytime. More frequently there is a primary fever which is in most cases of moderate intensity, although the temperature may rise to 104° F. The duration of the fever varies greatly: sometimes it continues but a few hours, but it may persist three or four weeks. The same variability is characteristic of the cerebral disturbance: apathy grades in the series of cases into stupor, and this into coma, whilst restlessness or isolated spasms pass into convulsive twitchings, and these into the fiercest of general convulsions. A most important practical fact is that there is not a constant relation between the severity of the constitutional disturbance and the extent or depth of the subsequent palsy.

The sensory disturbance is habitually moderate, but pains in the back and limbs are often complained of, and may be intense. Anæsthesia and hyperæsthesia are so rare that their existence should waken a doubt as to the correctness of the diagnosis. Vomiting may be absent, or may be so intractable as to suggest that the case is one of gastritis. The fever rarely lingers long after the development of the palsy, and may disappear with an abrupt,

crisis-like defervescence. In the majority of cases the paralysis is complete before it is recognized, but, although its coming on must be very rapid, I believe the extreme suddenness of its discovery is often due to its having been overlooked. Certainly in a number of cases a progressive paresis, increasing for from a few hours to several days, has been noted, and still more often the paralysis, already complete in one limb, has under observation spread to other parts. During the period of acute constitutional disturbance there is often incontinence, or more rarely retention, of the urine, but true permanent paralysis of the bladder never occurs.

The situation and extent of the paralysis vary almost indefinitely. Nearly the whole muscular system may be so involved that a true general paralysis results and the child be unable to move hand or foot. The face seems, however, to be practically exempt, permanent paralysis of the facial or ocular muscles due to an acute poliomyelitis being, if it ever occurs, one of the rarest of nervous phenomena. The same is true of the intercostal muscles and of the diaphragm. The reason of this exemption is not known, but to it in great part is due the fact that the disease is so rarely fatal.

The subsidence of the constitutional disorder and the development of the paralysis are followed by a period of quiescence, which after from one to six weeks is succeeded by a peculiar, almost pathognomonic, regression of the paralytic symptoms. The extent of this regression varies so much that there is little relation between the final result and the amount of original paralysis. The improvement occasionally ends in complete recovery, but in the majority of cases after from two to three months spontaneous amelioration ceases and some of the muscles settle into permanent paralysis.

During the second period of the disease—*i. e.* that of widespread or general paralysis—the affected muscles are in a condition of extreme relaxation, with complete loss of the reflexes, and in a very short time a high grade of rapidly progressive atrophy manifests itself, especially pronounced in those muscles which are to remain paralyzed, and almost from the first accompanied by trophic changes similar to those which follow division of a nerve. The first change is probably *modal;* that is, the muscle simply responds more slowly to galvanic currents than it normally does. Very soon, however, qualitative as well as quantitative changes appear. In order to detect these changes the current must be brought in direct contact with the muscles, for if the electrode be applied to the nerve-trunk, it will be found that the electrical reaction is diminished in quantity, but not altered in quality. If the negative pole (*cathode*) of a weak battery be placed over a normal muscle, but not over its motor point, a strong contraction occurs at the closure of the circuit; when, however, the positive pole (*anode*) is placed over the normal muscle, the contraction is much less: in neither case is there any contraction when the circuit is broken: in other words, with the normal muscle and a feeble current we obtain good cathodal closing contraction, slight anodal closing contraction, and no motion whatever at either cathodal or anodal opening. When a current of sufficient power is used, opening contractions are produced and the anodal contraction is greater than

the cathodal. The "reaction of degeneration" consists merely in a more or less perfect reversal of the above formula. The anodal (positive pole) closure then causes a stronger contraction than the cathodal (negative pole) closure. When there is only a slight degree of degeneration present there is a correspondingly slight increase of anodal closing over cathodal closing contraction. A minimum degeneration would be indicated by an equality of the two closing contractions.

These alterations in the electrical reactions of a degenerating muscle are readily formulated, and in this way perhaps will be more readily grasped by the student. The symbols are as follows: An Cl C represents anodal closing contraction; An O C represents anodal opening contraction; Ca Cl C represents cathodal closing contraction; Ca O C represents cathodal opening contraction: $<$ represents is less than; $>$ represents is more than (the point of the $<$ being toward the lesser quantity).

Then the formulas are—

An Cl C $<$ Ca Cl C
An O C $>$ Ca O C } muscle normal.

An Cl C $=$ Ca Cl C
An O C $=$ Ca O C } muscle in first stage of degeneration.

An Cl C $>$ Ca Cl C
An O C $<$ Ca O C } muscle in more advanced stage of degeneration.

After the reaction of degeneration (D R of some authors) has been established, if the muscle continue to undergo change, the galvanic irritability slowly diminishes, stronger and stronger currents being required to produce an effect. When a certain stage is reached all reactions cease save a feeble An Cl C, and at last this is lost and the muscle does not respond at all. When recovery occurs the electrical reactions of the muscle pass upward along the pathway they have descended.[1]

The distribution of the permanent paralysis varies indefinitely, but monoplegias are much more common than bilateral symmetrical paralysis. A more or less complete crural paraplegia is indeed often seen, but paraplegia cervicalis, or paralysis of both upper extremities, is so rare that its existence has been denied. Even when a bilateral or symmetrical paralysis occurs, it

[1] The diagnostic importance of the reaction of degeneration is greatly lessened by the circumstance that its demonstration on the person of a terrified or enraged struggling child usually requires much skill and patience, and that it probably is never present when a muscle retains its integrity as regards the faradic current. For the purposes of diagnosis the failure of response to the rapidly-interrupted faradic current is usually a sufficient test of the condition of a muscle. When a muscle loses its power of responding to the rapidly-interrupted faradic current in a week or ten days after the occurrence of paralysis, whether the reaction of degeneration can or cannot be satisfaciorily demonstrated, the inference is positive that trophic changes are taking place in the muscle. If a few days later such muscle is unable to respond to any faradic current, this inference becomes a certainty. For the purpose of prognosis the study of the reaction of degeneration may be necessary, but it will, according to my experience, often be found disappointing.

can usually be made out that the paralysis is really a multiple palsy—that is, is due to the separate implication of various centres—because it will be noted that in each involved limb certain groups of muscles escape altogether or in part, and that there is no close correspondence between the affected groups in opposite sides of the body.

Crossed palsies and hemiplegias are infrequent as a result of poliomyelitis, and, like paraplegia, are to be looked upon as formed out of a number of multiple palsies. Indeed, the paralysis of poliomyelitis is a paralysis of muscle-groups, and the selection of the grouping seems to depend not so much upon the proximity of the muscles in the limbs as upon their being habitually used together in the activities of normal life.

In the description of the symptoms of poliomyelitis I have followed the ordinary division into four stages: first, that of constitutional disturbance; second, that of general paralysis with quiescent symptoms; third, that of regression; fourth, that of the permanent paralysis. This fourth stage is, however, not a portion of the disease, but a condition which has resulted from the disease. It is the wreck left by the storm. The permanent paralysis has no direct tendency to shorten life, the disablement being confined to those organs which are connected with locomotion, the digestion, the general nutrition, and the sexual functions remaining intact. The affected limb is limp or rigid, often bluish in color (always, if the paralysis be entirely complete, habitually cold), and losing its heat with the greatest rapidity upon any exposure. The electrical reaction of the muscles, as well as the atrophy, varies with the original lesions. When this is complete the muscles waste to a fibrous band, incapable of responding to any electrical current. Other structures of the limbs also suffer. The growth of the bones is retarded, so that in the growing child gradually the arm, the leg, the hand, or the foot, as the case may be, becomes shorter as well as smaller than its fellows.

The interference with the bone development is not always in direct proportion to the atrophy of the muscles; indeed, the growth may be permanently arrested, although the paralysis entirely disappears. Relaxation of the joints, due probably in part to lengthening of the tendons, caused by the limbs dragging upon them whilst unassisted by their natural allies, the muscles, becomes more and more pronounced as the child grows older, until at last the head of the bone may be entirely out of its socket. Even during the most acute stage of poliomyelitis bedsores are unknown, and in the chronic after-condition there are never trophic inflammations or destructive lesions of the skin.

Various deformities arise, not simply from failure of development of the limb, but also from the permanent shortening of the muscles, with consequent active displacement. The contractures which produce these deformities occur chiefly either in muscles which have escaped entirely or have only been partially affected, though there is reason for believing that the interstitial development of fibrous tissue in the remains of muscles sometimes plays a part in the fixation of a joint. The contractures sometimes appear as early as four weeks after the first development of the paralysis, but are usually late phenomena.

The mechanism of the production of the deformity is differently viewed by different observers. The original theory of Delpech, that it is the outcome of contraction of sound muscles which have shortened on account of their not being opposed as they naturally should by their antagonists, has been widely but certainly not universally accepted. Another theory accounts for the deformities by supposing that they are due to the influence of weight upon joints from which have been withdrawn the natural support of muscles and ligaments. Thus, the weight of the body, pressing unresisted on the arch of the foot, which has lost its natural stays, so to speak, gradually displaces the bones from their normal relations, until it entirely flattens the arch or distorts the whole extremity into some form of club-foot.

It does not, however, seem possible to account for some of the deformities by any theory of pressure. Thus, how could the drawn, contracted fingers seen in atrophic paralysis of the forearm and hand be the result of any pressure upon the part? In the lower extremity pressure probably does have a direct influence in the development of the club-foot. Thus, the weight of the body would tend to produce in the feeble foot equino-varus. It tends, therefore, to intensify the action of contraction in the sural muscles after paralysis of the anterior tibial, but to diminish the intensity of contraction of the anterior group of muscles when the gastrocnemius is paralyzed. In this may be found one reason for the rarity of pes calcaneus and the comparative frequency of talipes equinus after infantile paralysis. It is probable, however, that the chief cause of the infrequency of pes calcaneus after infantile paralysis is to be found in the fact that the calf muscles are much less frequently affected than are the anterior muscles. The most reasonable explanation of the production of the deformities seems to be that they are results of several coacting or reacting causes present in varying degree in various cases.

The deformities of poliomyelitis may affect any portion of the body. All varieties of club-foot, knock-knees and inverted knees, rigid flexion of the knees, cyphosis, lordosis, extraordinary scoliosis, subluxation of the thighs or of the humerus, claw-like distortions of the hands,—any of these may result, or the withered, shrunken limb, mobile almost as a rubber tube, may dangle from the trunk, an untoward memory of the past.

The **course** and symptoms which have been given of acute poliomyelitis are those seen in children. In the rare cases in which the disease occurs in the adult the general course is not essentially different from that which it holds in childhood. In the first stages, however, the cerebral symptoms are usually less severe and the vomiting more frequent than in very young subjects, whilst in the fourth or last stage of the disease the deformities are less pronounced than in childhood. Whether occurring in the young or the old, the disease is essentially the same.

Diagnosis.—The recognition of the true nature of an incipient attack of poliomyelitis with grave constitutional disorder is usually attended with much difficulty. Indeed, it is commonly impossible to do more than suspect, and such suspicion must rest upon exclusion; as, this attack is for such and such reasons

not one of the exanthemata, etc. etc.: no cause for ephemeral fever can be found, and therefore it may be poliomyelitis. The posture of habitual distrust upon the part of the practitioner is exceedingly important, as it leads to watchfulness for the appearance of paresis. Whenever such paresis appears the diagnosis at once becomes plain. The only affections which may be confounded with poliomyelitis in the early paralytic stages are peripheral neuritis and ascending paralysis. The completeness of the palsy and rapid alteration of the electrical relations of the muscles, together with the absence of nerve-pains and nerve-tenderness, demonstrate that the case is not one of peripheral neuritis, whilst the course of the paralysis and the occurrence of febrile and of trophic disturbances separate the affection from Landry's paralysis. Moreover, the latter disease is extremely infrequent in children, whilst acute poliomyelitis is extremely infrequent in adults.

Prognosis.—In the first or active stage of an acute poliomyelitis the prognosis has to do with two essentially different questions: first, as to the danger to life; second, as to the probable extent of permanent paralysis. Death has probably happened from the grave constitutional disorder that ushers in a poliomyelitis without the true nature of the malady having been recognized, but certainly death from a recognized poliomyelitis is exceedingly rare, so that in regard to immediate danger the prognosis is most favorable. No opinion, however, ought to be given during the first stage as to the probable extent and completeness of the permanent palsy that may result, since there seems to be no relation between the severity of the primary constitutional disorder and the gravity of the permanent disablement. The wildest storm may eventuate most happily, and the most insidious development may end in widespread ruin.

Even in the second stage, when the paralysis has reached its maximum, the prognosis must be guarded, for although there is a general relation between the severity of the paralysis of this stage and the final result, this relation is by no means fixed: a seemingly mild case may turn out most unfortunately, and a very widespread and profound paralysis may clear up entirely. After the end of a week, if the affected muscles have suffered no loss of faradic irritability, the prognosis becomes very hopeful; if, on the other hand, the electrical relations of the muscles are distinctly disturbed, then long-continued atrophy and loss of function must be expected. The earlier the electrical reaction of the muscles are altered the more serious is the prospect; and, *vice versa*, if after three weeks the muscles still respond well to the faradic current, the recovery will almost certainly be rapid and complete. When in an advanced stage the muscles are unable to respond to any electrical current, the case is almost hopeless. When the power of responding to the direct or chemical current is retained, although the faradic current produces no effect, the prognosis becomes hopeful in direct proportion to the length of time during which the paralysis has lasted; the longer the period that has elapsed the better is the outlook. The preservation of the power of reacting to galvanic currents proves that the spinal cells have not lost their power of influencing to some extent the nutri-

tion of the muscles, and affords ground for the hope that, although unable to stimulate the muscular nutrition to recover that which has been lost, they may still be able to hold up a muscle whose nutrition has been artificially restored.

Treatment.—When poliomyelitis commences with violent general disturbance, active local or even general antiphlogistic treatment may be instituted with the hope of moderating the activity of the inflammatory process, provided the strength of the patient be sufficient. After the paralysis has been developed it may in some cases be allowable to take blood locally from the back, but general venesection should never be practised.

The proper treatment of the second stage of the disease is still an unsettled problem. With the idea of diminishing congestion and lessening inflammation authorities recommend the ventral decubitus, the continuous application of cold by means of ice-bags along the spinal column, the administration of ergot, iodide of potassium, and mercury, and the use of the actual cautery or other violent counter-irritant; in a word, the treatment of an acute myelitis.

Erb and some other authorities apply the direct galvanic current steadily, without interruption (from three to ten minutes by some electricians, or as long as several hours by others), the positive pole being placed at the nape of the neck, the negative upon the lower end of the spinal column or upon the affected muscles.

There are at least seemingly sound theoretical reasons in favor of the antiphlogistic method, but, as has been shown elsewhere, there is no probability that the galvanic current as applied to the vertebral column reaches the spinal cord, and neither physiological nor clinical data to prove that if it did reach the cord it would accomplish any good. Its application may sometimes have salutary mental effect upon the little patient and upon the parents, against which is to be set the annoyance of the procedure.

My own belief is that in the second or paralytic stage the treatment should be largely expectant, but that extract of ergot should be given in as large doses as the stomach will bear, and that calomel should be cautiously administered, and the actual cautery be lightly but freely applied, provided that the patient be old enough and intelligent enough for it to be used without causing spasms of terror. In the very young or timid, if it be decided to employ the cautery, ether anæsthesia should be induced without the patient knowing what is to be done.

During the stage of regression medicinal treatment should be limited to the use of tonics and the persistent administration of very minute doses of corrosive sublimate, whilst the health of the patient should be built up in all possible ways and the nutrition of the muscles maintained by use of electricity, massage, etc.

In the fourth or permanent condition strychnine and phosphorus may be administered with the hope of stimulating ganglionic repair. Tendencies to the development of deformities are to be mechanically combated and the muscles locally treated. In some instances the hypodermic injection of the strychnine salts into the paralyzed muscle has seemed to do good.

In the local treatment of the muscles three distinct measures are available:

First: Mechanical vibratile treatment, combined with the application of heat (and perhaps also of a Junod's boot), by means of Zander's or some other similarly acting mechanism;

Second: Massage, and also passive gymnastics;

Third: Electrical treatment.

The action of the first of these measures is, I have no doubt, of value by stimulating the capillary circulation, and whenever the requisite machinery is at hand the treatment should be carefully and persistently tried over some months. Massage and passive gymnastics have the same aims as the mechanical treatment just spoken of, and are to be used when they can be commanded: to accomplish anything at all they must be employed very persistently as well as skilfully. It should be remembered that rubbing the skin by an untrained person is not massage, and does not, like that procedure, reach the deeper circulation: what is wanted is kneading of the paralyzed muscles.

Electricity has been extensively employed in acute poliomyelitis with very widespread disappointment. It is, however, a really valuable agent when used with a proper understanding of the methods of its application and the limitations of its usefulness. It has no influence whatever for good over any of the structures involved except the muscles themselves, and its application to the spinal cord or nerve-trunks at any stage of the disease is worse than useless. In regard to the time when electrical treatment should be commenced, my own opinion is that so soon as paralysis is detected electricity may be carefully employed. At this time, however, great caution is necessary to avoid producing muscular fatigue or any reflex irritation of the nerve-centres. The séances should therefore be short and the current only sufficient to produce feeble muscular contractions. The good accomplished is largely, but probably not altogether, due to the functional excitement of the muscle by the electricity, and consequently I have formulated the law that the current to be employed is that which will produce the greatest muscular contraction with the least pain. This law applies to all stages of the disease. Ordinarily, the faradic current fails entirely, and the direct chemical or voltaic current must be employed. It must be remembered that improvement of the muscles is of no avail unless the spinal cord recovers its power, but the effect of partial rehabilitation of the ganglionic cells is greatly increased by keeping the muscles in such a condition that they are able to respond to whatever impulse may come from these cells.

If the case be first seen by the neurologist in the advanced stage, it may be taken almost for granted that the amount of paralysis is greater than that which the state of the cord necessitates, so that electrical treatment offers a good hope of amelioration. This is especially true if the muscles have still some power of responding to the electrical current, and even when they seem at first entirely dead, trial for two or three weeks should be made, as sometimes muscles under these circumstances are awakened by electricity into new life and some voluntary power is regained.

In the administration of the current a single well-wetted electrode should be put over the motor point of the muscles, with a larger electrode at a little distance, so placed that as much of the muscles as possible shall be reached by the current. This procedure may be varied from time to time by placing the poles so as to include between them the whole length of the muscle. The galvanic current may be slowly interrupted, but the effect upon the muscles is much greater if by mechanical arrangement instead of simple interruption there is reversion of the current, so as to make alternating to-and-fro currents. If after eight weeks of electrical treatment no gain is achieved, nothing is to be hoped for.

In all cases of infantile paralysis it is essential to prevent, as far as may be, the development of deformities. Contractures are to be overcome, if possible, whilst forming by thoroughly stretching the muscles morning and evening with the hand. When, in spite of this, the contracture persistently increases, section of the tendons should be resorted to. The operation is simple, without danger, and experience shows that the relief to the limb has a distinct effect upon the nutrition of the muscles. So true is this that I think that after such section a renewed attempt to develop the muscles by electrical treatment should always be made. The application of braces or other appliances to the legs to aid in locomotion is often imperatively demanded. It is very much better for the child to exercise the limb, even partially, than to add to the failing nutrition of spinal disease the depressing influence of loss of use.

Subacute or Chronic Poliomyelitis.

In 1849, Duchenne described a peculiar palsy of which various cases have from time to time been since reported, and which appears to have very close relations with acute antero-poliomyelitis. The symptoms are rapidly-developed paralysis, usually commencing in the lower extremities and extending upward, associated with complete muscular flaccidity; loss of reflex excitability; rapidly-progressive atrophy; and changes in the electrical relations. This disease is said to be distinguished from the acute poliomyelitis by the absence of the stages of general stationary paralysis and of regression, and also by its progressive course. It is distinguished from progressive muscular atrophy by the paralysis producing—not following—the atrophy, and by the appearance of well-marked reactions of degenerations early in the case, as well as by the loss of the reflexes. Undoubtedly, cases of neuritis have in the past been reported as instances of subacute poliomyelitis, but they are to be distinguished by the nerve-pain and tenderness.

When subacute poliomyelitis shows a distinct tendency to ascend, there is always grave danger to life by implication of the muscles of deglutition and of respiration. In the majority of cases recovery occurs with more or less damage to muscles and consequent defects of motion.

The treatment may be that of chronic myelitis, with the superaddition of local electrical treatment for the maintenance of nutrition to the muscles, as in the acute disorder. The results which I have obtained in metallic polio-

myelitis (next paragraph) would seem to quite justify the trial of heroic doses of strychnine.

Atrophic paralysis, produced by arsenic or lead, sometimes closely simulates subacute poliomyelitis. Probably in the majority of cases it is the outcome of a peripheral neuritis, when its nature is to be recognized by the existence of nerve-pains and tenderness. I have, however, seen cases lacking in such tenderness, in which rapid loss of power, with atrophy and atrophic changes in the muscles, occurred without pain or nerve-tenderness, precisely as in subacute poliomyelitis, and in which I believe the lesion was purely centric.

The true nature of metallic subacute poliomyelitis can usually be made out by attending to the following points: first, the case occurs in an adult; second, the paralysis is much more widespread than in the subacute or mild cases of poliomyelitis, and develops itself to the fullest extent only after some weeks; third, muscles not usually affected in true poliomyelitis are impaired almost as much as their fellows (thus the sphincters are paralyzed, the bladder rapidly loses power, and the respiratory muscles grow weak); fourth, sensation is often, but not always, affected to some extent; fifth, suspicion being aroused, evidences of metallic poisoning can be obtained from the history, from the presence of a blue line on the gums, or by finding the metal in the urine. The treatment of this condition is that of metallic poisoning, added to the local use of electricity upon the muscles and the employment of massive doses of strychnine; which alkaloid I have seen, when pushed to its physiological limit, act with almost as much force and certainty as does quinine in malarial diseases.

Syringomyelia.

Definition.—A chronic disease dependent upon the formation of pathological cavities in the spinal cord, and clinically characterized by peculiar alterations in the sensibility, and loss of power usually accompanied by trophic disturbances.

Etiology.—Concerning the causes of syringomyelia we have no definite knowledge. The disease usually begins between fifteen and thirty-five years of age; is more frequent in men than in women; and does not appear to be distinctly hereditary, although there is some reason for believing that it depends upon some embryological affection of the cord which diminishes the power of the nerve-elements to resist the hyperplastic tendency inherent in neurogliar tissues.

Pathology.—The principal lesion in syringomyelia is spinal, with secondary trophic lesions in muscles, bones, cellular tissues, skin, and probably also in the peripheral nerves. To macroscopic examination the cord presents the appearance of a large blood-vessel empty and collapsed. It is irregularly increased in size, deformed, soft and fluctuating to the touch, or feeling like a hard, firm, rigid cord, as the case may be. Section reveals a cavity, or more rarely two or even three cavities, situated in the horns of the gray matter. The size of the cavity and its length vary indefinitely, and its shape and cross dimensions also vary not only in different individuals, but in different

parts of the same cord. Its contents are liquid or gelatinous, and even to the naked eye it is surrounded by a smooth, yellowish membranous coating.

The majority of investigators believe that the primary histological lesion of syringomyelia is a neoplastic hyperplasia of the neuroglia of the gray matter, but others insist that it is a hyperplastic myelitis. The new tissue is yellowish-brown, and usually composed of one or two nucleated, spider-like cells, heaped together and anastomosing with one another by their nervous branch-like processes. In the interspaces thus made are granular elements, pigment-granules, and small, illy-defined, yellow refractile bodies of doubtful character. The limiting layer lining the whole of the cavity is a dense, fibrillary felting, which is not sclerotic, but has probably been formed from prolongations of the cell-process. The parts around the new growth are compressed and irritated, and so secondary inflammation, hæmorrhage, and widespread sclerotic degeneration are set up. The peripheral nerves have been in various cases found altered, enlarged, with parenchymatous and interstitial neuritis, or finally atrophied. In a very careful study of Dejerine it was found that the intramuscular nerves were normal or atrophic according as their muscles were normal or atrophic, indicating that the changes in the nerves are secondary and trophic, and not primary lesions.

Symptomatology.—Syringomyelia commences insidiously, with weakness and some disorder of sensation in the upper extremities, followed after a time by muscular atrophy, with increase in the sensory disorders; then by spinal curvature in the form of scoliosis; and finally development of motor palsy in the lower limbs. Vaso-motor and trophic changes in the skin, subcutaneous cellular tissues, and perhaps in the joints and bones, soon follow the appearance of the muscular atrophy.

The symptoms of syringomyelia are best discussed in detail by an analysis of the individual groups:

Sensibility.—The disturbances of sensation are the most characteristic of any of the symptoms. The ordinary sensations are disassociated, so that whilst sensibility to touch, the muscular senses, and the special senses remain perfect, the sense of pain and the power of recognizing heat and cold are more or less completely lost. In some rare instances the general rule is deviated from, either in the preservation of some form of sensibility commonly lost, or more frequently in the depression of some of the sensibilities commonly preserved. Cases are on record in which pain and thermic sensibility have been increased; further, thermic sense perversions may exist, so that hot bodies feel cold and cold bodies hot.

The degree of the thermic anæsthesia varies from the simple inability to note slight differences of temperature up to such complete loss that a patient may be burned without being aware of it. The loss of the power of recognizing heat does not necessarily coincide in degree or position with the loss of the perception of cold. The distribution of the thermo-anæsthesia varies in different cases, and to a limited degree from time to time in the same case. It usually occupies considerable zones—sometimes nearly the whole surface of

the body, and even the mucous membranes, as well as the skin. Analgesia varies in intensity and in distribution, precisely as does the thermo-anæsthesia. In spite of complete analgesia and thermo-anæsthesia the slightest prick will be recognized by the tactile sense, whilst the eye, the nose, the mouth normally perform their seeing, smelling, and tasting functions. In some cases the patient complains of subjective pains which may mock the sensations of burning or of freezing.

As already stated, loss of motor power in the arms is a common primary symptom. In the legs the disturbances of motion, which are usually secondary and develop late in the disorder, commonly consist of spasmodic paraplegia, but sometimes are especially shown in marked ataxic inco-ordination. Following one or other of these, the patellar reflexes may be either exaggerated or abolished. Loss of motion is followed in the upper extremities by muscular atrophy and secondary contractures, with the production of claw-like deformities like those of progressive muscular atrophy. The muscles of the back suffer paralysis and trophic disturbance almost as soon as do the muscles of the upper extremities, and therefore scoliosis is an almost constant and sometimes an early symptom. It is said almost universally to affect the dorso-lumbar region and to produce convexity to the left.

The atrophy which first appears in the muscles of the forearm usually extends slowly and symmetrically up the arm, and is sometimes accompanied by the reaction of degeneration, although usually the electrical excitability is only diminished. It should be noted that both paralysis and atrophy may first appear in the scapular region, or even in the lower extremities, and that a few cases of facial paralysis with atrophy have been reported.

The superficial trophic changes are very marked. The skin may become glossy or covered with a thick epidermis or with bullous, eczematous, or herpetic eruptions. Perforating ulcers have been described, and in rare cases there has been a primitive gangrene of the skin, followed by loss of substance and leaving a whitish cicatrix. The distorted, thickened, often furrowed nails sometimes fall out. The subcutaneous cellular tissues may be œdematous or the seat of abscesses and especially of whitlows. The bones and joints sometimes undergo arthropathic changes similar to those seen in locomotor ataxia; and acromegalia, coinciding with, if not dependent upon, syringomyelia, has been reported.

The secreting nerves seem to suffer; at least sweating becomes irregular, absent in some regions, or it may be exaggerated. The vaso-motor system is also attacked, the extremities cyanosed, with their temperature distinctly below the norm, or else they become swollen, scarlet, and hot. Polyuria has also been noted. Sometimes cystitis is severe, and perforating ulcer of the bladder has been reported.

The general type of syringomyelia is departed from when the lesion is atypically located. Thus, bulbar paralysis, with disturbance of deglutition and of speech, may occur when the change is very high up in the nervous system. Amaurosis, unequal pupils, cardiac disturbances, all have been noted.

More strangely, the characteristic lesions of syringomyelia have been found after death in cases where no symptoms have been manifested during life. The only plausible explanation of this is that offered by M. Bruhl, which is that these latent forms occur only in young patients in whom there has not been time for development of symptoms.

Two clinical varieties of the disease are described by Blocq. In the first of these the atrophy commences in the muscles supplied by the ulnar nerve, and is followed by spastic paraplegia; in the other the atrophy commences in the muscles of the radial nerve, and is followed by tabetic inco-ordination. The course of syringomyelia is a prolonged one, disturbed often by exacerbations and remissions. If the patient do not die of some intercurrent disease, death results from some of the trophic lesions (gangrene, cystitis, perforating ulcer), from bulbar complications, or occasionally from sheer exhaustion.

Diagnosis.—Syringomyelia is distinguished from cervical pachymeningitis by being much less painful and not accompanied by rigidity of the neck, and by the existence of the peculiar disturbances of sensation. In cases of sclerodactylitis, simulating syringomyelia, sensation is preserved, whilst the inflammation of the skin is a dominant, not a secondary, feature of the case. In alcoholic paralysis thermo-anæsthetic disturbances resembling those of syringomyelia sometimes occur, but the symptoms usually appear in the lower extremities and are developed very rapidly, whilst tenderness of the muscles or nerve-trunks upon deep pressure can be made out. Charcot has pointed out that hysteria may closely mark syringomyelia, but an error of diagnosis can always be avoided by carefully examining the patient and her or his history. Hysteria is rapid in its onset, and manifests its presence by nervous symptoms not belonging to syringomyelia.

The question as to the distinctness of Morvan's disease is still *sub judice:* those who believe in the non-identity make the diagnosis to depend upon the following points:

In Morvan's disease the tactile sense nearly always disappears with the other forms of sensibility; the trophic changes predominate, and almost exclusively consist of multiple whitlows, deep cracks and fissures in the skin, and arthropathies of the smaller joints. Moreover, in certain cases these affections are symmetrical on both hands and feet and do not attack the remainder of the body. Finally, the muscular atrophy is slightly marked, and is not, as a rule, progressive.

Prognosis.—The prognosis of syringomyelia is exceedingly serious, although it has been affirmed that recovery is possible, and life may certainly be much prolonged.

Treatment.—Counter-irritation by means of the actual cautery and suspension, as in the treatment of locomotor ataxia, are recommended by French authors. There seems no probability that any drugs have direct influence upon the disease, but of course symptoms must be met as they arise and defective nutrition combated. Electricity may be employed for the prevention of muscular atrophy. The most important indications are for careful hygienic

and medicinal management to protect from injury the analgesic skin and to prevent muscular fatigue or cystic complications.

Locomotor Ataxia.

Definition.—A disease of which the lesion is sclerosis of the posterior root-zones of the spinal cord, and in which the symptoms are vertigo, disturbance of the movements of the eyeballs, Argyll-Robertson pupil, contraction of the field of vision, pain or other disturbance of sensation, loss of the knee-jerk, loss of co-ordination, without true paralysis or spasm.

Synonyms.—Posterior spinal sclerosis; Tabes dorsalis; Duchenne's disease.

Pathology.—The general structural changes of posterior sclerosis are very evident, and its pathological histology well determined, although our knowledge of the true nature and of the development of the lesions is imperfect and the theory of the disease still somewhat speculative. When in the typical case the spinal cord is examined macroscopically by means of repeated sections, the structural alteration is seen to be most intense and widespread in the lumbar region of the cord, whilst the posterior columns appear to be replaced by a gray, almost gelatinous-looking, substance (gray degeneration). When the cord has been hardened in chromic-acid solution the difference between the sound and diseased tissue is very marked, and it usually can be readily made out that the tract of degenerative tissue continually narrows from below upward by a loss of its lateral or external portions, so that at last it becomes a narrow band or column (in section a zone) upon each side of the posterior spinal fissure. This arrangement of the degeneration is not, however universal, and the upper portion of the cord, or even the medulla, may be primarily and most severely involved.

The sclerotic process usually begins in that portion of the posterior column through which the posterior roots of the spinal nerve run, and to which the name of the "posterior root-zones" has been given. This portion of the spinal cord has in it ascending nerve-bundles, which in their passage upward escape into the slender columns in immediate juxtaposition to the posterior fissure (columns of Goll). In travelling up the cord the degenerative process extends along the root-zones and along the columns of Goll. In some cases the columns of Goll seem to bear the brunt of the disease, in others the root-zones are especially affected.

Microscopic examination of those portions of the cord where the pathological process has reached its farthest limit reveals a mass of connective tissue, with scattered through it here and there minute points, the atrophied axis-cylinders from which the myeline sheaths have wasted away. The connective tissue is fibrillated and nucleated, with excessive trabeculæ. The blood-vessels also have undergone a marked sclerotic change, which especially affects the outer coats and adventitial sheaths, and lessens the lumen of the vessels. When the sclerosis is less advanced, the change consists in an increase in the nuclei and the mass of the connective tissue, accompanied by wasting

of the myeline sheaths, and, it may be, a disappearance of some of the axis-cylinders.

The exact beginning of the sclerotic process has not yet been positively determined, but the view of Westphal that there is a stage of granular change preceding that of pronounced sclerosis is received with favor by most pathologists. On the other hand, pathologists are divided in regard to the question whether the lesion of locomotor ataxia commences in the connective tissue or in the nerve-elements; some maintaining that the disease is originally interstitial, and that the nerve-elements are wasted by the pressure exerted upon them by the hyperplastic neuroglia, whilst others teach that the disease is parenchymatous—*i. e.* that the original change occurs in the nerve-filaments, and that the connective-tissue hyperplasia is a secondary result. Some, notably Spitzka, go so far as to believe that there are two varieties of locomotor ataxia: one parenchymatous, one interstitial—a view which seems to me highly improbable.

As was first pointed out by Westphal, the peripheral nerves frequently suffer in tabes, the degeneration beginning in the white matter and ultimately spreading to the axis-cylinders, and being accompanied by an increase of connective tissue. The sensory filaments appear to suffer alone. That the degeneration is not due to propagation by contiguity of the spinal lesion is shown by the fact that the change begins in the cutaneous filaments and leaves the large nerve-trunks free. Further, the degree of alteration in the nerves bears no proportionate relation to the changes in the spinal cord. There is much reason for believing that in some cases of tabes the change in the nerves precedes the change in the cord, and that both alterations are the result of a common cause.

Atrophy of the optic, pneumogastric, trigeminus, and other cephalic nerves, with great wasting of fibres and hyperplasia of connective tissue, and even involvement of the central ganglia, are occasionally found.

The gray matter of the cord is unaffected save in very advanced cases, when the columns of Clarke show atrophy of their fine nerve-fibres, and in some cases wasting of their cells. When there has been during life widespread muscular atrophy there is corresponding atrophy of the spinal cells.

Etiology.—Locomotor ataxia is not hereditary; is much more frequent in males than in females; is a disease of middle life, although it may occur at any age. The causes in individual cases are usually very obscure. In a large majority of cases there is a past history of syphilitic infection, but the disease is not, strictly speaking, syphilitic, and is not relieved by antisyphilitic treatment. Professor Strümpel's theory that it occurs only in the syphilitic, and is produced by a post-syphilitic chemical poison, is highly improbable. The old belief that sexual excess is the ordinary cause of locomotor ataxia is certainly not true, though such excess may aid in its development. Over-work, nervous strain, worry, emotional excitement, the immoderate use of alcohol and tobacco, have been assigned as causes, but their influence is very obscure. Habitual or even single unaccustomed exposure to wet and cold, especially when combined

with exhausting labor, appears to have had distinct influence in some cases. Peripheral traumatisms seem at times to produce tabes, probably by causing an ascending nerve-degeneration.[1] How far lead and other poisons act is uncertain, but Tuczek has described under the name of *tabes ergotica* an affection closely simulating locomotor ataxia. It is probable that in the majority of cases various causes work together for the one result. Syphilis, venereal and alcoholic excesses, worry, mental strain, over-exertion, and undue exposure are often coincident factors in the life of one individual.

Symptomatology.—The development of locomotor ataxia is usually very slow, insidious, and without fixed regularity in the relative development of symptoms. Usually the first manifestations are in the legs, but sometimes double vision, giddiness, or pains about the head take precedence, and the affection may for years be seated almost exclusively in the upper extremities. This is evidently due to the fact that although usually the pathological changes begin in the lower portions of the spinal cord and work upward, sometimes they pass from above downward. The nature of the symptoms of the earlier stages will be discussed under Diagnosis. The general symptoms can be best studied in a brief space under the headings of the functions involved:

First: Disturbances of sensation;

Second: Disturbances of motion;

Third: Disorder of the organs of the special senses;

Fourth: Trophic changes.

Disturbances of Sensation.—Pain is present in nine-tenths of the cases of tabes. It is variously described by the sufferers as shooting, darting, as a feeling as though lightning were running through the part or as though a red-hot wire or a sharp dagger were thrust through the limb. In some cases these so-called *fulgurant pains* occur continually; in other instances they come on in paroxysms; but almost invariably they temporarily disappear at intervals. They may follow the distribution of the nerves, but more commonly are felt most severely in the neighborhood of the joints, especially in the inside or the outside of the knee or of the ankle. Usually they are not associated with redness or any soreness, and often the patient seizes the affected part forcibly and obtains by the pressure some relief. A certain amount of redness and tenderness may, however, be present during the pain, and in exceedingly rare cases trophic eruptions occur. Sometimes the pain seems widespread and superficial, and is then usually spoken of as burning or more rarely as a sensation of intense cold.

Pain crises, which are almost pathognomonic of locomotor ataxia, consist of paroxysms of excessive pain without fever, located in some viscus or organ, and accompanied by excessive functional disorder of the part. They frequently come on and disappear with great rapidity and abruptness, and may last from a few minutes to several days. When the agony is supreme syncope may bring relief, or there may occur a wild outburst of maniacal mel-

[1] See Klemperer, *Thesis*, Berlin, 1889.

ancholy with attempts at suicide. The paralyzed functional activity is usually recovered with remarkable rapidity when the paroxysm ends. The most important of the pain crises are the muscular, the gastric, the rectal, the urinary, the genital, the cardiac, and the laryngeal.

The *muscular crises*, which are extremely rare, consist of a feeling of lassitude, deepening into an excessive muscular weariness and soreness (like that which follows violent exercise in one unaccustomed to it), and at last entirely paralyzing, for the time being, the affected muscles.

The *gastric crises*, the most frequent of any, are characterized by violent shooting and burning pains, having their focus in the epigastric region and radiating in all directions, laterally, upward, downward, until at times they seem to fill with agony the whole abdomen and chest. They are generally increased by epigastric pressure and by the ingestion of food, and are always accompanied by nausea and excessive vomiting. After the stomach has been once emptied the discharge is glairy or ropy, neutral or acid, and often streaked with blood; rarely there is abundant coffee-ground vomiting or even pronounced hæmatemesis. In some cases the focus of the pain is in the neighborhood of the umbilicus, when the crises might properly be spoken of as *intestinal*. Occasionally large quantities of gas form in the gastro-intestinal tract and produce a very obstinate meteorism, or there may be copious bilious, mucous, or serous stools. In such cases loss of the voice, suppression of urine, extreme coldness and cyanosis of the body, cramps, and collapse may closely simulate cholera, and death may result.

In the *rectal crises* the pains radiate from the rectum, or this receptacle feels as though it were filled up by an enormous body heated to redness, burning and scorching every part near it.

The *genital crises* are of two characters. In one, violent paroxysms of pain centre in the testicles and shoot along the penis to its head, or in the female burn and bore in the ovaries, the labia minora, and the clitoris. In other cases frequent spontaneous venereal orgasms replace the pain-paroxysms. At first the paroxysms end in a fury of voluptuous delight, but more and more pain tramples over pleasure until at last the orgasm is but unspeakable torture.

The *genito-urinary crises* may simulate renal colic, the pain radiating along the ureters into the genitalia and being associated with retraction of the testicles. More frequently, however, intense burning or lancinating darts of agony in the urethra, associated with unconquerable cystic tenesmus, suggest the presence of calculi—a suggestion which is especially forcible in those cases in which the pain is persistent and anæsthesia of the bladder gives rise to urinary retention and ammoniacal fermentation, with subsequent deposits of phosphates.

The symptoms of the *cardiac crises* are violent lancinating and constricting pains in the region of the heart, associated with great dyspnœa, intense distress, and irregularity of the pulse, with or without intermission of the heart-beats.

The *laryngeal crises* consist of violent paroxysms of hoarse coughing, ending

in a raucous inspiration like that of whooping cough, and attended by great laryngeal disturbances of respiration and atrocious fulgurant pains in the shoulders and along the spinal column. The expectoration is of a scanty, saliva-like secretion, or rarely of little pellets of mucus stained with blood. Asphyxia may in these cases be so extreme as to produce coma and even death. In some cases laryngeal paralysis or anæsthesia occurs, and death has resulted from the pneumonia produced by food passing into the larynx.

Amongst the various paræsthesias of locomotor ataxia are formications, the feeling that water is running over the part, crawling of ants, etc., and especially the so-called girdle sensation, a feeling as though a tight band was drawn around the head, the neck, the body, or the limbs, in accordance with the seat of the lesion in the cord. Numbness may develop early or late. When it is situated in the feet the patient feels as though he were walking upon velvet or upon cushions of down. The mucous membrane of organs, such as the larynx and rectum, in which the crises occur is often completely anæsthetic.

In the earlier stages the numbness is not invariably associated with loss of sensibility, and the æsthesiometrical points may be distinctly recognized, but later tactile sensation becomes impaired. The anæsthesia may exist with or without analgesia, although the pain sensation is usually also lost. Sometimes a separation occurs between tactile and pain perception, so that a distinct interval exists between the perceiving of the contact of a sharp point and the pain which it causes. The temperature sensation is usually diminished, but Donath affirms that there are cases in which it is exalted. The lack of co-ordination of tabes is, I believe, largely due to loss of muscular sense, but as physiologists are not all agreed even as to the existence of this sense, the discussion would require more space than can be afforded here. Delayed sensation is not uncommon, and five, ten, or even fifteen seconds may elapse between the time of the contact and its perception. Mendelssohn affirms that the normal reaction of the sensory nerve to electricity may be reversed, so that on closing of the circuit the earliest sensation is at the positive instead of at the negative pole, as in health. The localizing power is sometimes curiously perverted: a single prick may be felt in many places (*polyæsthesia*), or a prick on one leg may be located on the other (*allocheiria*).

Motion, including the Reflexes.—The influence of locomotor ataxia upon the cutaneous reflexes varies. In the onset of the case they are, in rare instances, increased; when anæsthesia exists they are usually diminished; but sensation may be well preserved and the cutaneous reflexes be lessened or even abolished. The deeper reflexes are profoundly affected. Complete loss of the knee-jerk (*Westphal's symptom*) is one of the earliest and most constant phenomena. When, however, the disease commences in the upper portion of the cord, it may be late in coming on.

Loss of co-ordination shows itself both in station and in locomotion. When it exists only in a slight degree, the patient may be able to stand with the feet close together or on one foot, or may be able to walk fairly well;

but if the eyes be closed, the lack of control becomes at once manifest, and as the disease develops a gait so peculiar as to be spoken of as the *ataxic gait* results. In the earlier, but fully-developed stages the patient walks with his head a little bent forward and the eyes directed to the ground. The trunk inclines upon the thighs, whilst the feet are held in advance of the buttocks, with the legs widely separated from each other. At the same time, owing to the excessive contractions of all the muscles of the lower extremities, the leg proper is extended somewhat rigidly upon the thigh, and there is very little movement at the knee-joint. The advancing leg is therefore raised from the ground in some degree by an elevation of the pelvis, although at the same time some flexion does occur at the knee-joint. By these conjoint movements the foot is freed from the ground, and, having been flung forward and outward by a rapid muscular jerk, comes down with a thump like a solid mass. In some cases the heel is the last to leave the ground and the first to touch it. Not rarely the pelvis is so much inclined during walking as to carry the centre of gravity too far toward the side of the stationary leg. To counteract this and maintain the balance of the body the upper portion of the trunk is curved toward the advancing leg by a contraction of the erector spinæ muscles, or the arm corresponding to the advancing leg is thrust out laterally. The alternation of these movements at each step may give a pendulum-like swing to the body. In a more advanced stage of locomotor ataxia the patient is able to walk only by the help of two sticks or crutches. The body is thrown forward in order to counteract the tendency to fall backward produced by the peculiar position assumed by the legs, which are held in advance of the buttock on account of the tendency to undue contraction of their extensor muscles: the foot is usually at an obtuse angle to the leg, and the thigh at an obtuse angle to the trunk. If under these circumstances the trunk be erect, the line of the centre of gravity would fall through the buttocks posterior to the point of support—*i. e.* the foot—and consequently the patient would fall backward. To overcome this, the trunk is often bent so far forward that the line of the centre of gravity is in front of the feet, and the patient would fall forward if he were not supported by a stick or crutches. All the movements executed with the legs are performed with great stiffness and by sudden jerks. The straddle is usually very marked, and the leg is raised from the ground by an elevation of the pelvis in the method already described. Still later in the disorder the legs are entirely beyond the control of the patient. They are thrown around in wild, irregular, choreiform movements, which render them of no use whatever in walking. Under these circumstances progression is impossible. When the lesion travels up the spinal cord, all power of co-ordinating the muscles of the trunk may be lost, so that the patient is no longer able to sit in a chair.

Organs of Special Sense.—Of the organs of special sense, the eye is the one most frequently attacked in locomotor ataxia. Of the external ocular muscles, the rectus is the most frequently paralyzed, but any muscle may be affected. The loss of power may be transient or permanent. Transient

ocular palsy, with its resultant transient diplopia, belongs to the earliest period of the disorder. The permanent palsy is seen in the later stages, and may produce ptosis, internal or more rarely external squint, and even a general ophthalmoplegia.

The pupil is affected sooner or later in a majority of cases of locomotor ataxia. It is usually contracted, but mydriasis may occur, and irregularity of the pupil is sometimes seen. The most characteristic alteration is that known as the *Argyll-Robertson pupil*, or as *reflex iridoplegia*. In this condition the pupillary reflexes are abolished, although the normal relations between the pupil and accommodation are preserved; consequently, no pupillary movement occurs when the skin of the neck is violently pinched or when light is thrown suddenly into or shut off from the disordered eye, although the pupil dilates when the gaze is suddenly directed from a near to a distant object.

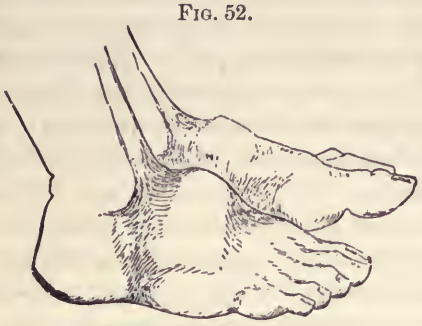
The most characteristic visual results of locomotor ataxia are contractions of the field of vision, with disorder of the color sense. The contraction is concentric, but is usually somewhat irregular. The power of perceiving yellow and blue is kept for a long time, whilst blindness for green or red is early developed. The contraction of the field of vision and the disorder of the color sense are due to degeneration of the nerve-fibres of the optic nerve; which degeneration usually commences in the periphery.

I know of no observations on disorder of either taste or smell in locomotor ataxia. Deafness occasionally occurs either as an early transient or a late permanent symptom. According to Gowers, it is accompanied by a progressive limitation of the range of hearing analogous to the contraction of the range of visual field; the notes of the scale, beginning at the top, dropping out of the range of hearing, one after the other, until all are alike inaudible.

Trophic Changes.—The most important trophic changes in locomotor ataxia are alterations of the bones and joints, perforating ulcer, and perhaps cardiac disease. Sclerotic arthropathy may first show itself by a peculiar articular crepitus, but the first fully-formed stage is that in which a serous effusion, free from blood, pus, or albuminous flocculi, occurs in the articular cavity, whence it may extend into the tissues around the joint and even into the affected limb. The joint at this time is enormously swollen, hard, usually pale, and so resistant as not to pit on pressure. In rare cases the effusion is absorbed, but usually the second stage is soon developed. At this time the joint is much swollen, hard, and bony, with an evident increase in the size of the bony surfaces. In the third stage there is destruction of the articulating surfaces, and in some cases so much absorption of the bone and changes in the ligamentous structure as to produce great alterations in the power of movement. The epiphyses especially undergo atrophy and change; the ligaments are elongated, probably as a consequence of prolonged stretching by the excess of fluid; and at last a condition of subluxation, or perhaps of complete luxation, of the joint occurs, so that the ataxic may be able voluntarily to put out of joint a shoulder, a knee, or other joint without pain, though marked grating can be felt during

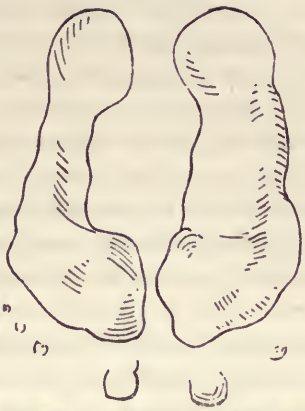
movement. The ataxic arthropathy is sometimes unilateral, but is frequently more or less symmetrical. It attacks especially the knees, and next in order of frequency the other joints of the lower extremities, but it may occur in any articulation of the body. When the small joints of the hand or foot are affected, peculiar deformations result, constituting

Fig. 52.



Tabetic Feet (after nature).

Fig. 53.

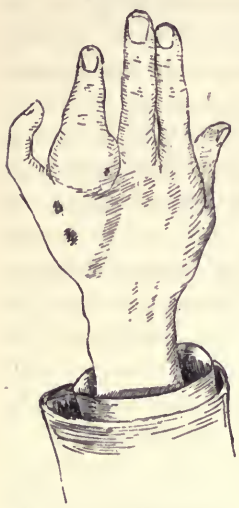


Impression of Tabetic Feet (after Ball).

the so-called "tabetic foot" and "tabetic hand." (See Figs. 52, 53, and 54.)

The shafts of the bone may atrophy and spontaneous fractures occur. The primary change is probably always an hypertrophy, which is followed by a pronounced atrophy. When the jaw is attacked the wasting of the alveolar processes results in the dropping out of the teeth, which, though entirely sound, may be shed one by one or tumble out *en masse*.

Fig. 54.



Tabetic Hand (after Ball).

Perforating ulcer may attack the hands or probably even the internal organs, but especially affects the vicinity of the metatarso-phalangeal articulations of the feet. The first symptom usually is a severe pain. This prodromic pain may, however, be entirely wanting. A small hæmorrhagic or ecchymotic spot now appears under the epidermis: in the course of a few hours the skin detaches itself, or more frequently becomes excessively thickened into a large, dry, corn-like mass; a small slough soon separates, leaving the ulceration round, with sharp, acute edges, piercing usually to the deeper tissues and in many cases reaching the articulation or the bone. Around the ulceration there is apt to be serous infiltration and swelling. The perforating ulcer may be recovered from either without loss of bone or with the throwing off of small necrosed flakes, but in most cases the bone becomes seriously diseased and a sinus forms. In this condition the lesion appears as a small aperture leading by a narrow sinus to diseased bone and surrounded by thickened, superimposed layers of epidermis. The surface of the spot is usually cold and anæsthetic, the characteristic feature

of the ulcer being its insensibility to irritants and its freedom from pain during rest. The attack may end in death from erysipelatous inflammations of the foot and leg, or in recovery with ankylosis and various deformities.

In 1879, Vulpian called attention to the frequency of valvular disease of the heart in locomotor ataxia, and his observations have since been confirmed by both German and French writers. Insufficiency of the aortic valve appears to be the most frequent lesion, but any valve, and even the heart-muscle, may be involved. How far these lesions ought to be considered as strictly trophic and due directly to centric nervous disease is at present uncertain. Nevertheless, when, as in some cases, a sharply-defined perforating ulcer forms upon a valve, the existence of a trophic influence can scarcely be denied, whilst the fact that cardiac lesions habitually occur when the cardiac crises indicate diseases of corresponding centres is certainly noteworthy.

Termination.—Very few of the symptoms of locomotor ataxia in any way compromise life, so that even when almost the whole length of the spinal cord is affected the patient may live on, without control of his voluntary muscles, for a quarter of a century. In women tabes does not prevent successful pregnancy; *per contra*, whilst pregnancy may for the time being hold in abeyance the symptoms, it has no permanent effect upon the disease. Usually in tabes life is sooner or later cut short by some intercurrent disease; especially are the kidneys liable to become diseased. Again, not rarely the tendency of the lesions to pass to other portions of the nervous system leads to the fatal result. Of all portions of the nervous system, one of the most prone to be secondarily affected is the gray matter of the cerebrum, with result of mental disturbance. Under these circumstances violent insanity, amounting even to an acute, rapidly-fatal mania, may develop, but more commonly the cerebral symptoms are less severe, taking the form it may be of a melancholia or slowly developing into a peculiar garrulous imbecility. Progressive paralysis of the insane and locomotor ataxia not rarely coexist, offering a clinical picture of two intermingled diseases. Sometimes the one, sometimes the other affection appears to be the first in development. Subacute myelitis is said sometimes to occur as a complication, and the lateral columns, or even the gray matter, may be involved.

Diagnosis.—The diagnosis of fully-developed locomotor ataxia is usually so easy as to require no discussion. The only affection which resembles it is the hysterical disorder known as *astasia* and *abasia* (*hysterical ataxia*, *automatic ataxia*). In this disorder, however, there is no lack of co-ordination in the movements of the limbs when the patient is upon the back, even though walking is impossible. The knee-jerk is rarely, if ever lost; the pains, if present, are cephalalgic or rhachialgic, and are very rarely if ever closely comparable with those of true ataxia; whilst the true nature of the hysterical disorder is unmistakably shown by its suddenness of onset, by its irregularity of course, and by the presence of choreic movements, convulsions, globus hystericus, or other distinctly hysterical symptoms.

The recognition of locomotor ataxia in its early stages is more difficult. The

most characteristic symptoms are the pains, loss of knee-jerk, loss of co-ordination, and the trophic and visual phenomena. Any of these may precede the others in order of development, and may continue for years without other symptoms: the co-existence of any two of these symptoms renders the diagnosis almost certain.

The pains of locomotor ataxia are usually to be distinguished from other pains by their being bilateral and not associated with persistent tenderness either on pressure or movement. They are, however, sometimes so closely simulated by fleeting gouty pains that the true nature of the latter can only be made out by the recognition of other symptoms of gout.

The visual phenomena are most important, but not absolutely decisive. As tabes may begin with a diplopia, a strabismus, or a laryngeal palsy, any such symptom occurring without obvious cause in a middle-aged person, not hysterical or syphilitic, should arouse suspicion.

When tabetic crises occur as a very early symptom, their true nature may be overlooked: it is, however, usually to be recognized by noting the repetition of the attacks, the suddenness of their onset, the severity of the pain, and especially the rapid recovery of the functional activity of the part, and the absence of evidence of local organic disease when the nerve-storm is past. Most of the crises are dependent upon sclerosis high up in the spinal cord: the lower extremities may be free from disease, but failure in co-ordination in the upper extremities or characteristic ocular disturbances may usually be discovered. The cardiac crisis is especially liable to be mistaken for angina pectoris, but the seat of the pain does not actually correspond to that of angina pectoris. The focus is usually in one or the other axilla, whilst the pains do not radiate down the left arm, or, if they do so, at the same time shoot through the body itself and into the right arm. The pains are also more lancinating than in angina pectoris, and especially are the individual paroxysms more prolonged.

Peripheral neuritis, when confined to the lower limbs, may produce an ataxia with pain which has been attributed to centric disease. In such a case, however, there is always tenderness over the nerve-trunks, whilst in locomotor ataxia no such tenderness exists, except when the centric disease is complicated by neuritis. Under the latter circumstances great care must be exercised to avoid making a mistake. The occurrence of crises or of visual symptoms would establish the presence of centric disease, but it must be remembered that gummatous syphilis may simultaneously attack the nerves and the nerve-centres, and produce symptoms like those of locomotor ataxia, with or without neuritis.

Prognosis.—The prognosis of locomotor ataxia is very serious, and in the fully-formed disease, so far as cure is concerned, I believe hopeless, the few reported cases of cure having, in my opinion, been founded either in errors of diagnosis or of observation or in the too early report of the case. Gummatous syphilis may give rise to symptoms very closely resembling those of locomotor ataxia, and be relieved by antisyphilitic treatment. Improvement of the symp-

toms under treatment, and indeed arrest of the disease, do, however, occur in locomotor ataxia, especially in the early stages.

Treatment.—In the management of a case of locomotor ataxia it is of the first importance that all sources of exhaustion or of nervous depression be cut off. Rest, both bodily and mental, is vital. The life of the patient should be permanently arranged in such a way as to avoid all unnecessary expenditure of vital force. Physical labor is of course impossible, and mental work should be so reduced that it will only be sufficient to divert the attention of the patient from himself. There can be no doubt that the disease may be sometimes arrested, temporarily at least, by placing the patient in bed for a series of weeks, and at the same time using massage to prevent the bad effects upon the general health which such confinement tends to produce. Even when the patient is going about and in the best condition, long walks should be avoided, it being remembered that a single hour's exhaustion may overthrow the good achieved by many weeks of rest. The diet should be nutritious, but non-stimulating, and a moderate use of wine is not harmful, although the slightest excess of alcohol is certainly very deleterious. Tobacco must be used only in the greatest moderation.

Sexual intercourse should be as far as possible avoided. It is affirmed by good authority to be especially harmful in those cases in which there is a tendency to atrophy of the optic nerve, with increasing impairment of vision, rapid blindness having, under these circumstance, followed a newly-contracted marriage. Whilst open-air life is useful, the most scrupulous care should be exercised to avoid exposure to wet or cold, and, when it is possible to the patient, the winters should be passed in a warm, dry climate.

The effect of internal medication upon pure locomotor ataxia is very slight. Antisyphilitic treatment is of no value, even though the history of syphilis be very clear. It is true that medical literature abounds with reports of cases which seem to oppose this statement, but I have no doubt that in such cases the diagnosis has been incorrect. Minute doses of mercury—one-fortieth of a grain of corrosive sublimate—are believed by some writers to be of service. As in these minute doses mercury has a distinct tonic influence, it is allowable to employ it in locomotor ataxia with the hope, rather than the expectation, that it may have some influence upon the spinal lesion. The free use of mercury is distinctly contraindicated. Nitrate of silver was at one time very commonly employed: I have never seen it achieve any good, but the large history of its use justifies its employment by those therapeutists who have more respect for the statements of authorities than for the results obtained under their own eyes. Chloride of gold and chloride of barium, more recent remedies, are probably harmless when not given in too large doses. If phosphorus have any influence upon the spinal cord, it is that of causing nutritive excitement, and its administration would therefore appear to be contraindicated in tabes. Almost all of the nervine vegetable drugs have been given in tabes, but there is no reason for believing that any of them have a direct influence upon

the lesion. The active influence of ergot upon relaxed blood-vessels has led to its very free use in locomotor ataxia. There is, however, no sufficient reason for believing that the spinal lesions are in any degree due to a preceding relaxation of the blood-vessels. The effect of ergot in producing tabetic affections shows that the drug has some influence upon the nerve-centres, and justifies those who believe in the doctrine of *similia similibus curantur* in the use of it in small doses. I have myself seen it used frequently, and have never been able to perceive the slightest good effects from it. The favorable results which have been reported from it have usually been in the earlier stages of the disease, and are alleged to have been seen in the lessening of pain. These pain-symptoms, however, vary so greatly and so inscrutably in the individual case that not much importance seems to me to attach to any apparent improvement. Certainly in locomotor ataxia it is dangerous to give ergot in the enormous doses which have been employed by some practitioners.

My own belief is that the employment of drugs should be confined to the administration of harmless remedies, which should give to the patient, when necessary to be given, the moral support that comes to certain individuals from the feeling that something is being done; to the giving of tonics, laxatives, and other mild remedies from time to time as symptoms may call for them; and to the careful use of narcotics for the relief of pain. In severe crises hypodermic injections of morphine are often necessary, but the practitioner must never forget that the attempt to relieve the frequent attacks of sclerotic pain by opiates greatly endangers the formation of the opium habit. Antipyrin, antifebrin, and phenacetin certainly have a distinct controlling influence over nerve-storms even when due to such deep-seated cause as posterior sclerosis. They are much safer than opium, and I have seen them relieve a crisis which opium in moderate doses had failed to control. It is necessary, however, to carefully husband these remedies, so that their influence may not be worn out in the course of so long a disorder.

So far as the disease itself is concerned, I do not believe that any counter-irritation is of avail, although some practitioners claim to have had good results from the use of the actual cautery along the spine. In the very earliest stages of the disease this may be justifiable, but certainly in the later stages the amount of relief does not compensate for the suffering and distress involved in the treatment. In cases of crises repeated mild counter-irritations, in the form of sinapisms, over the seat of the pain and also over the root of the nerve supplying the affected part, are urgently called for by moral reasons. They sometimes seem to bring relief, and may therefore always be applied, care being taken to see that the application be not sufficiently severe to produce local destruction. Blisters must be employed under these circumstances with the greatest care, as there is danger of the local inflammation becoming uncontrollable. This is especially true when anæsthetic portions of the skin are involved. Any blisters or sores upon the feet should always receive the most careful attention, it being affirmed by competent authorities that a perforating ulcer has followed so small an operation as the cutting of a corn. In a crisis

the application of moist heat in the form of the warm bath and hot fomentation is often more successful than the counter-irritant.

It is very common for cases of locomotor ataxia to go to certain springs for treatment, notably in Europe to Aix-la-Chapelle and in this country to the Arkansas Hot Springs. I do not myself believe that there is any virtue whatever in the water, at least of our Arkansas springs. Most cases of true locomotor ataxia are not distinctly and permanently benefited, and whatever good is achieved is the result of the stimulating effect of travel and new scenes, to the freedom from care, rest, and to the hydrotherapeutic measures employed. Very many of the practitioners at these hot springs use mercury in enormous amounts, and old cases of syphilis, which perhaps have been mistaken for selerotic disease, are often benefited, but the cases of true sclerosis are not rarely greviously injured. I do not think that these waters act any differently from waters that have been heated artificially, and the home use of the warm bath or the warm pack is often of service in tabes by quieting the patient, reducing the amount of pain, and aiding rest and massage in bringing about relief. One, two, or even three warm baths or packs may be employed daily according to the strength of the patient. The temperature of the water should not be over 100° F.

In all cases it is essential that the bladder be thoroughly emptied at regular intervals. Even in the earlier stages micturition may be so imperfect that there is a residuary urine, which undergoes fermentation and sets up a cystitis, that, although slight, may yet be sufficient to gradually involve the ureters and the mucous membranes of the pelves of the kidneys, and finally the kidneys themselves. In this is probably found the cause of frequent death from kidney disease in locomotor ataxia. No hesitation should be felt, in any case, in using the soft catheter, and when the urine is ammoniacal the bladder should be washed out thoroughly every other day with a dilute solution of some antiseptic.

Very much has been claimed by certain writers for the use of electricity in locomotor ataxia, but I have myself never seen it do the slightest good. It has been the habit of certain electro-therapeutists to apply the galvanic current to the spinal column. Some authorities recommend that the current should be passed upward, some that it should be passed downward, the positive or negative pole, as the case may be, being placed at the nape of the neck, its antagonist at the end of the spine. I do not myself believe that under these circumstances any portion of the electrical current passes through the thick bony envelope and reaches the spinal cord. Every physiologist knows that the slightest electrical stimulation of the cord will produce muscular contraction, but no current that can be used for remedial purposes, applied in the manner just spoken of, ever provokes any evidences of spinal functional activity. For similar reasons I do not believe it possible to galvanize the cervical sympathetic ganglia, as has been recommended by various writers, in the treatment of this and other nervous diseases; moreover, there is no sufficient clinical or scientific reason for believing that galvanization of these gan-

glia, if possible, would do any good. The local use of the wire brush with a current of moderate strength is said to be sometimes useful in relieving the excessive numbness of locomotor ataxia.

Two surgical procedures have been proposed for the cure of tabes, and have received sufficient laudation to require notice here. The first of these, as proposed by Langenbach, consists of the stretching of the sciatic or of such other nerves as take their origin in the affected region. This procedure is so devoid of known scientific basis that I have myself never felt justified in using it; and, although a few cases of remarkable results have been reported, the almost universal consensus of medical opinion is that nerve-stretching in tabes is not justifiable. The operation has itself caused death, and has been followed by inflammation of the spinal cord.

In 1883, Motschoutkowski published a paper in the *Wratsch* proposing the use of suspension by the neck for the cure of tabes, and giving account of several cases in which the symptoms of ataxia had been in this way very much relieved. Motschoutkowski believed that there is an absolute stretching of the vertebral interspaces and a direct influence upon the cord, but in the present state of our physiological knowledge the use of suspension in tabes must rest absolutely on empirical basis, no probable theory being at hand to account for the value of the method, if indeed it have value. The subject did not attract wide attention until its exploitation by Charcot, whose reports were most favorable, it being claimed that out of 50 consecutive cases in his clinic treated by suspension, 38 were undoubtedly ameliorated. Thorough trials have now been made with suspension in every portion of the civilized globe, and certainly the published results do not coincide with the enthusiastic Parisian reports. Thus, of 75 English cases which I have collected from various papers, not one is recorded as having received any distinct benefit. The German reports are scarcely more favorable. In none of the 114 cases reported by Hirt was there any very distinct result. In Professor Mendel's clinic, out of 61 cases only 5 distinctly improved. On the other hand, 6 cases of death from suspension have been reported, the death sometimes taking place during the suspension, and sometimes occurring within the twenty-four hours afterward. Charcot teaches that œdema, obesity, phthisis, valvular or other cardiac lesions, emphysema, and marked atheroma of the arteries are contraindications to the use of suspension; and certainly when either of these exists the practitioner is not justified at all in experimenting with the method. Motschoutkowski practised suspension ten minutes three or four times a week; others have used it every few hours. It is probably best to give it once a day, from five to fifteen minutes. The best form of apparatus is probably that which is known in America as the Weir Mitchell. Space is wanting for its description. The practitioner should always see that the patient is suspended from a spring balance, so that an absolute knowledge of the amount of force used can be obtained.

Antero-lateral Sclerosis.

Definition.—A disease due to sclerosis of the antero-lateral columns of the spinal cord, characterized by stiffness of the affected parts, marked muscular contractions with partial loss of power, exaggerated reflexes, without pronounced disturbance of sensation and without trophic changes.

Synonyms.—Spastic paraplegia; Spastic spinal paralysis.

Etiology.—Antero-lateral sclerosis has relations with syphilis similar to those of locomotor ataxia; seems also to be produced by various poisons, notably lead; has been believed by some authorities to be the result of sexual excesses, and appears not rarely to be the outcome of excessive exposure with overwork—in a word, our present knowledge indicates that the causes which produce in one case posterior sclerosis, in another case bring on antero-lateral sclerosis, but give us no clue for deciding why in one case one region, in another case another region, of the cord is attacked.

Pathology.—In spastic spinal paralysis the lesion commonly found is sclerosis of the lateral columns, especially affecting the so-called cross pyramidal tract, and containing the fibres passing between the cerebral ganglia and the motor cells of the cord. The microscopical changes are similar to those which occur in sclerosis of the posterior columns.

Symptomatology.—Antero-lateral sclerosis is an essentially chronic disorder, which is commonly developed in middle life, though occasionally seen at either extreme of age. As the lower segments of the cord are usually first affected, the disease generally first reveals itself by a loss of endurance during walking and a peculiar stiffness and awkwardness of gait.

Even before there is distinct loss of power the patient will be troubled at night, especially after a hard day's march, with clonic or tonic spasms, which cause the legs to stiffen suddenly or to be jerked about. A little later the stiffness and loss of power combine to produce a very characteristic gait. The contractures of the various muscles prevent the bending of the joints of the knee and hip, whilst the great power of the muscles of the calf tends to draw the heel up and to thrust the toe downward. Consequently, the foot can be lifted from the ground, sufficiently to make a step, only by raising and rotating the pelvis, so that the body is inclined toward the leg upon which the patient rests during the step, whilst the moving foot is slowly thrust forward. The toes appear to stick to the ground, and are only with the greatest difficulty sufficiently raised to be pushed forward. The steps are of necessity very short, it may only be three or four inches. As the leg is put forward, not rarely violent tremblings affect it, and in some cases these movements are so rhythmical as to throw the heels of the patient up and down in regular vibrations. As the disease progresses the contractures of the muscles of the calf become so great that the heels are permanently drawn from the ground and the patient rests upon the toes. Under these circumstances the trunk is of necessity thrown forward, and is preserved from falling only by means of crutches or canes held well in advance of the body. A little later than this all power of locomotion

is lost, and not rarely the patient is confined to bed, or, if he attempt to sit, must be propped up in a chair with his feet supported in front of him.

When the power of locomotion is lost the leg is usually flexed upon the thigh, the heel drawn up, and the toes turned inward, these positions being due to the superior power of the posterior muscles of the thigh and leg and of the abductor muscles as compared with their antagonists. In some cases the patient lies with the legs stiffly extended, very rigid, the feet inverted and often crossed.

Tonic spasm is especially characteristic of spastic paraplegia, but very early in a case clonic spasm may occur, and even that form of violent clonic spasm may be present in which the legs are vibrated rapidly to and fro, and to which the very inappropriate and incorrect name of *spinal epilepsy* has been given by Brown-Séquard. This clonic spasm is especially apt to happen during the night, and is usually painless, but is sometimes accompanied with cramp-like sensations.

The functions of the sexual organs, the bladder, and the rectum are usually not implicated until very late in the disorder. Sensory symptoms are commonly not present, or at most are confined to slight dull pains or a feeling of weariness or slight numbness or paræsthesia. When rheumatoid pains with distinct disorder of sensibility occur, the probabilities are that a neuritis has been set up or that the disease has extended to other portions of the spinal cord.

There is no wasting of the muscles and no trophic changes in the joints or other part. For reasons which at present are not very apparent the lesion has little tendency to spread throughout the nervous system, as it does in locomotor ataxia, and hence ocular and laryngeal implications are rare, as is also mental confusion or insanity, although lateral sclerosis and general paralysis may coexist.

Very early in the disorder the reflexes, both superficial and deep, will be found involved, and their increase soon becomes excessive. The slightest tap upon the patellar tendon produces a quick and violent response, and it is usually possible to produce not only an ankle-, but also a knee-clonus; but in advanced stages of the disease the rigidity of the muscles may be so great as to in a measure mask the condition of the reflexes, the muscles being already in such violent spasm that no open effect follows further irritation. In rare instances the excitement of the reflexes dominates the condition of the muscles, which may be partially relaxed and quiet when the patient is in bed, but are instantly thrown into violent contraction by the touch of the floor during attempts to walk.

The course of spastic paraplegia is usually chronic, the disease generally continuing for many years and having little direct tendency to shorten life. Chronic kidney disease is much less frequent than in locomotor ataxia, because the bladder is so seldom implicated.

Diagnosis.—Although the recognition of the nature of a case of lateral sclerosis is usually easy, sometimes it is almost impossible. The diagnosis of

lateral sclerosis rests upon the slowness of the development of a gradual loss of power, which is accompanied by muscular contraction and heightened reflexes, and so situated as to be evidently of spinal origin, combined with the absence of girdle sensation, of pain, and of disturbance of sensation, of paralysis of bladder or rectum, of trophic changes, and of disorder of coordination.

The diseases which produce groups of symptoms more or less closely simulating lateral sclerosis are spinal meningitis, chronic cerebral disease with secondary degeneration, and hysteria.

Spinal meningitis is accompanied by excessive pain, and any attempt at the extension of the affected limbs produces suffering which is so much greater than that produced by similar procedures in lateral sclerosis that the diagnosis should always readily be made out.

Cerebral or secondary contractures, especially as seen in the disease known as spastic paralysis of childhood, are probably always due to degeneration of the antero-lateral motor tract of the brain and spinal cord, produced by a chronic inflammation having its origin in the seat of the original cerebral lesion. The lesions in lateral sclerosis and in descending degeneration following brain disease are so similar that of necessity the cerebral contractures must simulate those caused by antero-lateral sclerosis; but the distribution of the spastic paralysis differs in the two affections. Cerebral lesions are usually unilateral, spinal lesions usually bilateral. Except, therefore, in rare cases, contractures due to secondary degeneration are readily distinguished from those of the primary spinal disease by their being one-sided. It is true that in spinal sclerosis one side of the cord may be, at least in the early stages, more affected than the other, and that under these circumstances the symptoms are more pronounced upon one side than the other; nevertheless the opposite side does offer some manifestation of disease. In the diagnosis between secondary and primary contractures the history of the case also plays an important part. Spinal spastic paralysis always develops *slowly* and *insidiously;* secondary contractures almost always have followed an acute attack with cerebral symptoms or are accompanied by symptoms plainly of cerebral origin. When spastic paralysis dates back to birth, unless due to hereditary syphilis, it is probably always of cerebral origin.

The greatest difficulties of diagnosis are in the separation between hysterical and spinal contractures; indeed, it would appear that organic contractures may supervene upon the hysterical variety. Charcot reports the case of a woman in whom contractures of all four extremities developed suddenly and continued for ten years, with but few temporary remissions. After the last seizure the contractures remained until death, and at the autopsy symmetrical sclerosis of the lateral columns was found to extend almost the entire length of the cord. In one of my own cases contractures which had apparently been originally hysterical did not relax during anæthesia, and were accompanied with much atrophy of the affected muscle. In accordance with the rule laid down by Charcot, that whenever marked *atrophy of the muscles and persistence*

of the contractures during anæsthesia are present *organic degeneration* of the spinal cord has probably set in, the diagnosis in my case was lateral sclerosis following an originally hysterical contracture.

In opposition to this view Dr. Gowers asserts: "Nothing resembling it (spastic paraplegia) ever occurs in hysterical paraplegia. Hysterical contracture is fixed, and does not vary with posture, as does the 'clasp-knife rigidity' of spastic paraplegia. When the spasm is trifling or absent, as in slight and early cases, the diagnostic difficulty is much greater, and is increased by the fact that slight excess of myotatic irritability occurs in some cases of so-called hysterical paralysis. This, however, scarcely ever reaches the degree necessary to give rise to a true foot-clonus or a rectus-clonus. There may be a spurious foot-clonus, or a true clonus may be obtainable if there be hysterical contracture, but apart from such contracture a true foot-clonus or a rectus-clonus deserves the greatest weight as all but conclusive evidence of organic disease." These statements are, however, contradicted by my own clinical experience. I have seen a most severe clonus in hysterical paraplegia in which the symptoms were persistent for many months without relief, and in which the case was repeatedly shown at a public clinic, both by Professor C. K. Mills and myself, as one of sclerosis, but in which recovery occurred in the course of a week during the administration of subnitrate of bismuth. I am therefore convinced that a positive diagnosis between spastic paraplegia and hysterical paraplegia is not always possible without a history of the patient.

Usually, however, the hysterical disorder can be distinguished by the suddenness of its development, by the history of hysterical attacks in the past, by the presence of anæsthesia or other distinct hysterical symptoms, and by the sudden remissions of contractures. I do not believe that the hysterical contracture always relaxes during etherization.

Prognosis.—What was said about the prognosis of locomotor ataxia (see page 785) applies to antero-lateral sclerosis, except that this disorder is much less prone to compromise life.

Treatment.—The treatment of lateral sclerosis is precisely that of locomotor ataxia. It is true that less has been claimed for the results of mechanical treatment, "nerve-stretching and suspension," but I see no reason for believing that these procedures have more influence upon one spinal sclerosis than upon another.

Ataxic Paraplegia.

Definition.—A disease dependent upon a combined sclerosis of the posterior and lateral columns, and presenting during life mixed symptoms of locomotor ataxia and spastic paraplegia.

Etiology.—The etiology is that of spinal sclerosis.

Pathology.—Chronic spinal lesions are apt to overflow the tract from which they originally started or consentaneously to invade several portions of the cord. In this way arise atypical cases, offering during life contradictory symptoms of spinal disease. Although the combinations in chronic spinal

disease are in nature various, two types may be well described—ataxic paraplegia and amyotrophic lateral sclerosis.

Symptomatology.—Ataxic paraplegia usually comes on most insidiously, and commonly follows, in the general development of its symptoms, a course parallel to that of other spinal scleroses already described. According as the lesion affects chiefly one column or the other the symptoms of the locomotor ataxia predominate, or the lights and shadows of the clinical picture are chiefly those of the lateral sclerosis. Usually, the tendency of the reflexes to be lost, so strongly pronounced in locomotor ataxia, is overcome by the irritation of the lateral columns, and so there are present together a loss of power of endurance, with loss of co-ordination and preservation or even excitation of the reflexes. The sensory symptoms are commonly not so severe as in tabes; fulgurant pains are uncommon; the girdle sensation is not present in a majority of cases. Ocular disturbance may or may not exist. The gait is a curious mixture of that of spastic paralysis with that of posterior sclerosis.

Diagnosis.—Probably of all diseases hereditary ataxia gives symptoms most resembling ataxic paraplegia, but in the latter affection a nystagmus does not occur and the family history is wanting; moreover, the knee-jerk is very rarely lost.

Prognosis and Treatment.—The prognosis and treatment of this combined sclerosis are similar to those of locomotor ataxia.

Amyotrophic Lateral Sclerosis.

Definition.—A disease due to sclerosis of the pyramidal tracts and atrophy of the motor cells of the gray matter of the spinal cord, characterized by trophic degeneration of the affected muscles, with loss of power, muscular contractions, and exaggerated reflexes; without pronounced disturbance of sensation.

Etiology.—The causes of amyotrophic lateral sclerosis are very obscure. It does not appear to be as closely connected with syphilis as are some other forms of spinal sclerosis.

Pathology.—The structural changes in the spinal cord are those of lateral sclerosis and of poliomyelitis, the anterior or direct pyramidal tract being much more apt to suffer than in pure lateral sclerosis. Atrophy of the nuclei of the cranial nerves is extremely common. The relative extent of the lesion in various parts of the cord differs very greatly in different cases, in some the sclerosis, in others the cell-atrophy, predominating. There is, at present, no sufficient reason for believing that either one of these lesions is secondary to the other, although certain authorities do teach that the sclerosis is a secondary degeneration.

Symptomatology.—The symptoms of amyotrophic lateral sclerosis are a combination of those of poliomyelitis and lateral sclerosis—namely, wasting of the muscles with loss of power (poliomyelitis), spastic contractions, and heightened reflexes (lateral sclerosis). The relative preponderance of one or the other of the two sets of symptoms varies indefinitely in individual cases

according as to whether one lesion or the other predominates. The upper extremities are usually affected first, the symptoms of the early stages being a sense of tire, loss of endurance of effort, slow wasting of the muscles, and perhaps some scarcely perceptible stiffness. Occasionally a tendency to a hemiplegic arrangement of the symptoms is seen, and very commonly the trophic changes predominate in the arms, the spastic symptoms in the legs. The cranial nerves are usually affected very early, and the symptoms may closely simulate those of glosso-labial paralysis. Inability to whistle, difficulty of speech, fibrillary contractions, loss of power of retaining secretions in the mouth, and finally impairment of deglutition, occur. Amyotrophic lateral sclerosis is much more serious, so far as life is concerned, than other forms of sclerosis, death frequently occurring in two to four years from changes in the motor cells in the medulla involving the vital functions.

Diagnosis.—The recognition of the true nature of a typical case of amyotrophic lateral sclerosis is so easy as to need no further discussion here. It does, however, seem necessary to point out that there occur in nature all grades of lesions between the pure poliomyelitis and the pure lateral sclerosis, and that if the motor cells degenerate very rapidly the loss of muscle-tone may be sufficient to more or less completely mask the sclerosis of the white matter. Under these circumstances a slight stiffness of gait ("the frozen attitude") may alone reveal the true nature of the case. In a case of spastic bulbar paralysis the symptoms of bulbar poliomyelitis may be so closely simulated that the only evidence of the sclerosis is an increase of the jaw reflex.

Prognosis.—The prognosis is very unfavorable, the disease being very rarely if ever arrested, and death almost invariably resulting in from one to five years.

Treatment.—The only treatment of amyotrophic lateral sclerosis which seems to have the least chance of influencing the patient for good is long-continued rest in bed, with massage and careful nursing.

Friedreich's Ataxia.

Definition.—A disease which occurs in various members of the same family, dependent upon degeneration of the posterior and lateral columns, characterized by ataxic symptoms, nystagmus, contractures, and widespread paresis, with subordinate disorder of sensation.

Synonyms.—Hereditary ataxia; Family ataxia.

Etiology.—In the causation of so-called hereditary ataxia direct inheritance from parents very rarely appears, but in the great majority of cases the ancestors of the affected persons have suffered from various forms of nervous disease, so that the family stock is distinctly neuropathic. The importance of this is shown in the fact that there are on record only five or six isolated cases—*i. e.* cases in which only one member of the family was affected. Among the generally recognized causes of the disorder are either intemperance, tuberculosis, or syphilis occurring in the parent, and consanguineous marriage. Of these alleged causes, tuberculosis seems the most important. It is plain that

these various causations have only this in common—namely, a tendency to lessen in the offspring general vitality and the power of development of the various organs of the body. In not rare cases Friedreich's ataxia seems to have been precipitated by the occurrence of some acute disease, the symptoms having developed after typhoid fever, scarlatina, inflammatory rheumatism, diphtheria, etc. in a remarkable number of patients.

Morbid Anatomy.—The characteristic pathological changes of hereditary ataxia consist of sclerosis of the pyramidal tract and of the posterior columns. In almost all of the autopsies this sclerosis has extended the whole length of the cord, and in a majority of the cases it has involved the anterior pyramidal or direct cerebral tracts. It seems, however, not to have been traced up into the cerebrum itself. In a proportion of the cases a large part of the periphery of the lateral column, the so-called cerebellar tract, has been found sclerosed. The gray matter of the cord, especially the column of Clarke, is usually more or less degenerated, and indeed not rarely the nerve-fibres seem to be materially reduced in number throughout the whole cord.

We have no knowledge as to which portion of the nervous system is first affected, but it seems probable that no portion of the sclerosis can really be considered as secondary to other portions, the widespread changes being the result of a common cause. The nature of these changes remains in doubt, but there is reason for accepting as correct the original thought of Kahler and Pick, that the foundation of the affection is the imperfect development of certain fibres of the nervous system. There is appearance of truth in the further generalization of Pick, that this failure of development is due to early vascular degeneration, which naturally would especially and primarily affect the posterior columns of the cord, because this is the most vascular part of the cord. It must be stated, however, that recent observers[1] affirm that there is no alteration of the vessels, and that in this fact the lesion absolutely differs from that of true locomotor ataxia. Sclerosis and degeneration of the posterior nerve-roots were noted in all of the eight autopsies collected by Griffith—a fact which seems to negative the assertion of Dejerine, that Friedreich's disease separates itself from true locomotor ataxia in that the root-zones of the cord are not usually affected. Very few examinations of the peripheral nerves are on record, but Auscher states that whilst these nerves have not undergone degeneration, they are characterized by the presence of a considerable number of filaments without myelin—true embryonal nerve-tubes. If this be correct, it seems possible that the same condition may have been originally present in the spinal cords of cases of Friedreich's disease, and predisposed their subjects to the development of sclerotic lesions upon the slightest provocation. In this view of the pathology of the disease the fact that in so large a proportion of cases the symptoms have followed some acute infectious disease is very interesting. What is needed is microscopic examination of spinal cords taken from members of strongly affected families who have not themselves manifested the disease.

[1] *Compt. Rendus Soc. Biolog.*, 1890.

Symptomatology.—Friedreich's ataxia almost invariably appears during childhood. Out of the 143 cases tabulated by Crozer Griffith, about 30 per cent. developed the disease before the sixth year of age, about 60 per cent. before the tenth year, and only 3 per cent. between the twentieth and twenty-fifth years.

Usually the attack comes on insidiously, without prodromic symptoms, but eclampsia, vomiting, vertigo, curvature of the spine, flexion of the toes, palpitation of the heart, choreiform movements, and other evidences of irregular nervous disturbance have been noted. The first characteristic symptom is commonly a peculiar awkwardness of movement, which may develop directly in any portion of the body, although in the majority of cases it is first present in the legs. In rare cases speech and the lower and upper extremities have been simultaneously affected. A monoplegic and even a hemiplegic form of attack have been recorded.

In contrast with true locomotor ataxia the inco-ordination is not always increased by closure of the eyes. In the fully-formed case the gait varies: sometimes it resembles that exactly of true tabes; sometimes the aberration from the norm shows itself only in a strong tendency to the lateral projection of the foot; sometimes the walk is rolling like that of a drunken man. In the upper extremities the loss of co-ordination is evinced by irregular jerky movements and the inability to perform delicate acts. Late in the disorder inco-ordination often becomes so extreme that in the impossibility of properly apposing the fingers one to another the action of the hand resembles that of the paw of an animal. The peculiar condition which Friedreich designates the "*ataxia of quiet action*," and which he states to be characteristic of the disease and never present in true locomotor ataxia, is usually a rather late symptom, and is shown in the inability of the subject to hold the arm still in extension or in other quiet though somewhat forced positions. In its most advanced stage this "*static ataxia*" even produces peculiar athetoid symptoms in the fingers when lying in the lap, or a wavy or non-rhythmic oscillation of the arms and legs when at rest. It is very common in the head, causing a peculiar oscillation which is sometimes described as tremor or, when the oscillations are excessive, as choreiform movements. Sometimes these oscillations occur only under excitement, and simulate somewhat an intention tremor. Spasms and cramps are rarely present.

In hereditary ataxia the knee-jerk is usually abolished early in the history of the case. It is, however, not always absent, as it has been found normal in a number of reported cases, and in some cases which seem in all other respects to have represented the disease it has been exaggerated. The most probable explanation of these rare instances is that in them the lumbar enlargement of the spinal cord has not been involved. In some of the cases in which the knee-jerk has been found exaggerated ankle-clonus is asserted to have been present. The cutaneous reflexes escape in the majority of cases, but are occasionally diminished, and have been noted as increased.

Incontinence of urine is a very rare symptom, and the sexual organs often

long preserve their integrity. Initial muscular weakness is spoken of as a symptom, but probably in a majority of cases is apparent rather than real; however, in the advanced disease true loss of muscular power frequently occurs, and in a case reported by Vizioli it became so widespread and complete that the patient was almost reduced to immobility. Atrophy of the muscles is the exception rather than the rule, and, although the electro-contractility of the muscles is rarely disturbed, the reaction of degeneration has been noted. Muscular contractions are very frequent in the later stages of the disease, and not rarely give rise to deformities, such as curvature of the spine (which has been noted in about one-third of the cases reported), talipes equinus, and other forms of club-foot, besides various distortions of the limbs, toes, and fingers.

In a few cases of Friedreich's disease fulgurant pains like those of locomotor ataxia have been present as an early symptom, but in the majority of instances they are absent through the whole course of the disease, the only disturbance of sensation consisting of aching pains, slight numbness, and various paræsthesiæ. The numbness, formication, and tingling are rarely severe, and the girdle sensation has been noted only in a small proportion of the cases. Disorder of speech may be an early, but is usually a late, though an almost universal, symptom. It varies in form and intensity: sometimes the subject speaks with hesitation and a drawl, sometimes the words are thrown out in a jerky, almost stuttering manner, whilst typical scanning has been reported. Irregularity of pitch, indistinctness of utterance, slurring of the syllables, in various cases have indicated that the laryngeal muscles are affected. With these disturbances of speech other evidences of bulbar paralysis are often present. Evident lack of control in the movements of the tongue and lips, tremors, choreic or oscillating movements of the tongue, fibrillary contractions of all the muscles about the mouth, loss of power of holding the saliva in the mouth, with a loss of tone in the muscles of expression,—any or all of these symptoms may be present as the outcome of a deep-seated bulbar involvement.

Trophic changes of the joints and bones, such as appear in true tabes, have not as yet been noted in the hereditary affection. On the other hand, vasomotor disturbances, as shown by coldness of the extremities, blueness or lividity of the surface, and even by œdema, are not very infrequent.

The eye-symptoms are peculiar. Strabismus with diplopia sometimes occurs; blepharospasm with ptosis has been occasionally noted; but the characteristic though usually late manifestation of the disease is *nystagmus*. This may take the form of what Friedreich calls "*ataxic nystagmus*"—namely, oscillating movements appearing when the eyes are turned upon some object held near; or that of "*static nystagmus*"—that is, movements when the eyes are supposed to be at rest. The pupillary movements may be sluggish, but they are always present, and the Argyll-Robertson pupil has never been noticed. Atrophy of the optic nerve is rare. Vision is occasionally impaired, but contraction of the field has only been observed a few times. Color sense seems not to have been studied. It is not rare for the intellect to be dulled in cases of hereditary ataxia, but distinct mental aberration is very uncommon,

and there seems to be very little tendency in the disease to distinctly involve the cerebral hemispheres.

The course of hereditary ataxia is always slow, death in almost all cases having been produced by some intercurrent affection. Survival after forty years of illness has been noted.

Diagnosis.—The symptoms of Friedreich's disease are so diverse and so closely resemble those of other scleroses that in some isolated cases there may be a difficulty of recognition. Usually, however, the occurrence of several cases in one family, the subordination of the sensory to the motor symptoms, the static inco-ordination, with the subsequent oscillating or choreic movements, and the presence of disturbance of speech, with nystagmus, make the recognition of the nature of the disorder very easy.

Prognosis and Treatment.—The prognosis in Friedreich's disease is absolutely hopeless, and no known treatment is of any avail.

Progressive Muscular Atrophy.

Definition.—A disease due to a slow degeneration of the trophic or motor cells of the spinal cord, clinically characterized by slowly progressive atrophy of more or less restricted groups of muscles, with proportionate loss of power, and without changes in the electrical reactions until very late in the disorder.

Synonyms.—Wasting palsy; Cruveilhier's palsy.

Etiology.—Chronic spinal muscular atrophy is most frequent in males between the ages of twenty-five and fifty. Heredity, and especially indirect heredity, seems to have etiological influence in a minority of cases. Overwork, mental distress, exposure, traumatisms, and syphilis are all assigned as causes, but their action is very obscure.

Pathology.—The anatomical changes found after death from progressive muscular atrophy involve the muscles, the nerves, and the spinal cord. The wasted muscles are exceedingly pale in color and show under the microscope various alterations in their fibres. Four of these changes seem to be well defined: first, narrowing of the fibres, with slight changes in striation; second, fatty degeneration, in which the transverse striation gives way to a granular appearance, the granules increasing in size until at last the sheaths are simply occupied by fat-globules; third, "vitreous degeneration," in which the muscle-sheaths contain only a clear material very faintly striated and containing fat-globules; fourth, an apparently longitudinal splitting up of the fibres, with degeneration of the transverse striation, or in other cases with the appearance of the transverse striation much finer than normal, this condition being followed by the accumulation of fatty globules and wasting of the fibre. In all these cases the fibre is ultimately destroyed, the sheaths being left empty and shrunken, but clearly distinguishable from the interstitial fibrous tissue.

The peripheral nerves contain many fibres undergoing a degeneration which ends in total destruction of the nerve-filaments, leaving only their empty sheaths. This degeneration appears to affect earliest the anterior roots, where it is always exceedingly pronounced; the posterior roots are normal.

The spinal cord usually, if not always, shows two distinct degenerations. In old cases sclerosis of both direct and crossed pyramidal tracts is probably always present. When the amount is so great as to dominate the ganglionic change and to manifest itself during life by symptoms, the case represents so-called amyotrophic lateral sclerosis. In the gray matter the ganglionic cells are always affected. The cells waste, losing their processes, becoming globular or irregular in form, and afterward being reduced to little angular masses which by and by disappear entirely. In the foci of most advanced disease all traces of the ganglionic cells have disappeared. The nerve-fibrillæ, which are prolongations of the processes, are probably affected very early in the disease: they are found wasted, or more commonly do not exist at all, being replaced by the connective-tissue elements, prominent among which are small angular stellate cells. The larger vessels are often distended, the minute vessels not much changed. The degeneration of the ganglionic cells is in typical cases confined to the anterior cornua, the posterior cornua remaining normal.

The onset of progressive muscular atrophy is always very slow and insidious. In most cases, before any marked change can be noted in the muscle, the sufferer perceives a loss of endurance, so that the part tires easily, or there may even be absolute loss of power for short exertion. Careful examination will now show, even if there be no sensible wasting, that the muscle is softer and more flaccid than normal. Sensibility is not impaired.

A symptom which often precedes, although it varies very greatly in amount in various cases, any marked change in the volume of the muscle are fibrillary contractions. In their mildest form the fibrillary contractions consist of slight, irregular twitchings, occupying now this, now that portion of the belly of the muscle, and producing no effect except a corresponding movement of the skin over the contractions. In their severest manifestations the fibrillary contractions may amount to stormy peristaltic movements hurrying through the muscle one after the other in immediate repetition. When the fibrillary contractions are very severe the disease-process, at least in my experience, is rapid, the wasting of the muscle notably increasing from day to day under observation. In the slowest forms of the disease, in which many months or even years are required for much destruction, the fibrillary contractions are usually sluggish.

The loss of power takes the form of a multiple paralysis; that is, groups of muscles more or less isolated are attacked in different parts of the body. In the majority of cases the changes are somewhat symmetrical. Thus, if one region of the hand be attacked, the same region upon the other hand will be affected. This rule is not invariable, and even when the symmetry is decided, it may often be noted that not precisely the same muscles are affected upon the opposite side of the body. Although loss of endurance, or even partial paralysis, may apparently precede the loss of muscular substance, the loss of power is due to the loss of muscular substance, and not the loss of substance to the loss of power; or, perhaps more correctly, it may be considered that both

symptoms have a common basis—*i. e.* when a spinal ganglionic cell is attacked the fibres of the muscles individually supplied by it suffer simultaneously in their nutrition and in their motor functions. Usually the hands are the first portions of the body to be affected, the symptoms frequently being much more severe in the right hand.

According to Eulenberg, the interosseous muscles are almost invariably the first to be attacked, whilst Roberts, Wachsmuth, and Friedreich state that the ball of the thumb is usually implicated before the interosseous muscles. The first external interosseous is said to be the first to feel the influence of the disease, whilst the opponens and the adductor pollicis are more apt to suffer than the extensors, the abductors, and the flexors of the thumb. In the few cases in which I have had an opportunity to see the disease in its earliest stage the interosseous muscles were the first affected. The wasting of the muscles of the hand is usually readily perceived by the flattening of the thenar eminence and by the falling in of the interosseous spaces. The diminished power of the interosseous muscles can usually be detected by noticing that when the patient attempts to abduct the index finger he separates it with less vigor from the middle finger than normally. When only one hand is attacked the contrast of movement is often decided.

Instead of attacking the hand, progressive muscular atrophy may first make itself felt in other portions of the body, and especially is this true of the deltoid muscle; but it is stated that the pectoralis major, the serratus magnus, or even the lumbar muscles may have to bear the onset. The upper extremities, the neck, and the trunk are certainly much more frequently affected than are the legs; nevertheless, the latter do not always escape.

Owing to the loss of power in certain muscles and to the tendency to contractures in their antagonists, the sufferers from progressive muscular atrophy are prone to assume peculiar positions or to have extraordinary deformities. In a patient under my own care the loss of power in the muscles of the neck was so great that the head perpetually fell forward, the chin resting upon the breast. In this case the upper arms were much more prominently affected than were the forearms, so that whilst the man still preserved a good grip the arms were perfectly flaccid and helpless, owing to the complete paralysis of the deltoid, biceps, and triceps.

The most characteristic of the deformities is that which is known as the "*clawed hand*" (*main en griffe, Klauenhand*), and which is produced by the permanent flexion of the last two phalanges of the fingers, which are extended at the metacarpal joint. As was shown by Duchenne, this deformity is the result of atrophy of the internal and external interosseous muscles with the preservation of power by the extensors and flexors of the fingers. It must be remembered that this deformity is really pathognomonic of paralysis of the interosseous muscles, and is characteristic of progressive muscular atrophy only for the reason that loss of power of the interosseous muscle is rare from other causes. If, however, from local disease of the nerves the interosseous muscles are paralyzed, the clawed hand is developed. If only one hand be clawed, the

suspicion of local disease should be at once aroused. When the muscles about the shoulder-joint are paralyzed, either by sharing in the trophic changes or by the loss of the support of the muscles, the ligaments suffer elongation, and the joints become very loose, so that a subluxation readily occurs.

A very important symptom in the diagnosis of progressive muscular atrophy is the preservation of the electro-muscular contractility. This at first sight may appear to be at variance with the theory that the lesion in the muscle is the result of destruction of the trophic cells in the anterior cornua of the spinal cord. The explanation of the paradox, however, is simple. The destruction of the ganglionic cells progressively involves individual cells one after the other, and, consequently, the trophic destruction of the muscles compromises individual bundles of fibres one after the other. The muscle, therefore, loses power, not *en masse*, but fibre by fibre, and that portion of the muscle which retains its functional activity preserves its normal electrical reactions.

I have never seen the reaction of degeneration demonstrated in progressive muscular atrophy, although it is affirmed by Eulenberg that in the later period of the disease there may be qualitative alterations in the muscular reaction—*i. e.* an increased reaction under anodic closure and less commonly under cathodic opening. Eulenberg states that he has never seen in progressive muscular atrophy extreme degrees of qualitative deviation from the normal reaction. The so-called diplegic contractions which Remak has affirmed to be of frequent occurrence in progressive muscular atrophy are rarely to be demonstrated. The following paragraph from Eulenberg explains the method of developing these contractions:

"Remak found that the contractions could be produced in the atrophied muscles of the arm when the positive electrode was placed in an 'irritable zone,' which extends from the first to the fifth cervical vertebra, or, still better, in the carotid fossa or the triangle between the lower jaw and the external ear, while the negative was put below the fifth cervical vertebra. The contractions were always on the side opposite to the anode, but when the electrodes were applied in the median line they occurred on both sides. If the current was very weak they were limited to the muscles most severely affected. Remak regarded these as reflex contractions originating from the superior cervical ganglion of the sympathetic, and especially as the patient perceived a sensation behind the ball of the eye when the current was closed."

In some cases of progressive muscular atrophy the response to the faradic current appears more active than normal. This may in some instances be due to wasting of the muscle, enabling the current more rapidly and thoroughly to reach the portion of the muscle left; but it would seem that there is sometimes a heightened irritability of the muscular fibres which have not suffered degeneration; and I have thought that this was especially present when the fibrillary contractions were very severe. Again, in those cases in which the muscle as it wastes is replaced by fatty tissue the electro-muscular contractility may appear to be below normal on account of the resistance which the fatty matter offers to the faradic current.

The condition of the reflexes varies; there is, of course, no change in unaffected muscles. Probably owing to the sclerotic changes which affect the disease the myopathic irritability is very frequently increased. In advanced stages, however, it may be diminished or even lost.[1]

When the lesions of progressive muscular atrophy are situated high up in the spinal system the involvement of the muscles of the head and face gives origin to peculiar groups of symptoms, one or two of which have been described as distinct diseases.

Ophthalmoplegia progressiva of Von Graefe (*O. externa* of Hutchinson) may be caused by pressure on the nerve-trunks, but probably in the majority of cases is due to the lesion of progressive muscular atrophy attacking the nuclei of the affected muscles. In it all the external muscles of the two eyes are more or less completely paralyzed. If the palsy be nearly complete, there is marked drooping of the upper lid, with complete immobility of the eyeballs, giving rise to a very peculiar expression of the face. Usually the internal muscles of the eye are also implicated, but, according to Mr. Hutchinson, they occasionally escape.

Glosso-labial Paralysis, first described by Duchenne in 1861, is a progressive muscular atrophy in which the degeneration of the motor nuclei in the medulla—that is, in the upper physiological segment of the spinal cord—produces a progressive palsy of the tongue, lips, palate, and throat muscles. The degeneration of the nuclei of the medulla may accompany that of other spinal ganglia when the patient suffers from both progressive muscular atrophy and glosso-labial paralysis, or the bulbar nuclei may alone suffer when a pure glosso-labial palsy results. The mode of onset varies: frequently the paresis of the tongue is the first to appear, but the tremulousness and loss of the labial articulation may precede the lingual affection.

The course of the disease is entirely parallel with that of other forms of progressive muscular atrophy. There are the same progressive weakness, the same slow wasting, and the same fibrillary contractions in the affected muscles, with persistent retention of electro-contractility, as in other forms of progressive atrophy. The tongue is protruded more and more slowly and imperfectly, and becomes more and more tremulous. Owing to loss of control over it the pronunciation of the lingual vowels and of the dental consonants is imperfect. The weakness of the lips shows itself by failure in articulation of the labial consonants, by the inability to whistle, by tremulousness, and, finally, by the loss of the power to contain the saliva in the mouth, which dribbles constantly. As the disease is almost always symmetrical, the mouth is not drawn to one side, but the wasting of the parts about it may be sufficient to make the orifice appear much larger than normal and to confuse the naso-labial folds. Sometimes the lips during laughter separate themselves, but are incapable of spontaneously returning to their natural position, so that the patient is forced to

[1] It must be remembered that the separation of amyotrophic lateral sclerosis as a distinct disease is an artificial division, and that cases in nature grade up from those with least sclerotic changes to those in which the sclerotic changes are dominant over the ganglionic.

replace them with his fingers. If the palate be markedly affected, the voice becomes nasal. Deglutition may be affected early or late in the disorder, and, as the loss of power of swallowing is paralytic, liquids are swallowed with much difficulty and are apt to be returned through the nose. In some instances the larynx is attacked and the voice becomes almost inaudible, without, however, being completely lost. In those cases in which the nuclei of the respiratory nerves are implicated the respiratory muscles undergo wasting and the respiration becomes much affected. Any attempt at violent movement, or, later in the disease, even ordinary walking, may cause a severe attack of dyspnœa. At last these cyanotic crises comes on spontaneously in furious paroxysms, which may occur either by day or by night. A peculiar symptom which especially characterizes this dyspnœa is a sensation of excessive fulness of the chest, which is probably produced by the feebleness of the muscles preventing them from thoroughly emptying the lungs. In some cases the nuclei of the cardiac nerves appear to be attacked, and cardiac crises become violent and alarming. These are especially apt to be present in those persons in whom the respiration is affected, but may occur without the respiratory muscles suffering. The pulse in the cardiac crises is very feeble, irregular, intermittent, and at last may be imperceptible. The face is exceedingly pale and anxious, and there is habitually an intense terror, with a sense of impending death. The ocular muscles may be affected in glosso-labial paralysis, although they usually escape.

The "ophthalmoplegia externa" of Hutchinson is in some cases the expression of a progressive muscular atrophy.

Diagnosis.—The slow progression of the symptoms, the occurrence of atrophy before paralysis, the preservation of the electrical relations of the muscles, the absence of distinct disturbances of sensation and of pronounced tenderness, make the recognition of progressive muscular atrophy usually very easy. The only disease with which it can be confounded is pseudo-hypertrophic paralysis. This disease, however, belongs to childhood, and is usually attended with apparent increase in the size of some of the muscles. (For further details see Pseudo-hypertrophic Paralysis.)

Prognosis and Treatment.—The prognosis of progressive muscular atrophy is hopeless. The treatment is to be conducted upon general principles, with especial avoidance of muscular fatigue. We have no known agent capable of curing the degenerative lesion, but possibly the conjoint use of rest and massage may delay the process. Experience seems to show that the local treatment of the muscles by electricity, massage, etc. is of very little avail; it may, however, be essayed, care being taken not to overdo the matter.

DISEASES OF THE NERVES.

By WILLIAM OSLER.

NEURITIS.

Inflammation of the nerve-fibres may follow direct injury, disease of neighboring parts by extension (as in suppurative processes), exposure to cold, and is frequently due to toxic agents.

For convenience of description localized and general forms may be recognized.

Localized Neuritis.

Etiology.—(1) Injury is the most frequent cause, either direct laceration by fractures, gun-shot and other wounds, or bruising, as in prolonged pressure upon a nerve-trunk. In the subcutaneous injection of ether an intense neuritis may be excited by puncture of a nerve. A rare cause is the direct muscular compression of a nerve during sudden, violent muscular exertion. In certain occupations pressure on the nerves, as upon the ulnar in glass-workers, possibly, too, the constant straining of the muscles and of the nerves in repeated movements, as in rolling cigarettes, may cause local neuritis.

(2) Extension of inflammation from neighboring parts, particularly from disease of the bone. In otitis media the facial nerve may be involved, and in caries of the spine the intercostal nerves. Syphilitic disease is a less frequent cause. Tumors of various sorts, particularly cancer, may involve the nerve-trunks and produce an intense neuritis.

(3) Cold. The direct action of cold may cause neuritis, a common example of which is in the facial nerve after exposure to a draught of air, as from an open window of a railway-carriage. This is sometimes spoken of as a rheumatic form. Less commonly other nerves, such as the sciatic or branches of the brachial plexus, are affected by cold.

(4) Neuritis is not an infrequent accompaniment of joint-disease, particularly periarthritis of the shoulder, hip, and knee.

And lastly, the various toxic agents, which will be considered under Multiple Neuritis, though more frequently causing disseminated lesions, may act upon a single nerve or a single nerve-root.

Morbid Anatomy.—In acute neuritis the nerve-trunk is swollen, the sheath reddened and infiltrated, and in the more intense grades, due to extension of inflammation from carious bone, there may be a suppurative perineuritis. In the majority of examples the connective tissue uniting the nerve-

fibres is infiltrated with serum and leucocytes, the nuclei of the sheath of Schwann are increased, and there is marked fragmentation of the medulla. The axis-cylinders become varicose, and finally granular and disintegrated. In the final stage the nerve is represented by a very fatty connective tissue.

Symptoms.—There is no constitutional disturbance unless the process extends and involves many nerves. There is pain both in the part of the nerve-trunk involved and in the region to which it is distributed. There is sensitiveness on pressure, and the nerve-trunk may be felt to be enlarged. As Weir Mitchell states, the pain is in all probability due to the irritation of the nervi nervorum. In the region of distribution there are marked sensory disturbances, such as numbness, tingling, and often pain of a shooting or a stabbing character, which may radiate over adjacent nerve-territories. Movements of the muscles to which the nerve-fibres are distributed may be impaired, and occasionally there are twitchings or spasms. Trophic changes are sometimes seen, the temperature in the affected part may be raised, the skin is sometimes reddened, local sweating may occur; more serious changes are herpes zoster and arthritis. The duration of the symptoms is variable. A slight traumatic neuritis may pass away in a week; very frequently the process becomes chronic and persists for months. In the more chronic cases, such, for instance, as the neuritis of the brachial plexus after dislocation, the nerve-cords are swollen and painful, the pains persist for some time in the arm and hand, and the muscles gradually waste; in the more extreme cases the skin becomes reddened and glossy and there are trophic changes in the joints and in the finger-nails.

Although, as a rule, the symptoms of a localized neuritis are confined to the part of a nerve affected and its peripheral distribution, there are instances in which the process ascends the nerve, the so-called migrating neuritis; thus, after an injury to a finger in which the nerves are involved, sensitiveness and swelling of one of the nerve-trunks of the arm may supervene, and the process may even extend to the corresponding cord in the brachial plexus. Gowers thinks that the ascending neuritis may even reach the spinal cord and cause subacute or chronic myelitis, and that the so-called reflex paralysis in visceral disease is perhaps caused in this way.

The electrical changes in localized neuritis are variable, depending on the extent of the inflammation. If slight, the nerve and muscle reactions may be but little disturbed. In other cases the reaction of degeneration develops rapidly.

Multiple Neuritis (Peripheral Neuritis; Polyneuritis).

Etiology.—The cases may be classified as follows: 1, those in which the disease sets in after exposure to cold or follows exertion, the so-called idiopathic form; 2, toxic form, by far the most important variety, causing which the following poisons may be mentioned:

(*a*) *Diffusible Stimulants:* alcohol, carbon monoxide, carbon bisulphide, dinitro-benzine (roburite), aniline.

(*b*) *Metallic Poisons:* lead, arsenic, mercury, and phosphorus.
(*c*) *Animal Poisons:* diphtheria, typhus and other fevers, syphilis, tubercle, malaria, and leprosy.
(*d*) *Vegetable Poisons:* ergot, morphine, etc.
(*e*) *Endogenous Poisons:* rheumatism, gout, arthritis, diabetes, the puerperal state, chorea (James Ross).

3, dyscrasic form, in which the neuritis develops in the cachectic states, such as cancer, anæmia, and marasmus; 4, endemic neuritis or beri-beri.

Morbid Anatomy.—The lesions are those already described under Localized Neuritis. In a majority of the cases it is a degenerative process not associated with much connective-tissue or nuclear proliferation, and no superficial changes may be observed. The alterations are invariably more marked at the peripheral distribution of the fibres than in the trunk. The medulla is swollen, fragmented, and granular, and in extreme cases forms a molecular *débris*. The axis-cylinders also become granular and subdivided, and finally all trace of separation between the two essential constituents of the fibre becomes lost and the sheath of Schwann alone remains. The change may exist for a variable extent along a nerve, and may not be continuous, but interrupted. The nuclei of the sheath of Schwann in many cases show active proliferation, and they probably play a very important part in the regeneration of the fibres. In other cases interstitial changes exist with the parenchymatous degeneration. They are usually much more marked in the medium-sized and larger nerve-trunks.

Symptoms.—It will be best, perhaps, to describe certain well-characterized types, as the symptomatology of multiple neuritis is extremely complex.

Acute Febrile Polyneuritis.—The attack may begin acutely or subacutely after exposure or after prolonged exertion, occasionally during convalescence from an infectious disease. The affection may set in with a chill, followed by pain in the back and limbs, and moderate fever, reaching in some instances to 103° F. Headache, loss of appetite, and the general features of an acute infection are, as a rule, present. Pain, numbness, tingling, or hyperæsthesia is felt in the peripheral parts. Sometimes the pains are lancinating, and are usually more intense in the legs than in the arms. The nerve-trunks may be painful on pressure, particularly, according to Leyden, in the vicinity of the joints. By the second or third day loss of power is noticed, first in the legs, chiefly in the extensors, and in the course of a few days it extends, reaches the muscles of the thighs, attacks the arms, and within a week there may be widespread paralysis, which may even extend to the muscles of the thorax and to those of the face. The muscles may rapidly waste, and there is marked diminution of the faradic and galvanic contractility. The reflexes, as a rule, are abolished. Vaso-motor changes are not infrequent, such as congestion of the extremities and sweating. The clinical picture in many cases is that of an acute ascending paralysis. In the most intense forms death may occur within a week; more commonly not until the third or fourth week. In other instances the onset is more subacute and the disease does not run a rapid

course. There may be no fever, very little pain, and the gradual loss of power, with wasting, may give a picture very similar to the subacute spinal paralysis of Duchenne.

Alcoholic Neuritis.—This form, first recognized by James Jackson, Sr., of Boston in 1822, is the commonest type of the disease. It appears to be more frequent in women than in men, and follows prolonged drinking, either of beer or of spirits. The onset is marked, as a rule, by sensory symptoms, such as numbness and tingling in the hands and feet, and not infrequently painful cramps in the muscles. These symptoms are usually regarded as neuralgic, and the nature of the affection for a time is overlooked. There may be marked sensitiveness of the skin; more rarely areas of anæsthesia. The hands and feet are cold, sometimes a little swollen; not infrequently there is congestion or a blotchy redness, rarely urticaria. In women the condition of glossy skin is sometimes present in the hands and feet. Following these sensory symptoms, paralysis is usually noticed, beginning in the hands and feet, and attacking, as a rule, the extensors, causing foot- and wrist-drop. (See Fig. 55.) The invasion of the extremities may be simultaneous and progres-

FIG. 55.

Multiple Alcoholic Neuritis: palsy of extensors of wrist and flexors of ankle (Gowers).

sive, and in a few instances within ten or twelve days there may be complete paralysis of all extremities, and occasionally the muscles of the face, and even the sphincter muscles, are involved. Death may be caused by involvement of the muscles of respiration. The affected muscles are soft, waste rapidly, and, as a rule, the faradic irritability is early lost and the galvanic irritability is decreased. The superficial and deep reflexes are lost. In protracted cases the legs may be strongly flexed on the thighs and deformities produced in the feet; more rarely in the hands.

The cerebral symptoms of alcoholic neuritis are very remarkable. Convulsions may occur even at the onset or in the course of the disease. Mental symptoms are very commonly present—either delirium, which may be acute, or hallucinations, with extravagant ideas, resembling sometimes those of dementia paralytica. James Ross, who has specially studied the psychical disorders of multiple neuritis, divides them into four stages: first, a premonitory stage, in which the special senses and the imaginative faculties are exalted; second, a stage of depression or melancholia; third, a transition to mania or melancholia, with excitement, or of convulsions, passing on to, fourth, a final

stage of dementia. A special feature in certain instances is the complete loss of appreciation of time and place, and a patient describes long journeys which he has taken or tells of interviews which he has had with individuals. Eye-symptoms are rare, though there may be a remarkable hazy, turbid condition of the retina.

Post-febrile Neuritis—Under the various infectious disorders mention will be made of the forms of paralysis liable to occur during convalescence, a majority of which are now known to be due to involvement of the peripheral nerves. The most common and serious is the diphtheritic paralysis, which may be local, involving the nerves of the palate, eyes, or heart; or general, involving the nerves of the extremities. Following typhoid fever, small-pox, measles, and scarlet fever, a neuritis may be limited to the nerves of the leg, producing a paraplegia, or in some instances cause widespread loss of power and rapid wasting, so that the cases are very often regarded as acute or sub-acute myelitis. In rare instances neuritis occurs in malaria.

Some of the most characteristic features of leprosy are due to invasion of the peripheral nerves by the *bacillus leprœ*, a neuritis which is responsible in great part for the trophic changes and the anæsthesia.

A very important group of cases of neuritis depends upon poisoning by the metals, such as lead, arsenic, copper, and mercury. Lead palsy, the most common form, will be considered with Plumbism.

Special attention has been directed lately to the paralysis following poisoning by coal-gas from furnaces and illuminating gas. The effects may be limited to certain groups of nerves or may be widespread.

The cases grouped by Ross as due to endogenous poisons probably result from the action of toxic materials produced as a result of faulty metabolism. The most important is the diabetic neuritis. Very similar is the dyscrasic group of cases occurring in marasmus and the cachexias of various diseases.

Endemic Neuritis (*Beri-beri*).—In China, Japan, parts of Africa, and in the East and West Indies there is a widespread endemic disease which has been shown by Sheube and Baelz to be a peripheral neuritis. In mild cases there is slight difficulty in walking, associated with numbness, pains in the legs, and œdema. Anæmia is often present, with palpitation of the heart and general malnutrition. In the severer cases the paralysis is more extensive, and involves arms and legs, and even the trunk and face. Wasting proceeds rapidly and the patient becomes completely helpless. In the other instances, known as the "wet type" of the disease, with the paralysis there is great œdema with effusion into the serous sacs. General anasarca may completely cloak the muscular atrophy. And lastly, there is a pernicious form, in which the nerve-phenomena are not so striking, with gastro-intestinal symptoms, suppression of the urine, and signs of greatly enfeebled circulation; death may result within a few days. While but little doubt exists as to the marked involvement of the peripheral nerves in beri-beri, there is the greatest discord as to the exact causation of the disease: some attribute it to ankylostomiasis;

others to the eating of fish, more particularly, according to Miura, decomposed fish; others, again, to a vegetable diet, particularly rice. On the other hand, a micrococcus has been isolated from the disease, and cultures are stated to induce degeneration in the nerves in inoculated animals.

Cases of multiple neuritis have been reported by J. J. Putnam and others among the New England fishermen who frequent the Grand Banks. The prominent symptoms are œdema, shortness of breath, muscular paralysis, and sensory disturbances. The paralysis may be very extensive and prove fatal. Birge has reported an outbreak in one vessel with a crew of thirteen sailors, eleven of whom were attacked, two cases ending fatally.

Diagnosis.—In the majority of cases of multiple neuritis the mode of onset, the combination of sensory and motor symptoms, the involvement of the distal parts of the extremities, the tenderness of the nerve-trunks, and the wasting and sensitiveness of the muscles, suffice to render the diagnosis easy. In those forms which rapidly spread and involve all the extremities the diagnosis from acute poliomyelitis and from Landry's paralysis is sometimes difficult.

In anterior poliomyelitis the febrile onset, the rapid paralysis with wasting, and the electrical reactions afford no differential criteria. The sensory symptoms are, however, rarely present, and in multiple neuritis the paralysis is more symmetrical in its onset and as a rule develops more slowly. The sensory phenomena give the most important aid in the diagnosis of multiple neuritis, and when these are only transitory or present in a slight degree, the difficulty may be very great. In such instances the subsequent course of the disease usually affords grounds for separation; thus, in multiple neuritis, although the atrophy may be extreme and contractures present, recovery in time almost invariably occurs, whereas in poliomyelitis, though many of the muscle-groups may gradually recover, others remain permanently damaged. In the alcoholic cases the mental symptoms already described are very characteristic. The cases described by Duchenne under the term subacute diffuse anterior spinal paralysis (paralysie générale spinale antérieure subaigue) are now usually regarded as examples of polyneuritis. They occur without fever, following usually the infectious disorders, or result from poisoning by arsenic or lead. The sensory symptoms may be very slight, and cases are on record in which the face has been involved and also the sphincters. Every feature described by Duchenne may be present in these cases, and yet the general course of the affection and the ultimate recovery would indicate a peripheral lesion. In a case under Dejerine at the Bicêtre the first attack in 1880 came on without definite cause, and involved the extremities, the muscles of the eyes, the lower facial muscles, those of the tongue, and of the pharynx. After persisting for two years complete recovery took place. A second attack followed, and within three months the patient was completely paralyzed, and there were suffocative attacks, owing to the involvement of the muscles of the pharynx and larynx. The sensibility was unaffected. The reflexes were abolished and there was diminution of the contractility; gradual recovery took place in

about fifteen months. The distinction between multiple neuritis and Landry's paralysis will be considered under the latter affection.

The etiological factor is of the first importance in the diagnosis of many of the forms of polyneuritis, particularly those occurring in alcoholics, in the cases following fevers, and in those due to lead and arsenic.

From locomotor ataxia the diagnosis is easy. Unfortunately, the term *tabes* has been applied to several forms of partial paraplegia which are really of neural origin, such as the so-called arsenical tabes and diabetic tabes, or, as some call it, the sensory form of tabes. The pains in neuritis rarely have the lightning-like character, there is sensitiveness in the course of the nerves, and a combination of paralysis and atrophy, particularly in the extensors of the feet and toes. Romberg's symptom is not present, the inco-ordination is slight or not at all marked, and lastly the gait is entirely different; thus in the post-febrile neuritis and in the form due to lead the loss of power in the extensors of the feet and toes causes the leg to be lifted high in order to avoid the catching of the toes in consequence of the foot-drop. This *steppage* gait, as it is called by Charcot, is quite distinctive of peripheral neuritis, and is recognizable at a glance from that of ataxia, in which the foot is thrown up and out and brought down in a stamping manner.

Prognosis.—Danger to life exists only in those cases in which the paralysis extends rapidly and involves the muscles of respiration. In such instances death has occurred as early as the sixth day. Death occasionally occurs in alcoholic polyneuritis, and more frequently from complications than from the disease itself. The majority of cases of multiple neuritis of all forms recover, though it may be months before power is regained in all the muscles. Even with an extreme grade of atrophy with contractures it is remarkable how in time recovery may take place.

Treatment.—When the neuritis is widespread the patient is more comfortable upon a water-bed. The diet should be light and nutritious, and in the alcoholic cases special care must be exercised that the patient is not surreptitiously given beer or spirits. The fever of onset rarely demands treatment. In the cases following cold and exposure the salicylates may be tried. For the pains in the joints and limbs antifebrin or antipyrine may be given. In some instances morphine is necessary. Local applications are often very serviceable, particularly chloroform liniment. The thermo-cautery, lightly applied along the sensitive nerve-trunks, is sometimes of the greatest service. As the patients often suffer from cold, the limbs should be wrapped in cotton-wool, and it is advantageous when much congestion is present in the hands to keep them slightly raised on pillows. After the acute stage has passed arsenic and strychnia may be administered, either together or alternately. Systematic massage should at this time be thoroughly carried out, since it is probably the most serviceable of all measures in the paralysis and wasting of multiple neuritis. Electricity may be applied to the affected muscles, one pole placed over the trunk of the nerve, and the other over the muscle. The slowly interrupted current is the best. Care must be taken to prevent, if possible, contractures,

and when they exist they can usually with patience be overcome by passive movements and systematic rubbing.

Neuroma.

Properly speaking, this term should be applied only to growths containing nerve-substance, but it is applied somewhat indiscriminately to all tumors of the nerves. The distinction, however, may be made of true and false neuromata. The true contain either nerve-cells, *neuroma cellulare*, or nerve-fibres, which may be either medullated or non-medullated.

The *neuroma cellulare*, also known as the ganglionic or medullary neuroma, is an extremely rare form of growth met with occasionally in the basal ganglia and in other parts of the central nervous system, more rarely attached to the auditory or olfactory nerves. Certain forms of neuroglioma may resemble it very closely.

Tumors containing nerve-fibres are met with most frequently on the nerves of the skin and in the ends of the nerves in amputation stumps. The former constitute the small painful tumors which have been termed *tubercula dolorosa*, which sometimes occur in numbers about the joints, occasionally in the skin of the face or on the scrotum. Though painful to the touch, particularly at certain seasons, these are not always true neuromata, but may consist of firm connective tissue, and sometimes are adenomata of the sweat-glands.

The amputation neuromata, perhaps the most common, form ovid growths the size of peas or beans on the extremities of the nerve-trunks in a stump. They are made up of connective tissue and medullated or non-medullated nerve-fibres, and are sometimes extremely painful. Neuromata are occasionally met with on the nerves of the cauda equina and on the peripheral nerve-trunks, in which situation they may be felt as definite bead-like swellings.

The most remarkable variety is the *plexiform neuroma*, a congenital condition in which the nerve-cords in various parts of the body are the seat of tumor formations. In the remarkable case described by Prudden, the specimens of which are at the Medical Museum of Columbia College, New York, there were eleven hundred and thirty-two definite tumors on the various nerves of the body.

The false neuromata consist of nodular formations of connective tisssue—fibroma, more rarely myxoma, sarcoma, or lipoma.

There may be no symptoms even when the tumors are multiple. In other instances there is pain, which is often referred to the peripheral distribution of the nerve. The subcutaneous, painful tumor may be exquisitely sensitive, and when in a situation exposed to friction or to knocks exceedingly troublesome. The amputation neuromata often cause great pain in the stump. Muscular twitching may occur, and it is stated that in some instances epileptic seizures have been caused by them.

When painful the tumors may be excised. The amputation neuromata may recur after excision.

DISEASES OF THE CRANIAL NERVES.

Affections of the Olfactory Nerve.

The sense of smell may be lost or perverted, rarely increased.

Anosmia, loss of the sense of smell, may depend upon—

1. Involvement of the nerve-fibres in the mucous membrane. This, by far the most common cause, results from chronic nasal catarrh, the presence of polypi, and occasionally from strong irritants. Paralysis of the fifth nerve may be associated with loss of the sense of smell, owing to disturbance in the secretion and absence of the necessary moisture.

2. Lesions of the olfactory nerve and bulb, unilateral or bilateral, in fracture of the skull, caries, local meningitis, and tumors. A primary atrophy of the nerve is stated sometimes to occur in locomotor ataxia.

3. The loss of the sense of smell due to central disease is less common. The centre for the sense of smell is placed by Ferrier in the uncinate gyrus, and in a few instances the disturbance or loss of the sense has been noticed in connection with disease of this part. Occasionally the olfactory nerves and bulbs are congenitally defective.

Parosmia, or subjective sensations of a disagreeable nature, is met with most frequently in hysterical patients and in the insane. In epilepsy the aura may be olfactory in character, and the patient complains of an unpleasant odor, such as of the burning of rags, paper, or feathers. In other instances the parosmia exists alone in an apparently healthy individual: Morell Mackenzie mentions the case of a lady, aged about fifty, to whom the smell of cooked meat was so exactly like that of stinking fish that scarcely any animal food could be taken. After injury to the head the perversion of the smell may persist and odors of the most different character may appear alike.

Increased sensitiveness, or hyperosmia, is a rare condition met with occasionally in hysterical patients and in insanity. So acute may the sense become that individuals may be recognized by the odor alone.

The sense of smell may be tested by such substances as cloves, musk, and peppermint. In routine neurological work the sense should be tested systematically in brain cases, and it can readily be done by having small bottles filled with the essential oils. A careful rhinoscopic examination should be made in every case, as the disturbance of function not infrequently depends upon peripheral, not central, causes.

Affections of the Optic Nerve.

The lesions may be in the terminal expansion in the retina, in the optic nerve, at the chiasma, in the optic tract, or in the prolongation of the fibres within the brain and in the cortical centre for vision.

Lesions of the Retina.

(1) Retinitis.—The changes in the retina are of the greatest importance in diagnosis, and very valuable information may be obtained by the systematic

examination of this membrane, particularly in Bright's disease, leukæmia, anæmia, and syphilis. The chief changes are a cloudiness or turbidity due to the effusion of serum in the layers of the retina, hæmorrhages, which are in the layer of nerve-fibres and often follow the course of the vessels, and white spots or opacities. The hæmorrhages and opacities are the features commonly regarded as indicative of retinitis. When fresh they are bright red in color, but the effused blood gradually undergoes changes, and ultimately the spots become quite black. The opacities are due to inflammatory exudation, to fatty degeneration, or to sclerotic change.

The white spots also occur on the choroid as a result of atrophy of the pigment or the presence of new formations, particularly tubercles. Large areas of atrophy of pigment occur in certain cases of congenital syphilis. Tubercles in the choroid will be referred to in the section on Tuberculous Meningitis. The following are the more important forms:

Albuminuric Retinitis.—In chronic nephritis, particularly the interstitial form, a variable number of the cases—15 to 20 per cent.—present retinal changes. As disturbance of vision may be an early symptom, the diagnosis of Bright's disease is very frequently made by the oculist. The retinal arteries may be very small, and may be bordered by white lines, the result probably of perivascular changes. Small aneurismal dilatations may sometimes be seen. Gowers recognizes two forms of albuminuric retinitis—degenerative and inflammatory. The degenerative variety is characterized by small whitish spots, either punctiform or elongated, which are most abundant about the macula. Linear and flame-shaped hæmorrhages occur, and sometimes a diffuse opacity. In the inflammatory form there is much swelling of the retina and the arteries are obscured. Hæmorrhages are numerous.

There is a group of cases met with both in arterio-sclerosis and in chronic Bright's disease in which the optic nerve is chiefly involved, the disk being greatly swollen and striated, the vessels obscured, while the retina is either slightly involved or also presents hæmorrhages and signs of intense retinitis. When, as sometimes happens, this condition is associated with headache and transient aphasia, the diagnosis from brain tumor is very difficult. Among the complications of albuminuric retinitis may be mentioned hæmorrhage into the choroid, detachment of the retina, hæmorrhage into the vitreous, and embolism of the central artery.

A retinitis not unlike that of albuminuria also occurs in diabetes. In profound anæmia, in whatever way produced, retinal changes are common, chiefly in the form of hæmorrhages, more rarely as a neuro-retinitis. They occur particularly in pernicious anæmia, occasionally in chlorosis, in which the condition is more commonly a neuritis, and in malarial cachexia.

Leukæmic Retinitis.—A remarkable form occurs in leukæmia, usually in the splenic variety. There may be only a diffuse thickening and infiltration, with turbidity of the membrane or extensive hæmorrhage, but the most distinctive form is characterized by the presence of opaque white or yellowish-white spots, which may even resemble little tumors. So characteristic is this

condition that the diagnosis has been suggested by the ophthalmoscopic examination alone.

Hæmorrhages are also met with in purpura, scurvy, and in chronic lead-poisoning, and sometimes in association with suppression of the menses, more rarely with pregnancy.

(2) Functional Disturbance of the Retina.—*Toxic Amaurosis.*—In chronic Bright's disease sudden blindness may develop, and persist for some time without the existence of any retinal changes. This, known as uræmic amaurosis, is not necessarily associated with other toxic phenomena, and is usually a transient condition.

In cases of acute saturnism a similar amaurosis has been described. Toxic doses of quinine occasionally cause amaurosis. The amount, according to Gowers, which has caused the symptom has varied from the minimum of 80 grains in thirty hours to 1300 grains in three days. It is usually accompanied with loss of hearing. Recovery as a rule occurs, though it may be months before the vision is normal.

Tobacco amaurosis comes on slowly in both eyes, and presents a very characteristic effect in the centre of the field of vision—usually only dimness, not an actual loss of sight. The central defect or scotoma is much greater for colors. In a few instances the optic nerve has been involved and atrophy has developed.

Hysterical amaurosis may be complete, but more commonly involves only half the fields, causing hemianopia. The condition is usually transient, but may persist for months or even for years. In many cases there is marked restriction of the visual fields. Among other functional disturbances of the retina may be mentioned night-blindness (nyctalopia), a condition in which objects are clearly seen during the day, but become invisible in the shade or in twilight; and hemeralopia, in which objects can not be well seen in daylight or in strong artificial light. Hyperæsthesia of the retina is met with sometimes in hysteria, but is not a common symptom in inflammatory or degenerative conditions of the membrane. It may occur in connection with the intense throbbing of the retinal arteries in aortic insufficiency.

Lesions of the Optic Nerve.

Optic Neuritis (Papillitis).—In the early stage the edge of the disk is blurred and the rosy tint of the surface increased. Gradually swelling and opacity become evident, the physiological cupping disappears, there is marked striation, and hæmorrhages are present. The arteries may at first present very slight change, but later are narrow and the veins enlarged and congested. Very often the retina is simultaneously involved—neuro-retinitis. In mild grades the swelling of the disk gradually subsides and recovery may take place with but little damage. In other instances the swelling and exudation are very great, so that the disk is prominent and large, with marginal striation, hæmorrhages, and patches of inflammatory exudation. With the gradual subsidence of the swelling the nerve-elements undergo atrophy, and there is finally left the small pearly blue-white disk of optic atrophy. The neur-

itis may be chiefly retro-ocular, in which case the changes may be very slight at the papilla. In a variety of this form known as axial neuritis the central fibres of the nerve are chiefly involved.

Optic neuritis occurs most frequently in connection with intracranial disease, particularly tumors, more rarely in abscess. In a great majority of the cases it is bilateral. It is met with also in meningitis of the base, both simple and tuberculous. It occurs also in lead-poisoning, in anæmia, in chronic Bright's disease, usually as a neuro-retinitis, and there are cases known as idiopathic in which no etiological factors can be determined. A neuritis of an extreme grade may be present with very slight disturbance of vision, but gradually there is diminution of sight, which in the severe cases goes on to complete and permanent blindness.

The relation of optic neuritis to cerebral disease has been much discussed. It was formerly supposed that the swelling and hæmorrhage of the disk were largely due to increased intracranial pressure and obstructed return of blood from the eye. The term "choked disk," or *Stauungs-papille*, is an expression of this mechanical theory. Others think that the condition is produced by distension of the subvaginal space around the optic nerve, which has been shown to be continuous with, and can be injected from, the subdural space. A majority of observers now hold that the papillitis is always the result of a descending neuritis, the inflammation travelling down the course of the nerve-fibres. On no other theory, as Stephen Mackenzie has stated, can we explain the unilateral neuritis on the side opposite to a cerebral growth.

Optic Atrophy.—Following the papillitis, this is known as consecutive atrophy. Primary atrophy is met with in some instances as a remarkable hereditary affection which, as in Leber's case, affected all the males in a family shortly after puberty. A majority of the cases occur in connection with spinal disease, more particularly with locomotor ataxia, more rarely in general paresis of the insane, and in lateral sclerosis. Occasional causes are sexual excesses, migraine, syphilis, diabetes, and possibly tobacco, alcohol, and lead. The ophthalmoscopic appearances are different in the cases of primary and secondary atrophy. In the former the edges of the disk are well defined, the tint is of a steel-gray, the physiological cup is present, and the arteries look almost normal. In the consecutive atrophy the disk has a staring, opaque-white aspect, the outlines are often irregular, and the arteries are small.

The loss of sight in optic atrophy may vary from a very slight degree to complete blindness. Gradual contraction of the fields of vision takes place, and in a majority of the cases the color perception is altered. The primary defect as a rule is for green and red. In the cases in which there is a central scotoma it has been shown that the neuritis is axial.

The **prognosis** in optic atrophy is bad, particularly in the primary form. In the form following neuritis some vision is more commonly retained.

Lesions of the Chiasma and Tract.

At the commissure or chiasma the optic nerves undergo a partial decussa-

FIG. 56.

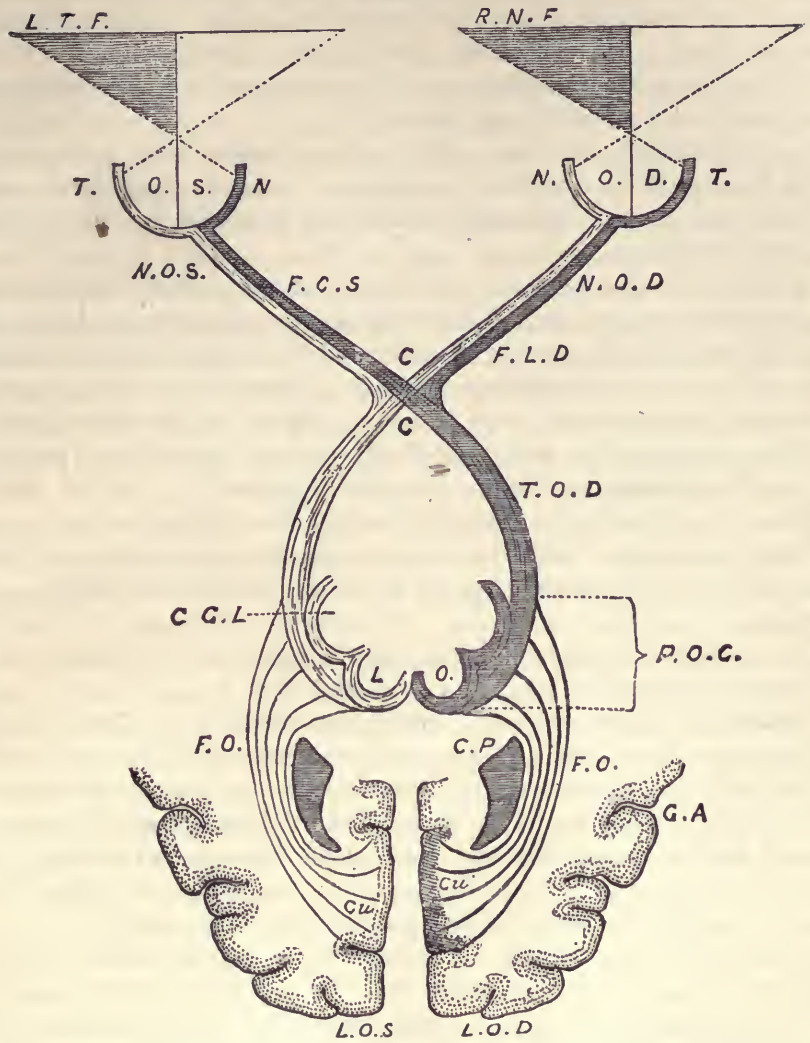


Diagram of Visual Paths, designed to illustrate specially left lateral hemianopsia from any lesion: *L. T. F.*, left temporal half-field; *R. N. F.*, right nasal half-field; *O. S.*, oculus sinister; *O. D.*, oculus dexter; *N. T.*, nasal and temporal halves of retinæ; *N. O. S.*, nervus opticus sinister; *N. O. D.*, nervus opticus dexter; *F. C. S.*, fasciculus cruciatus sinister; *F. L. D.*, fasciculus lateralis dexter; *C.*, chiasma, or decussation of fasciculi cruciati; *T. O. D.*, tractus opticus dexter; *C. G. L.*, corpus geniculatum laterale; *L. O.*, lobi optici (corpus quad.); *P. O. C.*, primary optic centres, including lobus opticus corp. genic. lat. and pulvinar of one side; *F. O.*, fasciculus opticus (Gratiolet) in the internal capsule; *C. P.*, cornu posterior; *G. A.*, region of gyrus angularis; *L. O. S.*, lobus occip. sinister; *L. O. D.*, lobus occip. dexter; *Cu.*, cuneus and subjacent gyri, constituting the cortical visual centre in man. (The heavy or shaded lines represent parts connected with the right halves of both retinæ.) (Seguin.)

tion. Each tract contains nerve-fibres which supply the temporal half of the retina on the same side and the nasal half of the opposite. The nasal or decussating fibres are more numerous and occupy the middle portion of the chiasma.

Lesion of one tract causes loss of function in the temporal half of the

retina on the same side and the nasal half of the retina on the other. If, for example, the right tract be involved, the patient has only half vision and is blind to objects on the left side. (See Fig. 56.) This condition is termed lateral or homonymous hemianopia. The hemianopia may be partial, only a section of the half field being lost.

Lesions of the Chiasma.—If the central portion alone is involved, in which the decussating fibres pass to the inner or nasal halves of the retinæ, there is loss of vision in the outer or temporal halves of the visual fields—temporal hemianopia.

A lesion limited to the outer part of the chiasma involves the direct fibres passing to the temporal half of the retina and causes blindness in the nasal field. If on both sides, there is bilateral nasal hemianopia. In a progressive lesion of the chiasma the different stages may often be traced from temporal hemianopia in one eye, then total blindness of that eye, then involvement of the fibres passing to the nasal side of the retina of the other eye, producing temporal hemianopia, and finally complete blindness.

When the left half of one field and the right half of another, or *vice versâ*, is blind, the condition produced is known as heteronymous hemianopia, in contradistinction to the homonymous, in which the blindness is in fields of the same side.

The accompanying figure illustrates the different forms of blindness resulting from involvement of the chiasma and tract.

Central Lesions of the Tract and Optic Nerve-fibres.—The tract divides at the hinder part of the optic thalamus, the larger portion entering the thalamus, the external geniculate body, and the anterior quadrigeminal body, from which fibres enter the occipital lobe through the hinder part of the internal capsule, forming the optic radiation which terminates in and about the cuneus. The fibres of the inner division of the tract pass to the internal geniculate body and to the posterior quadrigeminal body.

A lesion anywhere between the cortical centre and the chiasma will produce lateral hemianopia. The lesion may be situated either in the tract itself, in the region of the thalamus, in which case it is likely to be associated with hemianæsthesia (from involvement of the sensory tract in the hinder part of internal capsule) and sometimes with hemiplegia, or it may be in the fibres of the optic radiation within the occipital lobe; and, finally, at the centre in the occipital lobe in the neighborhood of the cuneus. It is possible that the different sections of the retina may be represented in different regions of the cuneus.

Color-vision is usually lost in the half field—hemiachromatopia—but in central disease the half field for color may be lost, while the field for white is intact.

The Significance of Hemianopia.—It is frequently a functional trouble, as in migraine and hysteria. In about half the cases there is hemiplegia, in some instances hemianæsthesia and aphasia. It is of great importance to determine, if possible, whether the lesion is in the optic tract alone or at the centres. This

can occasionally be done by a test devised by Wernicke, known as the *hemiopic pupillary inaction.* The reflex arc concerned in the contraction of the pupil consists of (1) the optic nerve-fibres, which receive and transmit the impression, (2) the nerve-centre in the geniculate bodies, which receives it and transmits it to the fibres of (3) the third nerve, along which the motor impulses pass to the iris. The integrity of this reflex arc is demonstrated by the contraction of the pupil when a bright light is thrown into the eye. In a case of lateral hemianopia the pencil of light may be so directed that it falls on the blind half, in which case, if the pupil react, the reflex arc above mentioned must be perfect; that is to say, there can be no interruption between the retina, the centre in the geniculate bodies, and the third nerve. In such a case the conclusion is justifiable that the lesion causing the hemianopia is situated behind the geniculate bodies, either in the fibres of the optic radiation or in the cortical centre. On the other hand, if when a light is thrown upon the hemianopic half of the retina the pupil remains inactive, the conclusion is justifiable that the path between the retina and the geniculate bodies is interrupted and the lesion causing the hemianopia is not central. Wernicke's test is not always easy to obtain. Seguin gives the following directions: "The patient being in a dark or nearly dark room with the lamp or gas-light behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus upon the eye, and note the size of the pupil. With my other hand I now throw a beam of light, focused from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye, then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all angles of incidence of the ray of light."

Affections of the Motor Nerves of the Eye.

Third Nerve.

This arises from a centre in the floor of the aqueduct of Sylvius, and, passing forward through the crus, at the side of which it emerges, it enters the orbit through the sphenoidal fissure, and supplies, by its superior branch, the levator palpebræ superioris and the superior rectus, and by its inferior branch the internal and inferior recti and the inferior oblique muscles. It also supplies the ciliary muscle and the constrictor of the iris.

Lesions of this nerve are of special importance in the diagnosis of disorders of the brain and spinal cord. The affections may be either of the centre or of the nerve in its course, and may cause either paralysis or spasm.

Paralysis.—A lesion of the nucleus may involve the centres for the other eye-muscles, causing general ophthalmoplegia, in which the power of movement of both the external and internal muscles of the eyeball is lost. The portion of the nucleus presiding over the iris may be involved alone, as in locomotor ataxia, causing loss of the reflex—the Argyll-Robertson pupil.

Much more frequently the nerve-trunk is involved in its course, either compressed in the exudation of meningitis by a gumma or an aneurism, lesions which may involve it either at the crus or where it enters the sphenoidal fissure. The nerve may be attacked by a neuritis, as in diphtheria and locomotor ataxia.

The following symptoms accompany complete paralysis of the nerve: loss of power in all the muscles of the eye except the superior oblique and the external rectus; the eye cannot be moved in any direction except outward and a little downward and inward; there is an external squint, owing to the unopposed action of the external rectus; the eyelid droops—ptosis—owing to paralysis of the levator palpebræ; the iris does not contract to light, the power of accommodation is lost, and the pupil is of medium size. The most striking features are the external strabismus, the double vision, and the ptosis. The affection of the nerve may be partial; thus paralysis may affect the branches passing to the superior, inferior, and internal recti muscles. The ciliary branches may be alone attacked, causing loss of the power of accommodation and paralysis of the iris.

Recurrent Paralysis of the Third Nerve.—There is a remarkable affection, met with chiefly in women, in which at intervals of a month or more the oculo-motor nerves are paralyzed. It may begin in early childhood, and has been known to continue for many years, the attacks occurring at longer or shorter intervals and lasting two or three days. They have been associated with headache and vomiting and symptoms resembling migraine. Twenty-three cases have been collected by Mary Sherwood from the literature.

Certain special features of third-nerve paralysis may here be considered:

Ptosis.—This common and important symptom occurs under the following conditions: (1) As a congenital defect which is sometimes hereditary and met with in many members of the same family. (2) From lesion of the third nerve, either at its nucleus or in its course. This may be associated with paralysis of the superior rectus or with the general features of third-nerve paralysis already mentioned. (3) There are instances in which with cerebral lesions the ptosis occurs alone, but the cortical centre has not yet been determined. (4) Hysterical ptosis is occasionally met with, and is readily recognized by its association with other hysterical manifestations. (5) Sympathetic or pseudo-ptosis is seen in paralysis of the cervical sympathetic, and appears to be due to loss of power in the fibres of Müller, which are innervated by the sympathetic and assist in keeping the upper lid in its proper position. With this form there are symptoms of vaso-motor disturbance, such as unilateral sweating, contraction of the pupil on the same side, and slight retraction of the eyeball. (6) In the facio-scapulo-humeral type of muscular atrophy, in which the facial muscles are affected, there may be bilateral ptosis. (7) And lastly there is a transient ptosis sometimes met with in delicate or neurasthenic women, particularly in the early morning hours. In exaggerated cases there may be great difficulty in lifting the eyelids. This is sometimes known as "morning" ptosis.

The symptoms of the greatest importance in third-nerve paralysis relate to the involvement of the ciliary muscle and iris.

Cycloplegia—paralysis of the ciliary muscle—causes loss of the power of accommodation, in consequence of which near objects cannot be clearly seen. It may be present in one or in both eyes. It is most commonly a symptom of nuclear disease, and is seen in diphtheria and in locomotor ataxia. It is of special value in the diagnosis of diphtheritic paralysis, in which it occurs with frequency and as a rule early.

Paralysis of the Iris.—*Iridoplegia.*—(1) *Accommodative Iridoplegia.*—In this the pupil does not diminish in size during the act of accommodation. To test it the patient should first look at a distant and then at a near object in the same line. Normally under these conditions the pupil contracts during the act of accommodation.

(2) *Reflex Iridoplegia.*—The path for the iris reflex is by the optic nerve and tract to the geniculate bodies, and then to the oculo-motor nucleus, along the trunk of the third nerve to the ciliary ganglia, and through the ciliary nerves to the iris. The eyes should be tested separately, and it is perhaps best to use an artificial light. The patient looks at a distant object in a dark room, so as to relax the accommodation completely; then a light is held at a distance of about four feet, and the state of the pupil is carefully watched. Loss of the iris reflex with retention of contraction on accommodation is known as the Argyll-Robertson pupil.

(3) *Loss of the Skin Reflex.*—Irritation of the skin, particularly that of the neck, is followed by dilatation of the pupil. As Erb has shown, this skin reflex is usually, but not necessarily, lost with the reflex contraction.

Iridoplegia is usually associated with small pupils; thus in locomotor ataxia the pupils are often much contracted—spinal myosis.

Inequality of the pupils—anisocoria—is met with not infrequently in general paralysis of the insane and in locomotor ataxia. It also occurs in healthy persons, and may persist for years.

Fourth Nerve.

The nucleus of this nerve is situated in the upper part of the floor of the fourth ventricle. Coursing around the crus in its passage to the orbit, it is liable to be involved in tumors, in the exudation of basilar meningitis, and may be compressed by aneurism. It supplies the superior oblique muscle. Nuclear paralysis is seen in connection with involvement of the centres of the other eye-muscles. Paralysis of the superior oblique causes defective downward and inward movement of the eyeball. There is double vision when the patient looks downward, which is obviated when the patient inclines the head forward and toward the sound side. The paralysis may be too slight to be noticed.

Sixth Nerve.

Arising from its nucleus in the floor of the fourth ventricle, it passes forward through the pons and emerges at the junction of the pons and medulla. Enter-

ing the orbit, it supplies the external rectus. The nerve is apt to be involved in tumors and meningeal morbid processes, and it is stated also to be sometimes paralyzed by cold. Paralysis of this nerve causes internal strabismus, and there is double vision on looking toward the paralyzed side. The defect in lesion of the nucleus is thus clearly and briefly described by Beevor: "When the nucleus is affected there is, in addition to paralysis of the external rectus, inability of the internal rectus of the opposite eye to turn that eye inward. As a consequence of this the axes of the eyes are kept parallel, and both are conjugately deviated to the opposite side, away from the side of the lesion. The reason of this is that the nucleus of the sixth nerve sends fibres up in the pons to that part of the nucleus of the opposite third nerve which supplies the internal rectus: we thus have paralysis of the internal rectus without the nucleus of the third nerve being involved, owing to its receiving its nervous impulses for parallel movement from the sixth nucleus of the opposite side. As the sixth nucleus is in such close proximity to the facial nerve in the substance of the pons, it is frequently found that the whole of the face on the same side is paralyzed, and gives the electrical reaction of degeneration, so that with a lesion of the *left* sixth nucleus there is conjugate deviation of both eyes to the *right*—*i. e.* paralysis of the left external and the right internal rectus, and sometimes complete paralysis of the *left* side of the face."

General Features of Paralysis of the Motor Nerves of the Eye.—Gowers recognizes five groups of symptoms:

1. *Limitation of Movement,* which is in proportion to the grade of the paralysis.

2. *Strabismus.*—In consequence of the paralysis the axes of the eyes do not correspond. Paralysis of the internal rectus causes a divergent squint—of the external rectus, a convergent squint. The deviation of the axis of the affected eye from parallelism with the other is known as the primary deviation.

3. *Secondary Deviation,* which depends upon the fact that when two muscles act together, if one is feeble and an effort is made to contract it, the increased innervation acts powerfully upon the healthy muscle, causing increased contraction. "Its existence and amount may be best ascertained by subsequently covering the paralyzed eye and making the patient fix with the unaffected eye, which, to do so, moves back to its former position. The hand or a piece of paper may be so placed as to intercept the vision of the one eye, while leaving it exposed to observation. A piece of ground glass placed over the eye answers the same purpose. The occurrence of secondary deviation depends on the fact that normally two muscles which act together are equally innervated for a given movement. If one is weak, and an effort is made to contract it (as in fixing with that eye), the increased innervation influences also the other muscle and causes an undue contraction. It is as if a rein acted equally on a hard-mouthed and a tender-mouthed horse yoked together; the effort to make the former deviate would cause an excessive deviation of the latter" (Gowers).

4. *Erroneous Projection.*—"We judge of the relation of external objects to each other by the relation of their images on the retina; we judge of their relation to our own body by the position of the eyeball as indicated to us by the innervation we give to the ocular muscles" (Gowers). If an object moves, we follow it with the eyes and judge of its position by the amount of movement. When one muscle is weak, the increased innervation "gives the impression of a greater movement of the eye than has really taken place, and suggests that the objects seen are farther on that side than they really are," and in attempting to touch it the finger goes beyond it. As equilibration in great part depends upon knowledge of the position and relation of external objects derived from action of the eye-muscles, the erroneous projection resulting from paralysis "destroys the harmony between the visual impressions and the others that are correct," and leads in this way to giddiness or ocular vertigo.

5. *Double Vision, or Diplopia.*—Owing to paralysis of the muscles the visual axes do not correspond, and there is a double vision: that seen by the sound eye is called the true, that by the paralyzed eye the false, image. When the false image is on the same side of the other as the eye by which it is seen, it is known as simple or homonymous diplopia, in which, for example, the right-hand image corresponds to the right eye, and the left-hand image to the left eye. In crossed diplopia the false image is on the other side; for example, the right-hand image belongs to the left eye, and the left-hand image to the right eye. The diplopia is simple in convergent squint, crossed in divergent squint.

Ophthalmoplegia.

Under this term is described a chronic progressive paralysis of the ocular muscles which may involve the external or internal groups alone or in combination, hence the names, "ophthalmoplegia externa" and "ophthalmoplegia interna." The condition is due to a degenerative change in the nuclei of the ocular nerves, and is described by Gowers as nuclear ocular palsy. In the external form the levators of the eyelids are usually first involved, then gradually the power is impaired in the other muscles, and finally the eyeballs become almost fixed, so that in order to view objects out of a straight line the patient has to move his head in a very characteristic manner. The eyelids droop and there is usually slight protrusion of the eyeballs. The affection is met with in association with general paralysis, locomotor ataxia, and sometimes in progressive muscular atrophy. Hutchinson regarded syphilis as the most important cause, but in the recent monograph of Siemerling it is stated that of the 62 cases on record, in only 11 could syphilis be definitely determined. Atrophy of the optic nerve and affections of the other cranial nerves are frequently associated with it. Mental disorders were present in 11 of the 62 cases analyzed by Siemerling. Bristowe has reported 2 cases in which the external ophthalmoplegia was probably functional.

Ophthalmoplegia interna is a term applied to slow progressive loss of power of the ciliary muscle and the iris. The condition may occur alone, but more

commonly is associated with the external form, and is then spoken of as total ophthalmoplegia. Possibly in some cases the internal form may depend upon disease of the ciliary ganglion. Although, as a rule, the ophthalmoplegia is a chronic process, there is an acute form which may lead to complete loss of power within ten or fourteen days, due to rapid softening of the nuclei of the ocular nerves. There are cerebral disturbances and sometimes ataxic symptoms. It was to this condition that Wernicke gave the name *polio-encephalitis superior acuta.*

Spasm of the Ocular Muscles.

In hysteria there may be an intermittent spasm causing rapid lateral movement of the eyes, with or without associated jerkings in other muscles of the body. In hysterical convulsions the eyes are usually drawn up, so that the corneæ are completely covered by the lids. In disease at the base of the brain, particularly meningitis, tonic, more rarely clonic, spasm may occur. The form known as conjugate deviation of the eyes, which is present in cerebral lesions, will be subsequently described.

The most remarkable form is the clonic rhythmical spasm known as nystagmus, in which the movements are bilateral and as a rule horizontal. When one-sided the movements are most frequently vertical. It occurs under very many conditions, particularly in congenital and acquired brain lesions associated with blindness, in albinism, in miners, and in many forms of sclerotic and chronic cerebro-spinal lesions, such as disseminated sclerosis and Friedreich's disease. The pathology of the condition is not yet well understood.

Spasm of the levator palpebræ is occasionally met with, and here may be mentioned the condition of *hippus,* or rhythmical contraction and dilatation of the iris.

Treatment of Ocular Palsies.—The paralysis due to diphtheria as a rule disappears with time and under a course of tonic treatment. When due to syphilis, iodide of potassium and mercury should be given, and the condition frequently improves rapidly under the use of these drugs. The forms associated with locomotor ataxia are the most obstinate and may resist all treatment. The group of cases due to chronic degenerative changes, as in progressive paresis or bulbar paralysis and the forms of ophthalmoplegia, are little if at all amenable to treatment. When there are acute symptoms hot fomentations, counter-irritation, or leeches may be used. The direct treatment of the paralyzed muscles by electricity is occasionally followed by good results, but in a large number of cases no special effect can be seen even after prolonged application. The diplopia may be relieved by the use of a prism. It is sometimes found necessary to cover the affected eye with an opaque glass. Various forms of spasm of the ocular muscles are little if at all affected by treatment.

Lesions of the Fifth Nerve.

We shall consider here paralysis, spasm, and neuralgia.

(1) Paralysis.—In comparison with the facial and other cranial nerves,

lesions of the trigeminus causing paralysis are rare. The nerve may be affected within the pons by hæmorrhage or tumors, rarely in chronic nuclear degeneration, which may be widespread without affecting the fifth nerve. At the base of the brain its position guards it, to a certain extent, from compression, but it is sometimes involved in fracture, caries, or meningitis. The branches may be affected as they pass to their distribution, the ophthalmic in the cavernous sinus by tumors or aneurisms, the superior and inferior maxillary branches by growths which invade the spheno-maxillary fossa.

Symptoms.—(*a*) *Sensory.*—When the whole nerve is involved there is loss of sensation in the skin of the corresponding side of the face and head, the conjunctiva, the mucous membrane of the lips, tongue, hard and soft palate, and of the nose on the same side. Tingling and numbness may precede the anæsthesia. The sense of smell is usually affected in consequence of dryness of the mucous membrane. Trophic changes sometimes occur, the salivary, lachrymal, and buccal secretions diminish, the gums may swell on the affected side, the teeth occasionally become loose, and abrasions of the mucosa tend to ulcerate. Herpes may develop about the eye or about the lips, and may be accompanied with much pain. The cornea may become opaque, and finally ulcerates. This is not, however, a constant sequence, and is absent unless the Gasserian ganglion is affected. Involvement of the individual branches of the sensory division causes loss of sensation in the skin and mucous surfaces upon which they are respectively distributed.

(*b*) *Motor.*—Inability to use the muscles of mastication on the affected side is the characteristic feature of paralysis of the motor division. It can be tested by asking the patient to close the jaw forcibly, when the temporal and masseter muscles on the affected side are not felt to contract or do so with great feebleness. Owing to involvement of the pterygoid, which cannot be moved toward the affected side in the act of chewing, the jaw when depressed deviates to the paralyzed side. Paralysis of the motor branches of the fifth nerve usually follows a lesion of the trunk. Occasionally the paralysis is due to cortical lesion, usually bilateral. Hirt has reported an instance of unilateral lesion, a psammoma involving the lower third of the ascending frontal convolution and the adjacent portions of the second and third frontal convolutions, associated with paralysis of the muscles of mastication.

Gustatory Symptoms.—The sense of taste is, as a rule, lost in the anterior two-thirds of the tongue on the affected side. The gustatory fibres pass from the chorda tympani to the lingual branch of the fifth. Loss of taste does not invariably follow paralysis of the fifth nerve. "Probably the exceptions are cases of partial disease or disease within the pons, where the taste-path has a separate course" (Gowers).

The **diagnosis** of trifacial paralysis rarely offers any special difficulties, the distribution of the anæsthesia and the loss of power in the muscles of mastication form such characteristic features. The preliminary pain and hyperæsthesia may be mistaken for neuralgia. The determination of the site of the lesion depends on the distribution of the anæsthesia and associated paralysis. When

the ophthalmic division is involved alone, the lesion is usually at the sphenoidal fissure or within the orbit. The lower divisions are not infrequently involved in tumors of the superior maxillary bone.

(2) Spasm of the Muscles of Mastication.—Trismus, or the masticatory spasm of Romberg, is often an associated feature in general convulsive attacks, sometimes an independent affection. The contractions may be either tonic or clonic. In the former the muscles of mastication are in firm contraction, so that the jaws are kept close together, the condition known as lockjaw, a symptom which occurs early in tetanus and is met with in some cases of tetany. Occasionally it is an hysterical manifestation. Less frequent causes are reflex irritation from the teeth and organic disease near the motor nucleus of the fifth nerve. Clonic spasm occurs either in a series of quick contractions, as in chattering of the teeth, or as forcible single contractions, which are sometimes seen in chorea and in hysteria.

Treatment.—For the organic lesions involving the fifth nerve little can be done beyond relieving the pain, which may require morphine. The preliminary irritation and hyperæsthesia are relieved by warm applications. If there be a history of syphilis, mercury and iodide of potassium may be given. Frictions and faradization of the affected side of the face are recommended.

(3) Neuralgia.—Neuralgia of the fifth nerve (prosopalgia; tic douloureux) is the most common and distressing of all painful affections of the nerves. All of the branches are rarely involved; most commonly the ophthalmic alone or the two upper divisions.

When the ophthalmic division is involved, the pain is referred to the distribution of the supraorbital branch, as a rule on one side only. There are tender points at the supraorbital notch, at the inner angle of the orbit, and sometimes on the nose at the junction of the cartilage with the bone. The pain is usually paroxysmal and may be of extreme severity. It is usually accompanied with intolerance of light, sometimes with spasm of the orbital muscles, lachrymation, and redness of the conjunctiva. The whole eyeball may ache or there may be an intense pain at the back of the eye. The pain extends over the brow and forehead, and the skin may be so tender that the patient may be unable to wear his hat. Owing to the paroxysmal character and the supposed association with malaria, neuralgia of this branch was formerly spoken of as "brow ague." The affection must not be mistaken for migraine, the painful symptoms of which are, however, due to involvement of this branch of the fifth nerve. It is to be remembered, too, in bilateral cases that errors of refraction may lie at the root of the whole mischief. Herpes may occasionally develop during an attack. Spasmodic contractions of the face-muscles on the affected side are occasionally present.

The superior maxillary is less frequently involved. There is a tender point at the infraorbital canal, and the pain is rather more concentrated and limited than in the neuralgia of the upper division, being chiefly along the upper teeth and gums. Salivation may occur with it. In inferior maxillary neuralgia there are painful spots along the auriculo-temporal nerve, and the pain radiates

about the ear and along the course of the inferior dental nerve. Tender points occur about the side of the head, particularly at the parietal eminence.

Trifacial neuralgia is most commonly met with in enfeebled subjects, particularly in women and in association with anæmia and chlorosis. There are instances in which it seems dependent upon malaria, but the malarial character in many of the cases has been attributed to the periodicity of the attacks. The cases vary extremely in their character and duration. There are instances in which the trophic and vaso-motor disturbances are particularly marked; thus the skin may become glossy and indurated and the subcutaneous fat may increase. Pigmentary changes sometimes occur on the skin, and the hair or the beard on the affected side may become gray.

The cases associated with spasmodic tic are sometimes of the most aggravated character, and the attacks occur with frightful intensity and render the patient's life unendurable.

Treatment.—Careful investigation should be made into possible sources of reflex irritation. Tonic and hygienic measures of all sorts should be utilized, as in very many cases neuralgia is, as has been expressed, the cry of a badly-nourished nervous system. A change of air will sometimes relieve a severe neuralgia, and even obstinate cases may yield to a prolonged residence in the mountains with an out-of-door life and plenty of exercise. Iron is often a specific in the cases associated with chlorosis and anæmia. Arsenic is also very beneficial in these forms, and should be given in full doses. Quinine, which is so much used, has probably no greater value in neuralgia than any other bitter tonic, except in the rare instances in which neuralgia is definitely associated with malarial poisoning. Strychnine, cod-liver oil, and phosphorus are sometimes useful. For the relief of the pain antifebrin and antipyrine may be tried, though their value has been much exaggerated. Morphine should be given with great caution, and only after other remedies have been tried in vain. Small doses given hypodermically are usually very efficacious, but on no consideration should a patient be allowed to use the hypodermic syringe. Gelsemium may be tried, and in frequent doses of the tincture is sometimes of value. Valerian, ammonia, ether, and above all alcohol, sometimes allay the pain. The last-named remedy should be used with the greatest caution, particularly in women. The pleasant, soothing effect of it in many cases of neuralgia has been the starting-point of habits which have finally enslaved the patient. Nitro-glycerin in full doses is a remedy which is sometimes efficacious, particularly in the chronic cases. Of local applications, liniments of belladonna, chloroform, and menthol, the ointments of aconitine and veratrine, and counter-irritation with the thermo-cautery or small blisters over the painful points, may be tried. Electricity is often of much service, particularly the continuous current, and when frequently repeated is very soothing.

Lesions of the Facial Nerve.

Paralysis (Bell's Palsy) may be due to—(1) involvement of the nerve-fibres from the cortex cerebri to the nucleus in the medulla; (2) to

lesions of the nucleus itself; and (3) to peripheral lesions involving the nerve-trunk in its tortuous course within the pons and through the wall of the skull or in its course after leaving the styloid foramen.

(1) Facial Paralysis of Cerebral Origin.—This, also known as the supranuclear form, may be due to a lesion of the cortical centre presiding over the lower facial muscles which is situated in the lower part of the ascending frontal convolution. Cases of limited lesion involving the facial centre alone and causing facial hemiplegia are rare; more commonly on the left side the speech-centres are also involved and the centres for the hand and arm. Softening from arterio-sclerosis, tumors, and localized meningitis, tuberculous or syphilitic, are the common causes of cortical facial palsy. The fibres may be involved between the cortical centres and the nucleus in the medulla, and with them, as a rule, the motor fibres of the arm and leg, so that the facial palsy is part of a hemiplegia.

The supranuclear facial paralysis is distinguished from the peripheral form by several well-marked features. The orbicularis palpebrarum and frontalis muscles are not involved, so that, for instance, in hemiplegia the patient can close the eye and frown on the paralyzed side. While voluntary movements are lost in the paralyzed muscles, during emotion, as in smiling, the paralyzed muscles may be moved, which is never the case in the peripheral form. Another difference of great importance is the persistence of the normal electrical excitability of both nerves and muscles. In rare instances of hemiplegia the orbicularis palpebrarum is involved in association, it is said, with lesion of the lenticular nucleus. Broadbent explains the immunity of the upper facial muscles in hemiplegia by the fact that the bilateral movements of the body, such as those of the eyes and trunk and the larynx, are represented in both hemispheres; that is to say, either hemisphere can excite bilateral movements.

(2) Nuclear Facial Paralysis.—The facial nucleus forms a group of large ganglion-cells, occupying that portion of the gray substance of the fasciculus teres which lies immediately behind the nucleus of the sixth nerve in the floor of the fourth ventricle.

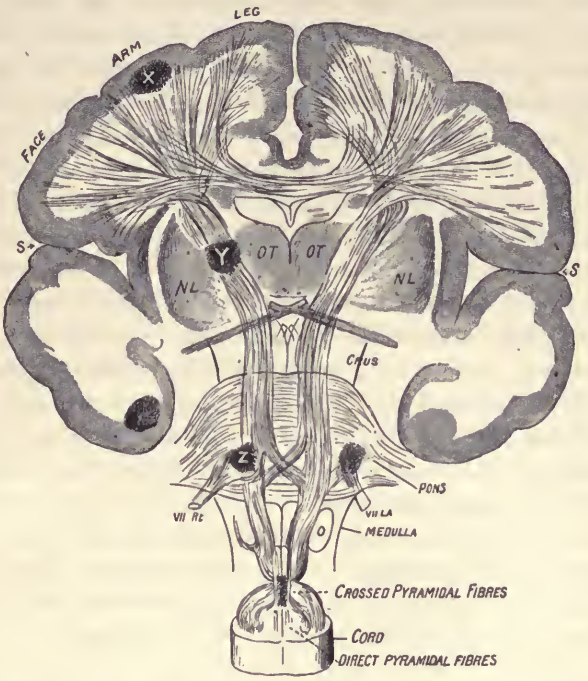
The nuclei are rarely attacked alone, but may be in tumors, hæmorrhage, and softening; more rarely in acute poliomyelitis. In lesions in the neighborhood of the pons the facial nucleus on one side, that of the sixth nerve too, and the motor path may be involved, producing facial paralysis on the same side as the lesion and paralysis of the arm and leg on the opposite side—a condition known as crossed paralysis. (See Fig. 57.) The symptoms of facial paralysis of nuclear origin are identical with those of involvement of the nerve itself. The superior facial muscles are involved and the electrical changes are present.

(3) Paralysis from Involvement of the Nerve-trunk.—The nerve may be involved as it passes through the pons from the nucleus, at the base of the skull, in its prolonged course through the temporal bone, or at its point of emergence. In the pons the fibres may be affected between the

nuclei in the floor of the fourth ventricle and the point of emergence of the nerve, in which case there may be an alternating or crossed paralysis, in which the face on the same side and the arm and leg on the opposite side are paralyzed. This only occurs when the lesion is in the lower region of the pons.

At the base of the brain the nerve is liable to be compressed by meningeal exudation or tumors, and is occasionally torn in fractures. Within the temporal bone the nerve is frequently attacked in otitis media. At the styloid

FIG. 57.



Motor Tract (after Starr): *S*, fissure of Sylvius; *NL*, lenticular nucleus; *OT*, optic thalamus; *O*, olivary body. The tracts for the face, arm, and leg gather in the capsule, and pass together to the lower pons where the face-fibres cross to the opposite seventh nerve nucleus, while the others pass on to the lower medulla, where they partially decussate to enter the lateral columns of the cord; the non-decussating fibres pass to the anterior median columns. The effect of a lesion situated at three points in the tract is shown on the left side of the figure at *X*, *Y*, *Z*. At *Z* the lesion would involve the left facial nerve and the left pyramidal tract above the decussation, producing facial paralysis on the left side and paralysis of the arm and leg on the opposite side—crossed paralysis.

foramen the nerve may be involved in blows, injuries, as by the pressure of forceps in an instrumental delivery, and the nerve may be cut in the removal of tumors in the parotid region. The most common cause is exposure to cold, particularly to a draught when riding in a carriage with the window down. This is usually attributed to a neuritis of the nerve in the Fallopian aqueduct.

Symptoms.—The onset is, as a rule, abrupt, and is not often preceded by pain or discomfort. In the cases following exposure to cold it is a common history to find that the patient wakes in the morning to find the face paralyzed. In the peripheral form all the branches are involved, and the face on the affected side is immobile and can neither be moved at will nor does it join

in any emotional movements. When at rest it is seen that the skin on the affected side is smooth and the wrinkles are effaced. This, of course, is not so noticeable in young persons. The angle of the mouth is somewhat lowered and the naso-labial fold is not so marked. The lower lid may droop a little and the eye waters. The eye on the affected side cannot be closed voluntarily. In smiling or laughing the angle of the mouth is drawn to the healthy side, while on the paralyzed side the lips remain in contact. The forehead cannot be wrinkled, nor can the patient show his upper teeth on the affected side, nor can he whistle. In sniffing there is no movement of the nostril on the paralyzed side. In speaking the pronunciation of labial sounds may be slightly impaired. Owing to the paralysis of the buccinator, food collects between the cheek and the jaw on the affected side; the tongue when protruded looks as if it were pushed to the paralyzed side, but if its position is estimated from the incisor teeth, it will be found to be in the middle line. It is usually stated that the soft palate and uvula are paralyzed on the same side, but this is denied by both Gowers and Hughlings Jackson, and, according to Horsley and Beevor, the levator palati is innervated by the accessory nerve. When the paralysis is due to an intratemporal lesion, there is, in addition to the above symptoms, loss of the sense of taste in the anterior part of the tongue on the affected side, owing to paralysis of the chorda tympani nerve.

Disturbance of hearing may exist with facial paralysis, and is usually due to extensive otitis media. There may be increased sensitiveness to sounds, owing to paralysis of the stapedius, which diminishes the amplitude of the vibration of the tympanic membrane. Tinnitus may be present.

Among other symptoms occasionally present in facial paralysis may be mentioned herpes and neuralgic pains. The electrical reactions in Bell's palsy are those of a peripheral paralysis. The nerve rapidly loses both faradic and galvanic excitability, which may completely disappear within ten days.

Erb gives the following rules: The prognosis is good, and recovery occurs in from fifteen to twenty days if there is no change, either faradic or galvanic. If the faradic and galvanic excitability of the nerve is only lessened and that of the muscle increased to the galvanic current, and the contraction formula altered (the contraction sluggish, ACC>CCC) the outlook is favorable, and recovery will probably take place in from four to six weeks or may be delayed for from eight to ten weeks. If the faradic and galvanic excitability of the nerves and the faradic excitability of the muscles are lost, and the galvanic excitability of the muscle quantitatively increased and qualitatively changed (reaction of degeneration), and if the mechanical excitability is altered, the prognosis is relatively unfavorable, and recovery may not take place for from two to eight months, or may even be delayed for as long as twelve or fifteen months.

The duration of the paralysis is variable. Recovery usually follows the paralysis from cold, though it may be delayed for months. In the traumatic cases recovery is possible, but the loss of power in these may be permanent.

When due to syphilis recovery is common. In the cases due to middle-ear disease the outlook is less favorable.

In any case the electrical reaction gives the most valuable indications upon which the prognosis can be based. When the paralysis is permanent, the muscles are toneless and there are no contractures. Spontaneous twitchings may be noticed at times in the muscles. In these late cases without any improvement there may be contractures in the muscles, drawing the mouth toward the paralyzed side, and the wrinkles may again appear, and in looking at the face the first impression may be that the affected side is the sound one, but this is soon corrected by asking the patient to smile, when it is seen which side of the face has the active movement.

Diagnosis.—The existence of facial paralysis is usually determined at a glance. The diagnosis of the site is sometimes difficult. The following résumé may be given:

(1) Paralysis due to a lesion of the nerve outside the stylo-mastoid foramen involves both the upper and lower divisions. All reflex movements are lost and the reaction of degeneration is present.

(2) When due to lesion within the Fallopian canal the features are the same as those just mentioned, and there are, in addition, alterations in the sense of taste and increased sensitiveness in hearing.

(3) A nuclear lesion produces a paralysis similar in distribution to the peripheral form. There may be crossed paralysis, involvement of the facial on one side and of the arm and leg on the other, and the sixth nerve on the same side is usually involved, causing internal strabismus.

(4) The facial palsy of cerebral origin (supranuclear) involves, as a rule, only the lower facial muscles, so that the patient can elevate the eyebrows and close the eye. The reflexes are preserved, and there is not the reaction of degeneration. If due to involvement of the fibres in the cortico-bulbar motor path, there is usually hemiplegia and the paralysis of the face and of the limbs is on the same side. The paralysis due to a cortical lesion may be a monoplegia confined to the facial muscles. On the left side it may be accompanied with aphasia, and in some instances the arm-centres are also involved.

Treatment.—In the so-called rheumatic cases hot applications may be made, but the disease rarely seems to be progressive, and the damage is done before any palliative treatment can be adopted. The thermo-cautery may be lightly applied at intervals over the mastoid region and over the course of the nerve. This is much more satisfactory and very much less painful than blistering. Iodide of potassium should be given internally, and in increasing doses, if syphilis be suspected. Subsequently the galvanic current should be systematically employed, and persevered with so long as there is any reaction, as when this is present there is always a prospect of recovery. The positive pole may be placed behind the ear and the negative pole passed over the zygomatic and other muscles. The application should be made daily for from ten to fifteen minutes. With the electricity may be combined massage of the muscles of the face.

Spasm of Facial Muscles (Mimic Spasm; Convulsive Tic).—The contraction, which is usually clonic, may be limited to certain groups of muscles or involve all those of one side; occasionally it is bilateral. Various groups of cases may be recognized:

(*a*) The secondary form following paralysis, and consisting in spontaneous clonic twitchings recurring at irregular intervals in the paralyzed muscles.

(*b*) Cases due to the irritation of an organic brain lesion, either of the cortex, as in a case of Berkley's in which a lesion of the ascending frontal convolution caused persistent clonic spasm limited to the zygomatic muscle, or pressure on the facial nerve by a new growth or an aneurism at the base of the brain.

(*c*) In many cases, particularly in adults, no cause can be assigned. This, which Gowers calls the idiopathic form, is most frequent in females, and follows in some instances mental anxiety and shock.

(*d*) Cases which appear to have a reflex origin, and which are associated with irritation in branches of the fifth nerve, as in eye-strain, diseased teeth, and naso-pharyngeal disorders. Some have attributed the affection in children to the irritation of worms. Cases due to these causes are much more common in children, in whom the spasm, known also as habit spasm and by some as habit chorea, may be limited to the facial muscles, but may be met with in other groups.

Lastly, facial spasm may form a part of the affection described by the the French as *tic convulsif* or as Gilles de la Tourette's disease, which is characterized by involuntary spasmodic jerkings in certain muscle-groups, particularly those of the face, explosive utterances, often bad language—coprolalia—and fixed ideas, such as arithmomania.

Symptoms.—The contractions are usually of a rapid, electric-like character, sometimes a series of quick, quivering contractions. The orbiculares oculorum are most frequently involved, causing a form known as the nictitating and blepharospasm, in which the eye is closed with lightning-like rapidity. In some cases, when both sides are affected, the patient can scarcely see, owing to the constantly-recurring contraction. More frequently the lateral facial muscles are also involved, and there is constant twitching of the side of the face, with partial closure of the eye. The frontalis muscle is not often involved. In severe cases the depressors of the angle of the mouth, the levator menti, and the plastysma myoides are affected. Occasionally the muscles of the tongue, which is protruded quickly as the patient talks, and occasionally the muscles of the palate and uvula, are involved. The contractions are aggravated by emotion and reduced by rest and quiet. There is no loss of power in the muscles and no pain. Tender points are sometimes found in the course of the fifth nerve, particularly in the supraorbital branch. The spasm occasionally extends from the face to the muscles of the neck and arms.

The outlook is favorable in the cases in which a source of definite irritation can be traced. The idiopathic cases coming on in the middle life in women are as a rule incurable.

Treatment.—In children the sources of reflex irritation should be carefully sought for. Eye-strain should be excluded, the naso-pharynx explored, and decayed teeth removed or filled. When tender spots exist along the fifth nerve, small blisters may be applied or the thermo-cautery. Electricity, which is given in nearly all cases a thorough trial, rarely proves successful. Hypodermics of strychnine are recommended. Freezing the face with the ether spray is in some instances beneficial. Stretching or section of the facial nerve has been employed in many cases, and the spasm has often disappeared temporarily. Strychnine, arsenic, and iron may be used.

Lesions of the Auditory Nerves.

The central relations of the auditory nerves are with the first temporal gyri. Experimentally, bilateral destruction of these gyri in monkeys causes deafness. Cases of disease in man indicate that the situation is the same. On the left side destruction of the first temporal gyrus causes the condition known as word-deafness, an inability to understand the meaning of words, though they may still be recognized as sounds.

Disturbance of function is not common as a result of lesion of the centre or of the auditory path; much more commonly deafness results from disease of the nerve after it has left the nucleus in the floor of the fourth ventricle, or much more frequently from involvement of its branches of distribution in the vestibule and cochlea.

Degeneration of the auditory nuclei is rare even in extensive and widespread bulbar disease. The nerve may be compressed at the base of the brain by tumors or the exudation of meningitis, or may be torn in fracture. In epidemic cerebro-spinal meningitis the auditory nerves are not infrequently involved: permanent deafness may result, which in the case of very young children leads to deaf-mutism. A primary degeneration of the nerves has been met with in locomotor ataxia, but it is extremely rare in comparison with the atrophy of the optic nerve in this disease.

In a large proportion of all cases with auditory-nerve symptoms the lesion is in the distribution; that is, in the labyrinthine branches. Three groups of symptoms may be produced: hyperæsthesia and irritation, diminished function or nervous deafness, and vertigo.

True hyperæsthesia—hyperacusis—a condition in which sounds, even those inaudible to other persons, are heard with intensity, is met with occasionally in hysteria, more rarely in cerebral disease. The term dysæsthesia or dysacusis is applied to the state in which ordinary noises are badly borne, as in headache.

Tinnitus aurium is a term used to characterize the subjective sensation of noises in the ears, such as roaring, ringing, buzzing, singing, ticking, etc. Tinnitus may accompany very many forms of ear disease, such as wax pressing on the drum, otitis media, and affections of the labyrinth. A very sudden and intense stimulation of the nerve, such as is caused by the loud whistle of a railway engine, has been known to cause permanent tinnitus. A not uncommon form is that in which a pulsating bruit or buzz-

ing murmur is heard in the ear, which may be caused either by abnormal conditions of the circulation, or more commonly by some special increase in the sensitiveness of the nerve, so that it is excited by the blood-current, which under normal conditions flows noiselessly. The murmur may be present as a very definite systolic bruit perceptible to the patient when he rests his head upon the pillow, or is even constantly present. In physicians I have known it to cause great uneasiness, owing to the supposition that an aneurism was present. It is usually associated with overwork, anæmia, neurasthenia, or gout. The epileptic aura may consist of subjective auditory sensations, which are also sometimes present in migraine. Occasionally a ticking or pulsating sound may be heard at a distance. The former is probably due to the action of muscles connected with the Eustachian tube or of the levator palati. The condition may persist unchanged for years and then suddenly disappear.

The **diagnosis** of tinnitus rarely offers any difficulty, but it may be impossible to say upon what it depends. A constitutional disturbance, such as gout, may be the cause, and I know of a case in which persistent and distressing nocturnal tinnitus ceased with the abstinence from stimulants. The pulsating forms, in which the sound resembles a bruit, are almost invariably subjective, and nothing is audible on auscultation over the ears. It is not to be forgotten that in young children there is a systolic brain-murmur sometimes audible at a distance. It is occasionally also heard in adults.

Impaired hearing due to involvement of the nerve is known as nervous deafness, in contradistinction to the loss of hearing due to disease of the tympanum. To test for this the tuning-fork should be placed against the temporal bone. If the vibrations are audible, the conclusion may be drawn that the nervous apparatus of hearing is not involved. On closure of the meatus the sound may also be intensified. The watch may be used in the same way, and if the sound is better heard in contact with the mastoid process with the meatus closed than it is when held opposite to the open ear, the deafness is probably not of nervous origin. Among possible causes of nervous deafness Gowers mentions the following: "(1) Symmetrical disease of the labyrinth, which is common; acute inflammation is sometimes, and chronic degeneration is often, bilateral; double otitis has sometimes been mistaken for meningitis; (2) symmetrical lesions of the two auditory nerves, which are very rare; (3) possible diseases of the medulla; (4) a tumor in the corpora quadrigemina, damaging the crustæ of the crura cerebri; (5) symmetrical disease of each temporo-sphenoidal lobe; syphilitic gummata caused this effect in a case recorded by Wernicke and Friedländer."

Vertigo, or giddiness, is a very special feature in certain forms of labyrinthine disease. It will be considered separately under the subjects of Vertigo and Ménière's Disease.

Lesions of the Glosso-pharyngeal Nerve.

This nerve distributes motor fibres to the stylo-pharyngeus and the middle

constrictor of the pharynx; sensory fibres to the upper part of the pharynx, the soft palate, and the back of the tongue; and is the nerve of the special sense of taste for the posterior part of the tongue and soft palate.

Isolated paralysis of this nerve is extremely rare. The pharyngeal symptoms of bulbar paralysis are associated with involvement of the nuclei of this nerve, in which case the adjacent ganglia are also affected. In tumors at the base in meningitis and in diphtheritic neuritis the nerves may be attacked, causing difficulty in swallowing, loss of sensation in the pharynx, and loss of the sense of taste in the posterior part of the tongue. It seems not improbable that the taste-fibres of the glosso-pharyngeal come from the fifth nerve, since "there is no instance on record of loss of taste at the back of the tongue from disease of the roots of the glosso-pharyngeal nerve, while there is evidence that disease of the root of the fifth nerve causes loss of taste on the back as well as the front of the tongue, and also on the soft palate and palatine arch" (Gowers).

Here may be mentioned appropriately the disturbances of the sense of taste. Loss of the sense of taste—ageusia—is a common effect of morbid conditions of the mucous membrane of the tongue and palate: thus in the dry tongue of fever and the furred tongue of dyspepsia taste is greatly impaired or may be completely lost. The application of very strong irritants, such as pepper, vinegar, and hot sauces, may dull the sense of taste. Affections of the nerve may be followed by a loss of the sense. From the tip and sides of the tongue the impressions are conveyed through the gustatory divisions of the fifth, and in disease of the middle ear there may be loss of taste in these parts of the tongue, owing to involvement of the chorda tympani, the nerve through which the gustatory fibres are distributed. As we mentioned above, it seems not improbable that the fifth nerve subserves the sense of taste in the posterior part of the tongue as well.

Perversion of the sense of taste—parageusis—is occasionally met with in hysteria and in the insane. Subjective sensations of taste may be present as an aura preceding the epileptic attack and in the hallucinations of the insane.

To test the sense of taste various substances should be placed upon the tongue in small quantities, and the taste must be perceived before the tongue touches other parts of the mouth. The patient's eyes should be closed and the following tests applied: for bitter, quinine; for sweetness, a solution of saccharine; for acidity, vinegar; for the saline test, common salt. An extremely delicate test of the sense of taste is the feeble galvanic current, which gives the well-known metallic taste.

Pneumogastric Nerve.

Nuclear lesions, either degeneration or hæmorrhage, occur as an important part of bulbar paralysis, associated, as a rule, with similar changes in the spinal accessory and hypoglossal. Within the skull the nerve-roots may be compressed by tumors, meningeal exudation, or aneurism. In the neck the nerve-trunk within the carotid sheath may be involved in aneurism or injured by

stab wounds or compressed by tumors. Occasionally the nerve is involved in a neuritis, either diphtheritic or alcoholic. The branches of distribution are both motor and sensory, the former being supplied to the pneumogastric chiefly, if not entirely, through the spinal accessory.

Involvement of the pharyngeal branches which supply the constrictors and the levator palati causes difficulty in swallowing, as the food is not passed on into the gullet. Unilateral involvement does not cause much impairment in deglutition. Spasm of the muscles supplied by the pharyngeal branches is met with in hydrophobia and occasionally in hysterical patients. The laryngeal branches are frequently involved, particularly the recurrents, which, owing to their remarkable course, are liable to pressure by tumors within the thorax, particularly by aneurism. The superior laryngeal nerve is sensory to the mucosa and supplies also the crico-thyroid muscle. The recurrent branch supplies the mucosa below the cords and the other intralaryngeal muscles. The various forms of paralysis and spasm will be described under the section on Diseases of the Larynx. Here it is sufficient briefly to mention the common sequence of hoarseness, loss of voice, and inability to cough, owing to unilateral abductor paralysis in involvement of the recurrent laryngeal nerve by aneurism or tumor.

Bilateral abductor paralysis results occasionally from involvement of the spinal accessory nuclei in the medulla in bulbar paralysis and in locomotor ataxia. Less frequently it is produced by pressure upon both vagi or both recurrent nerves. It has also been met with in hysteria. The characteristic symptoms are difficult respiration and a prolonged inspiratory stridor, with little or no impairment of the voice. Paralysis of the adductors is not uncommon in hysteria and causes the characteristic aphonia. It may follow also laryngitis. There is no dyspnœa and no stridor, and complete loss of voice.

Spasm of the muscles of the larynx is met with in laryngismus stridulus or child-crowing, in whooping cough, and in locomotor ataxia, forming the so-called laryngeal crisis. Paroxysmal attacks of laryngeal spasm may occur in hysteria.

Anæsthesia and hyperæsthesia, owing to paralysis of the laryngeal branches, are rare. The former, which occasionally occurs in diphtheritic paralysis, is a dangerous event, as the particles of food may enter the glottis and lead to deglutition-pneumonia.

Our knowledge of the disturbance of function in the pulmonary branches of the vagi is still uncertain. Motor fibres are distributed to the muscles of the bronchi, spasm of which is believed to play an important part in bronchial asthma, and which in consequence has been described as a vagus neurosis. Changes in the respiratory rhythm, such as the Cheyne-Stokes breathing, and the various forms of hurried respiration probably depend upon central, not peripheral, changes.

The vagus fibres of the cardiac plexus of nerves subserve motor, sensory, and probably trophic functions.

Through the motor fibres the inhibitory and regulating impulses pass to the

heart, the action of which in a few instances, as in the case of Colonel Townsend, can be slowed at will. Forcible pressure on both pneumogastrics in the neck is followed by slowing of the action of the heart. A similar effect has been produced by ligation of one pneumogastric. The central irritation of the vagus nuclei may be accompanied with retardation of the heart's action. With complete paralysis of the vagi the heart's action is greatly increased. This is sometimes seen in diphtheritic paralysis. Loss of the function of one vagus is not, however, necessarily followed by symptoms.

Normally, we receive no sensory impressions from the heart unless it be beating at an unusual rate or unless the rhythm be disturbed, when we may experience the sensation known as palpitation. The various disturbances under this heading, including angina pectoris, which is sometimes spoken of as a neurosis of the cardiac branches of the vagus, will be considered in the section on Diseases of the Heart.

The œsophageal and gastric branches preside over the muscular movements of the gullet and the stomach, and are concerned in the act of vomiting and in spasm and spasmodic affections. Gastralgia is in all probability a neuralgia of the branches of this nerve, though some attacks may be due to cramp in the muscles of the stomach. The gastric crises in locomotor ataxia are probably due to central irritation of the nuclei of the spinal accessory. The various forms of nervous dyspepsia and the motor disturbances of the stomach due to lesions of this nerve will be considered under the appropriate section. And, lastly, exophthalmic goitre is sometimes considered as a neurosis of the vagi.

Spinal Accessory Nerve.

The smaller portion of the nerve joins the pneumogastric as its important motor root. The larger external part is distributed to the sterno-mastoid and trapezius muscles.

The nuclei of the nerves are involved in bulbar paralysis, more particularly the accessory or internal part. The nuclei of the external portion, which are situated in the cervical portion of the cord, may be involved in the general nuclear wasting of progressive muscular atrophy. The nerve may be compressed by tumors or involved in the exudation of caries or meningitis, sometimes in fracture. When within the skull the paralysis which results involves half of the soft palate, the vocal cord on the same side, and the sterno-mastoid and trapezius. Within the spinal cord the fibres passing to these muscles may alone be involved, causing paralysis, which in the case of the trapezius is only partial, as the lower portion is innervated by the cervical nerves. In loss of power of one sterno-mastoid the head is rotated with difficulty to the opposite side. There is not necessarily torticollis, though in some cases the head is held obliquely. The paralysis of the trapezius is well indicated in the acts of shrugging the shoulders and of drawing a deep breath. The shoulder on the affected side droops a little and the elevation of the arm is somewhat impaired, since the trapezius does not fix the scapula as a point from which the deltoid can work. Bilateral paralysis of the muscles supplied by the

spinal portion of this nerve is seen in some cases of progressive muscular atrophy. When the sterno-mastoids are chiefly involved the head tends to fall backward. If the trapezii are wasted, the head drops forward, a very characteristic attitude in many cases of this disease. Drooping of the head is an important symptom in cervical caries in children. There are cases in which the child has difficulty in holding up the head during the first year of life, due, it is possible, as Gowers suggests, to injury of the accessory nerves during protracted labor.

The treatment of these cases is not very satisfactory. The paralysis from nuclear degeneration is, as a rule, hopeless. That caused by meningeal exudation and due to pressure from other causes sometimes disappears. The muscles should be stimulated by the use of galvanism and systematic massage.

The muscles supplied by the spinal accessory are very liable to a spasmodic affection known as

Torticollis, or Wry-neck.

(*a*) Congenital Torticollis.—This is known also as fixed torticollis, and is dependent upon the shortening and atrophy of the sterno-mastoid on one side, most commonly the right. It is usually attributed to injury during birth. It may not be noticed in a child for some years on account of shortness of the neck. The sterno-mastoid on the affected side is shortened, hard, firm, and in a condition of more or less advanced atrophy. This condition must not be confounded with local thickening of the sterno-mastoid muscle and the formation of a muscle callus following rupture at birth. In some instances the fibrous atrophy involves a part of the trapezius muscle. An interesting symptom in this form of torticollis is facial asymmetry, described by Wilks, which may not be noticed until the child is eight or ten years old. Golding-Bird suggests that the facial asymmetry and torticollis are parts of a central affection, the counterpart in the head and neck of infantile spinal paralysis.

(*b*) Spasmodic Torticollis.—Two varieties occur, the tonic and the clonic. Tonic spasm is usually limited to the muscles of one side: the occiput is drawn toward the shoulder of the affected side, the face is rotated toward the opposite shoulder, and at the same time the chin is raised. When the trapezius is affected, the depression of the head toward the same side is more marked, and the shoulder is also raised by its action. In long-standing cases the muscles are very prominent and rigid. Both muscles are rarely involved in the tonic form. The splenius capitis may be involved alone, or more commonly with the sterno-mastoid.

The clonic form is much more distressing. The jerking contractions recur every few minutes, either in the sterno-mastoid alone on one side or more frequently in several of the cervical muscles, particularly the splenius and the trapezius. More rarely the muscles on both sides are involved. There are instances of rotatory spasm of the head, due probably to clonic contractions of the obliquus capitis. In other cases there is a nodding spasm, in which the

deeper-placed recti capitis muscles are involved. The spasm not infrequently extends, and involves the muscles of the face, and even those of the arms.

The disease is most common in adults. In females it may be an hysterical manifestation. Cases have followed exposure to cold or have resulted from injury to the neck. In the majority of instances the cause of the disease is obscure, and nothing really is known of its essential nature. It is regarded as a functional neurosis, but it is possibly due to disturbance in the cortical centres presiding over the muscles. Cold is believed by some to have an important influence, and cases have been described as rheumatic torticollis.

The disease must be distinguished from the nodding spasm of epilepsy, which is usually seen in young children, accompanied with slight loss of consciousness. There is also seen in children about the time of dentition a unilateral jerking of the head from side to side, which, as a rule, is not of any special significance. Spasm of the muscles of the neck occurs in cervical caries, usually associated with tenderness over the spines: examination of the pharynx may reveal swelling and tenderness beneath the anterior ligament.

The disease varies greatly in its course. A majority of the cases persist for a long time, and too often the cure is only temporary.

Treatment.—In the tonic form section of the muscle with the application of a suitable apparatus may effect a cure. In the clonic variety fixation of the head mechanically can rarely be borne. Drugs are of little or no value, though it is stated that very large doses of potassium bromide lessen the intensity of the spasm. Morphine, which has been highly recommended, should be employed with great care. Electricity has been warmly recommended. Counter-irritation, particularly with the thermo-cautery, may be tried. In very obstinate cases surgical measures may be employed, and division or stretching of the nerve and section of the muscle have been resorted to, sometimes with benefit: as a rule the condition recurs. Personally, I have not seen a permanent cure in any case of spasmodic torticollis.

Hypoglossal Nerve.

This is the motor nerve of the tongue and for the extrinsic muscles except the mylo-hyoid and digastric. The cortical centre is in the lower part of the ascending frontal gyrus.

Paralysis.—This may follow a lesion of the cortical centre, as in hemiplegia, with which it will be considered, or is due to affection of the nuclei in the medulla or to involvement of the nerve in its course. Nuclear disease is usually part of a bulbar paralysis, and is bilateral and associated with paralysis of the lips and pharynx. Speech is greatly impaired, as the linguals and dentals cannot be pronounced. Mastication and deglutition are performed with difficulty. The tongue usually wastes and there are fibrillary tremors. The mucous membrane is thrown into folds, and in extreme cases the tongue lies motionless in the floor of the mouth and cannot be protruded. Unilateral paralysis and atrophy more commonly follow involvement of the nerve outside the nucleus, due to meningitis, syphilis, tumors, or caries, sometimes following

injuries of the neck and jaw. The atrophy is usually marked, and the mucous membrane on the affected side is thrown into folds. When protruded the tongue is pushed toward the affected side. The cases are rare. Birkett in a description of a remarkable instance states that he has only been able to collect thirteen cases in the literature. In his patient the paralysis resulted from inflammatory changes about the cervical glands at the angle of the jaw; and in such cases there may also be involvement of other nerves.

The **diagnosis** is rarely difficult. In supranuclear paralysis there is associated hemiplegia; the muscles do not waste and there are no electrical changes. The nuclear disease is almost invariably bilateral and part of a bulbar paralysis. The muscles waste and the reaction of degeneneration is present. Unilateral paralysis and atrophy are most common in infranuclear lesions.

Spasm.—This may involve one or both sides, and is usually part of some convulsive disorder, either chorea or facial spasm. It may occur in hysteria, and cases are said to result from reflex irritation in the fifth nerve. It is not uncommon to see the tongue protruded in a spasmodic manner just before the explosive utterance of words in stuttering. There are cases of clonic spasm in which the tongue is thrust in and out forty or fifty times a minute. The spasm in these instances may be unilateral.

The **prognosis** is good, as the spasm is rarely due to organic disease.

The **treatment** of paralysis of the hypoglossal nerve is rarely successful, except in the cases of unilateral disease due to syphilis. When due to bulbar atrophy it is incurable.

LESIONS OF THE SPINAL NERVES.

Cervical Plexus.

(1) *Occipito-cervical Neuralgia.*—This involves the occipitalis major and minor and the auricularis magnus nerves. The patient complains of pains in the neck, in the occiput, and in the ear, which sometimes radiate to the face and to the arm. It usually follows cold, and may be associated with a stiff neck or with torticollis. Occasionally it is caused by pressure, as in carrying a heavy load on the neck. Painful points may be present midway between the mastoid processes and the spine.

The **prognosis** is, as a rule, good except when the neuralgia is due to disease of the cervical vertebræ. Occasionally in syphilis there is a cervico-occipital neuralgia, which yields readily to iodide of potassium.

(2) *Phrenic Nerve.*—In the neck the nerve-trunk is occasionally divided by punctured wounds, and in the thorax compressed by tumors and by aneurism. Paralysis follows involvement of the motor centres in the cervical cord in progressive muscular atrophy. It may also result from lead-poisoning and neuritis. Owing to the inaction of the diaphragm, respiration is carried on by the intercostal and accessory muscles, the movements of the thorax are increased, and the abdomen is retracted instead of being pushed out during inspiration. When the patient is quiet and at rest there may be very slight

disturbance, but on exertion the respiration is quickened and there may be dyspnœa. When paralyzed on one side only, inspiration may show that the descent of the diaphragm is much less on one side. The difficulty of coughing and expelling the mucus renders pulmonary complications very dangerous.

The **diagnosis** is not always easy. In hysterical women the breathing may be entirely thoracic and the diaphragm scarcely moves during inspiration. Immobility of the muscle is present in diaphragmatic pleurisy and in large purulent effusions. The muscle may itself be degenerated, and instances are recorded by Callender of primary degeneration of the muscle-fibres.

The **prognosis** is unfavorable in the cases due to neuritis. W. Pasteur states that of 15 cases following diphtheria, only 8 recovered. A serious risk is in the tendency to œdema and engorgement of the bases of the lung, owing to the lessened action of the diaphragm.

The **treatment** is, as a rule, that of neuritis. Galvanism of the phrenic nerve may be tried: one pole may be placed just outside the lower part of the clavicular portion of the sterno-mastoid, and the other at the epigastrium.

Spasm of the diaphragm may be either tonic or clonic. The former is stated to occur sometimes and to be the cause of death in tetanus. Clonic spasm of the diaphragm causes hiccough or singultus. This may be a functional disorder, as in hysteria, but the spasm is not infrequently excited by the direct action of hot substances as they pass through the œsophageal opening of the diaphragm. Occasionally it arises from reflex irritation in the stomach or intestines, as in dysentery, cholera, and peritonitis. It may be caused by direct irritation of the phrenic nerve in its course. Much more rarely it is due to central irritation. In chronic alcoholism and in uræmia it may be a persistent and even dangerous symptom. In lead-poisoning it may occur without any obvious cause, and, persisting day and night, may prove fatal.

Though rarely causing alarm, protracted cases in delicate or elderly people may be serious and very difficult to relieve.

Among remedies which may be tried are inhalations of nitrite of amyl, which usually relieves with great promptness, and the good effect may be kept up by the administration of nitro-glycerin in the intervals. Inhalations of chloroform check the spasm at once, though usually only for a time. The hypodermic injection of a quarter to a third of a grain of morphine may be necessary to procure sleep. Nothing relieves the persistent hiccough of acute alcoholism better than a hypodermic injection of apomorphine. The hysterical form rarely resists the static electricity.

Brachial Plexus.

The nerves may be involved above or below the clavicle—in the former situation by direct injury, tumors, and other affections of the neck. The infraclavicular portion is specially liable to injury in dislocation of the shoulder, the strain of a sudden wrench of the arm, in laceration by a fracture, and more rarely in an ascending neuritis. Injury to the nerves in the operation of turning is not an uncommon form of the so-called obstetrical palsy.

The paralysis following dislocation of the shoulder, more especially the subcoracoid form, is particularly important. When the luxation is quickly reduced the symptoms disappear in a short time. In other cases, in which the dislocation has existed for some time, every muscle of the arm may be paralyzed. Very serious indeed are the cases in which the dislocation is undetected and remains unreduced for some time, as the prolonged pressure on the plexus may cause complete and permanent paralysis, with wasting of the muscles, contractures, and trophic changes of the skin. The medico-legal aspect of these cases is most important, and the practitioner should be on his guard against suits for damages which may be brought in case of permanent disability. In rare instances this may follow direct injury in the region of the shoulder without dislocation and apparently without fracture. The dislocation may be set at once, but the damage to the plexus results in permanent paralysis of certain of the nerve-trunks. In one instance seen by the writer the surgeon reduced the dislocation, but subsequently the head of the bone slipped out of the socket, and the patient was not seen again until irreparable damage had been done.

The obstetrical cases have very considerable interest, and have been studied by Duchenne and by Erb, whose name this form of paralysis sometimes bears. The muscles involved, as a rule, are the deltoid, the biceps, the supraspinatus, infraspinatus, the brachialis anticus, and supinator longus. The lesion is often not noticed for some days after birth, when the nurse or mother calls the attention of the physician to the fact that one arm of the child hangs loosely by its side. The movements of the wrist and of the fingers are unaffected, and the forearm can be extended upon the arm, but the arm cannot be lifted. At first not much difference can be noticed in the size of the arm, as the subcutaneous fat is well developed. The nerves involved are the fifth and sixth. It is particularly liable to occur in the extraction of the head in breech delivery, and, according to Starr, in the majority of the cases the injury is done by pressure of the fingers of the obstetrician. In other instances it is caused by pressure of the forceps or traction on the arm in version. The outlook is, as a rule, good, but occasionally the loss of power remains in some of the muscles.

Brachial Neuritis is a very important affection which may either follow a neuritis of one of the branches or be primary in the plexus. The ascending neuritis usually follows an injury of one of the peripheral nerves, and is associated with great pain and with wasting of the muscles.

The primary form is a perineuritis of the sheaths of the brachial cord, and is an affection "so closely analogous to sciatica that it may be called sciatica of the arm" (Gowers). Some of the cases would indicate rather that the nerve-roots were involved, as there is much pain about the spine. The affection is met with chiefly in females after fifty, particularly in persons of a full habit who have had gouty manifestations. The pain is severe, and felt either in the plexus itself or along the course of the nerves of the arm, more rarely about the shoulder and beneath the scapula. It may come on in

paroxysms, and is often described as a burning soreness accompanied with tingling of the skin. There is rarely great wasting of the muscles, though there is pain on motion. There may be slight œdema of the hands and glossiness of the skin; occasionally arthritis. The condition must be distinguished from the humeral periarthritis with atrophy of the muscles and neuralgic pains.

Lesions of the Individual Nerves of the Brachial Plexus.

(*a*) Long Thoracic Nerve (Serratus Palsy).—This is met with chiefly in men as a result of injury to the nerve in the neck by direct pressure, as in carrying a heavy load, rarely in consequence of long-continued effort with the arm raised, as in whitewashing a ceiling; more rarely it is due to cold. In progressive muscular atrophy and in acute poliomyelitis anterior the serratus may be involved with other muscles. The paralysis is readily recognized by the position of the scapula, the inferior angle of which is nearer the spine, owing to the unopposed action of the rhomboid and of the levator anguli scapulæ. The posterior border projects, so that the scapula looks winged, which is particularly noticeable when the arm is moved forward. As a rule, the fingers can be readily inserted under the margin.

The affection runs a slow course, and it may be months before the condition improves.

(*b*) Circumflex Nerve.—This is apt to be involved in injuries, in dislocations, and is sometimes bruised in the use of a crutch. It may be paralyzed also by neuritis and by pressure during acute illness. The nerve supplies the deltoid and teres minor muscles and the skin over the former muscle. In consequence of paralysis of the deltoid the arm cannot be raised, and the wasting which usually follows changes materially the shape of the shoulder. In time the articular ligaments become relaxed, and there may be a distinct space between the head of the humerus and the acromion. In other instances trophic changes occur about the joints, such as thickening of the ligaments and adhesions, resulting at last in a condition not unlike ankylosis. Anæsthesia of the skin over the muscle is not always present.

(*c*) Musculo-spiral Paralysis; Radial Paralysis (Sleep Palsies).—The exposed position of the musculo-spiral nerve as it leaves the plexus to wind round the bone makes it liable to injury, particularly in fracture. Bruising of this nerve in the use of the crutch is the commonest cause of the so-called "crutch-palsy." A still more frequent cause is pressure during sleep when the arm is hanging over the back of a chair, or pressure of the body upon the arm when a person is sleeping on a hard bench or on the ground. Neuritis due to cold or an infectious disease is a less common cause, and some of the cases attributed to these are really due to pressure. Direct muscular action, as in throwing a stone or a cricket-ball violently, may completely paralyze the nerve. Transient palsy may be caused by the accidental puncture of the nerve in a hypodermic injection. The common paralysis of lead-poisoning is the result of involve-

ment of branches of this nerve. A complete lesion of the musculo-spiral high up causes paralysis of the triceps, the brachialis anticus, both supinators, and the extensors of the wrists and fingers. In a lesion about the elbow the arm-muscles and the supinator longus are spared. In the pressure palsies, as a rule, the supinators are involved. The characteristic feature of this paralysis is the wrist-drop and the inability to extend the first phalanges of the fingers and thumbs. If the forearm be extended, the hand droops and cannot be raised, nor can the fingers or thumbs be extended. If, however, the hand and the first phalanges are supported, the action of the interossei and the lumbricales then extend the middle and terminal phalanges. Sensation is not always affected. There may be numbness or tingling, but rarely complete anæsthesia. Musculo-spiral palsy is readily recognized, though it may be difficult sometimes to assign the proper cause. The pressure palsies are, as a rule, unilateral, and involve the nerve high enough to include the supinator longus. In the neuritis from lead the affection is bilateral, as it is also in the alcoholic form, both of which are recognized easily by their concomitant features.

The outlook is good, particularly in the pressure cases, in which the paralysis may disappear in a few days. The electrical examination is of the greatest importance in the prognosis, and the rules apply which are laid down in paralysis of the facial.

(*d*) Paralysis of the Ulnar Nerve.—The ulnar supplies by its motor branches the ulnar halves of the deep flexor of the fingers, the muscles of the little finger, the interossei, the ulnar flexor of the wrist, and the adductor and short head of the inner flexor of the thumb. The sensory distribution is to the ulnar side of the hand, including two and a half fingers on the back and one and a half fingers on the front. The paralysis occasionally results from pressure, more commonly from prolonged flexion of the elbow, as in sleep and in illnesses. The hand deviates a little to the radial side, owing to paralysis of the ulnar flexor of the wrist. Flexion of the first phalanges is impossible, and also adduction of the thumb. In long-standing cases the first phalanges become very extended and the others strongly flexed, producing the so-called claw-hand or *main en griffe*. The loss of sensation is in the distribution already mentioned.

(*e*) Paralysis of the Median Nerve.—The motor distribution is to the radial flexor of the wrist, the flexors of the fingers with the exception of the ulnar half of the deep flexors, the abductor and flexors of the thumb, the radial lumbricales, and the pronators of the wrist. The sensory branches supply the radial side of the palm, the front of the thumb, the first two fingers, half of the third finger, and the skin on the back of these three fingers. This nerve is seldom paralyzed alone. It may be involved in fracture and occasionally in neuritis, and very rarely by violent contraction of the pronator teres. The wrist in flexion is drawn strongly to the ulnar side, and the thumb cannot be opposed to the tips of the finger. The second phalanges cannot be flexed on the first nor the distal phalanges on the second, but in the third and

fourth fingers this can be performed by the ulnar half of the flexor profundus. When the sensation is involved it follows the distribution of the fibres, as already mentioned. The wasting of the thumb-muscles forms a striking characteristic in this form of paralysis. The skin may be glossy and the nutrition of the nails impaired.

Thoracic and Dorsal Nerves.

The anterior branches of the twelve dorsal nerves supply the intercostal muscles, the levatores costarum, the abdominal muscles, and the serrati postici. The sensory branches supply the skin in the antero-lateral region of the thorax and abdomen. The posterior branches of the dorsal nerves supply the deep muscles of the back and the skin over the same.

Affections of these branches are not very frequent, except of the intercostal nerves, which are the subject of an intractable form of neuralgia.

Intercostal neuralgia occurs most commonly in women, and involves the nerves from the third to the ninth, most frequently the seventh, eighth, and ninth on the left side. The cases are most common in anæmic, overworked women. The nerves may be involved by aneurism or tumor, occasionally in chronic pleurisy, or in the adhesions of long-standing tuberculosis, or in caries of the spine. Though usually constant, the pain is subject to marked exacerbations, and may be very severe; movements such as coughing and deep inspiration aggravate it very greatly. Tender points are usually present at the intervertebral foramen, one near the sternum, one over the rectus muscle, and a third midway between these. The neuritis causing neuralgia is often accompanied with an eruption of herpes zoster, forming the so-called "shingles." The pain may be most intense prior to the outbreak of the rash, and in some instances persists long after its disappearance.

The diagnosis of intercostal neuralgia is usually easy, though special care must be taken to exclude the presence of spinal caries, of aneurismal tumor, and of pleurisy. Many cases prove very intractable.

A special form is the neuralgia of the branches passing to the breast—mastodynia. It is seen most commonly shortly after puberty in anæmic and hysterical girls. The pain may be very severe, either localized or involving the entire breast. Occasionally small hard nodules are felt beneath the skin. The condition may follow prolonged lactation.

Paralysis of the muscles supplied by the thoracic and dorsal nerves is rarely seen alone, and in cases of hemiplegia they are not involved, the muscles of both sides being innervated from either hemisphere (Broadbent). In the forms of primary muscular atrophy the weakness of the back muscles is very striking, and the attitude of the child, with marked arching of the lumbar vertebræ, prominence of the abdomen, and arching backward of the back, forms a very characteristic picture. In getting up from the floor the child has to lift his body on the arms and gradually climb up his legs, as in the familiar picture in Gowers' work.

Lumbar and Sacral Plexuses.

The lumbar plexus, made up of loops of communication between the anterior branches of the four upper lumbar nerves, supplies the flexors and adductors of the hip-joint, the extensors of the feet, and the cremaster. The sensory fibres are distributed to the skin of the lower part of the abdomen, the antero-lateral region of the thigh, and the inner side of the leg and foot.

The cords of the plexus itself are sometimes involved by tumors of the lymph-glands, in psoas abscess, and in caries of the vertebræ. Affections of the individual nerves of the lumbar plexus are not so common.

The *anterior crural* nerve may be involved in wounds, in psoas abscess, and in disease of the vertebræ, stretched in dislocation of the hip-joint, or invaded by pelvic tumors. When paralyzed there is loss of power in the extensors of the knee, and if the nerve is involved high up there may be loss of power in the psoas muscle. In prolonged involvement the muscles waste and walking may be difficult or impossible. There is anæsthesia of the greater portion of the skin of the thigh, except a narrow strip at the back part, and in the distribution of the internal saphenous nerve along the inner side of the leg to the big toe. Neuralgia of the crural nerve is not very common, apart from the pressure symptoms due to tumors and growths about the spine. The pain is in the antero-internal portion of the thigh and knee and extends along the inner surface of the leg and foot. There is often a painful spot where the nerve emerges below Poupart's ligament.

The *obturator nerve* is occasionally injured during parturition. When paralyzed there is loss of power in the adductors of the thigh, and the patient cannot cross one leg over the other. Owing to involvement of the obturator externus, rotation inward of the thigh is not well performed.

There are troublesome neuralgias of certain branches of the lumbar plexus. There may be pain in the course of the ilio-inguinal and ilio-hypogastric nerves, in the neighborhood of the crest of the ilium, and in the external abdominal ring. The ilio-inguinal nerve, which accompanies the spermatic cord through the inguinal canal and escapes at the external abdominal ring, is distributed to the skin of the upper and inner part of the thigh and to the scrotum. There are instances in which the distribution of this nerve is the seat of very severe pain, and the affection known as the *irritable testis* of Cooper is believed to be an affection of this nerve. Associated with this pain there may be sensations of fainting and the sickening feeling such as is felt on compression of the testis.

Sacral Plexus.—This is still more likely to be damaged by pelvic tumors and various affections of the pelvic bones. The branches may be injured during parturition. Neuritis is not uncommon, and is frequently an extension from the sciatic.

Of the branches, the *sciatic nerve* when paralyzed causes loss of power in the flexors of the leg and in the muscles below the knee. An affection or injury below the middle thigh involves only the muscles of the leg proper.

There is anæsthesia of the outer half of the leg, the sole, and the greater portion of the dorsum of the foot. The muscles frequently waste and there may be trophic disturbances. In paralysis of one sciatic nerve the leg is fixed at the knee by the action of the quadriceps extensor. Paralysis of the small sciatic nerve is rarely seen. The gluteus maximus is involved, there is difficulty in rising from a seat, and there is usually a strip of anæsthesia on the back part of the thigh in the region of distribution of the cutaneous branches. Of the branches of the sciatic nerve, the *external popliteal* when paralyzed causes loss of power in the peronei, the long extensor of the toes, the tibialis anticus, and the extensor brevis digitorum. As a result there is a foot-drop, the ankle cannot be flexed, and, as the toes cannot be raised from the ground in walking, the whole leg is lifted, producing the characteristic *steppage gait* seen in so many forms of peripheral neuritis. In long-standing cases the foot is permanently extended and there is wasting of the anterior tibial and peroneal muscles. The loss of sensation is in the outer half of the front of the leg and on the dorsum of the foot.

Paralysis of the *internal popliteal nerve* causes loss of power in the gastrocnemius, the plantaris, soleus, popliteus, the tibialis posticus, the long and short flexors of the toes, and the muscles of the sole of the foot. The foot cannot be adducted nor can the patient rise on tiptoe. In long-standing cases talipes calcaneus follows, and the toes assume a claw-like position from secondary contracture.

Among other neuralgic affections of the lumbar and sacral plexuses are the following: coccygodynia, an affection most common in women. The pain about the coccyx is greatly aggravated by the sitting posture, and is usually associated with other nervous phenomena. It is an extremely intractable affection, and the condition may be so intolerable that resection of the coccyx has to be performed—an operation which, however, is not always successful in relieving the pain.

There are certain neuralgic affections of the nerves of the feet which are very troublesome. In the affection known as painful heel, the pododynia of S. D. Gross, the pain is usually most severe in the heel itself, sometimes in a very limited spot on the under surface, sometimes in the line of the metatarsophalangeal joint. It is most common in women, and is not necessarily associated with any swelling, discoloration, or enlargement of the joint. In some instances it would appear to be a manifestation of hysteria; in others the patients have rheumatism or gout. Some of the worst cases occur in shop-girls as a result of standing for a long time on the feet.

Plantar neuralgia may be associated with a definite neuritis, and is sometimes seen after the specific fevers, and has been described by Hughes in caisson disease. The pain may extend along the sole of the foot or be confined to the tips of the toes, occasionally to the ball of the great toe. Numbness, tingling, hyperæsthesia, and sweating may occur with it. A curious tenderness of the toes, possibly due to a neuritis, is not infrequently seen in typhoid fever in patients who have been subjected to the cold-bath treatment.

In this connection may be mentioned, as possibly due to a neuritis of the nerves, the condition described by Weir Mitchell as erythromelalgia, which is accompanied with great pain in the heel or in the sole of the foot, and vascular change, either an acute hyperæmia or cyanosis. It is an affection similar in most respects to Raynaud's disease.

Sciatica.

This is either a neuritis of the sciatic nerve or of its cords of origin. It is regarded sometimes as a functional neurosis. It occurs most frequently in adult males. Rheumatism or gout is present in many cases. Exposure to cold, particularly after heavy muscular exertion, or a severe wetting is not an uncommon cause. The nerve-cords of the sacral plexus may be compressed by ovarian or uterine tumors, by lymphadenomata, or by the fœtal head during labor. Occasionally lesions of the hip-joint induce a secondary sciatica. The condition of the nerve has been examined in a few cases. In the operation of stretching it has been found swollen and reddened. Histologically, an interstitial neuritis has been present. The affection may be most intense at the sciatic notch or in the nerve about the middle of the thigh.

Of the **symptoms**, the most constant and troublesome is pain, which, as a rule, sets in gradually, and for a time may be slight and confined to the back of the thigh, and felt particularly in certain positions or after exertion. At the onset there may be fever. Soon the pain becomes more intense, and, instead of being limited to the upper portion of the nerve, extends down the thigh, reaching the foot and radiating over the entire distribution of the nerve. The patient can often point out the most sensitive spots, usually at the notch or in the middle of the thigh, and on pressure these are exquisitely painful. The pain is described as gnawing or burning, and is usually constant, but in some instances is paroxysmal, and often worse at night. On walking the knee is bent and the patient treads on his toes, so as to relieve the tension on the nerve. In protracted cases there is wasting of the muscles, but the reaction of degeneration can seldom be obtained. In these chronic cases cramps may occur and fibrillary contractions. Herpes may develop, but this is unusual. In rare instances the neuritis ascends and involves the spinal cord.

The **duration** and **course** are extremely variable. As a rule, it is an obstinate affection, lasting for months, or even, with remissions, for years. Relapses are not uncommon, and the disease may be relieved in one nerve only to appear in the other. In the severer forms the patient is bed-ridden, and such cases prove among the most distressing and trying which the physician is called upon to treat.

In the **diagnosis** it is important, in the first place, to determine whether the disease is primary, or secondary to some affection of the pelvis or of the spinal cord. A careful rectal examination should be made, and in women pelvic tumor should be excluded. Lumbago may be confounded with it. Affections of the hip-joint are easily distinguished by the absence of tenderness in the course of the nerve and the sense of pain on movement of the hip-

joint or on pressure in the region of the trochanter. There are instances of sacro-iliac disease in which the patient complains of pain in the upper part of the thigh, which may sometimes radiate, but careful examination will readily distinguish between the affections. Pressure on the nerve-trunks of the cauda equina, as a rule, causes bilateral pain and disturbances of sensation, and, as double sciatica is rare, these features always suggest lesion of the nerve-roots. Between the severe lightning pains of tabes and sciatica the differences are usually well defined.

Treatment.—The pelvic organs should be carefully and systematically examined. Constitutional conditions, such as rheumatism and gout, should receive appropriate treatment. In a few cases with pronounced rheumatic history, wherein the trouble comes on acutely with fever, the salicylates seem to do good. In other instances they are quite useless. If there be a suspicion of syphilis, the iodide of potassium should be employed, and in gouty cases salines.

Rest in bed with fixation of the limb by means of a long splint is a most valuable method of treatment in many cases, one upon which Weir Mitchell has specially insisted. I have known it to relieve, and in some instances to cure, obstinate and protracted cases which had resisted all other treatment. Hydrotherapy is sometimes satisfactory, particularly the warm baths or the mud baths. Many cases are relieved by a prolonged residence at one of the thermal springs. Antipyrine, antifebrin, and quinine are of doubtful benefit.

Local applications are more beneficial. The hot iron or the thermo-cautery or blisters relieve the pain temporarily. Deep injections into the nerves give great relief, and may be necessary for the pain. It is best to use cocaine at first, in doses of an eighth to a quarter of a grain. If the pain is unbearable, morphine may be used, but it is a dangerous remedy in sciatica and should be withheld as long as possible. The disease is so protracted, so liable to relapse, and the patient's *morale* so undermined by the constant worry and the sleepless nights, that the danger of contracting the morphine habit is very great. On no consideration should the patient be permitted to use the hypodermic needle himself. It is remarkable how promptly, in some cases, the injection of distilled water into the nerve will relieve the pain. Acupuncture may also be tried: the needles should be thrust deeply into the most painful spot for a distance of about two inches, and left for from fifteen to twenty minutes. The injection of chloroform into the nerve has also been recommended.

Electricity is an uncertain remedy. Sometimes it gives prompt relief; in other cases it may be used for weeks without the slightest benefit. It is most serviceable in the chronic cases in which there is wasting of the legs, and should be combined with massage. The galvanic current should be used; a flat electrode should be placed over the sciatic notch, and a smaller one used along the course of the nerve and its branches. In very obstinate cases nerve-stretching may be employed. It is sometimes successful, but in other instances the condition recurs and is as bad as ever.

DISEASES OF THE MUSCLES.

By WILLIAM OSLER.

MYOSITIS.

Primary myositis is an affection of which several well-characterized cases have been recorded within the past few years. In Jacobi's patient swelling and pain, with loss of power, began in the muscles of the lower extremities and gradually involved the other muscles of the body, which became firm, hard, and tender. Finally, atrophy supervened in certain groups. Death occurred in about two years and a half from involvement of the respiratory muscles. The condition found was a myositis and perimyositis. The cases may progress more rapidly: thus in E. Wagner's case the patient, a tuberculous woman, complained of stiffness about the shoulders and œdema of the back of the hand and forearm, with paræsthesia. The muscles felt doughy and were painful on pressure. The legs gradually became involved, and death occurred in about three months. The muscles, with the exception of the glutei, calf, and abdominal muscles, were stiff, firm, and fragile, and showed fatty degeneration and great proliferation of the interstitial tissue. In Hepp's case there was hyaline degeneration of variable grades of the muscle-fibres and the intermuscular tissue was scarcely involved. In the case of Unverricht the interstitial tissue was much infiltrated and the muscle-fibres greatly degenerated. In both of these cases the spleen was enlarged. According to Löwenfeld, the disease is characterized by three cardinal symptoms: (1) swelling of the extremities, due in part to the œdema of the subcutaneous tissues, and in part to increase in the volume of the muscles, with which is associated a corresponding disturbance of function; (2) extension of the inflammatory affection to the muscles of respiration and deglutition; and (3) the presence of a more or less extended exanthem.

The exanthem in the majority of the cases has been of an erythematous nature and irregularly scattered over the trunk and extremities, and has sometimes been followed by slight pigmentation. Disorders of sensation are not usually present.

The swelling of the muscles, the soreness and œdema, and the pain naturally suggest trichinosis. The diagnosis could only be made in some instances by examination of portions of the muscle excised. Wagner suggests that some of the cases are examples of acute progressive muscular atrophy.

Acute purulent myositis is not very infrequent in pyæmia. It is occasion-

ally one of the sequelæ of typhoid fever. There are instances on record in which it apparently has been the primary affection.

Myositis ossificans progressiva is a rare disease in which the muscles undergo a progressive calcification. In this remarkable disorder either in localized spots or in widespread areas the muscle-tissue undergoes gradual ossification. Of 22 cases collected by Seidel, a majority were in men, and in more than one-half of the cases the disease began before puberty. The onset is usually with indications of an inflammatory process in the muscle, sometimes with swelling and tenderness. This gradually subsides, and the muscle becomes firm, hard like gutta-percha, and gradually undergoes conversion into bony tissue. The process may be confined to one or two muscles or to certain groups. In other instances the process is widespread and involves many muscles of the trunk and extremities. In Rogers's case, for example, the first reported in this country, the disease began at thirteen years of age. At the time of observation "it was found that the pectoralis major muscle was ossified at its superior part and extended in the direction of the clavicle to the arm, the bony deposits forming high and irregular elevations. The sterno-cleido-mastoideus was ossified from the sternum to its middle portion, with several elevations. The back exhibited the greatest quantity of ossific matter, having a tubercular appearance. The scapula was fixed to the ribs and studded with bony excrescences. All the muscles going to the scapula appeared more or less affected—viz. the trapezius, rhomboideus, subscapularis, etc. The latissimus dorsi formed a large bony plate from its origin to the angle of the scapula; at this part it had united to the ribs, forming a large tubercle. The longissimus dorsi was in a similar condition, extending upward along the spine, resembling a splint, and to this may be attributed the entire loss of motion in the lumbar vertebra."

The disease lasts many years, and may ultimately lead to complete disability. No remedial measures have proved of any avail.

THE MUSCULAR DYSTROPHIES.

Idiopathic Muscular Atrophy.

The following classification, taken from Raymond's monograph, gives a useful synopsis of the varieties and causes of muscular atrophy:

Circumscribed atrophies.	Atrophy from compression. Atrophy in inflammatory conditions (pleurisy, joint disease, etc.). Atrophy from injury or inflammation of individual nerves.

- Progressive atrophies . .
  - Progressive spinal muscular atrophy; type Aran-Duchenne.
  - Progressive myopathic atrophy.
    - Pseudo-hypertrophic muscular paralysis.
    - Type Leyden-Mobius.
    - Type Zimmerlin.
    - Type Erb.
    - Type Landouzy-Dejerine.
    - Type Charcot-Marie.
- Diffuse atrophies . . . .
  - Anterior poliomyelitis . . . .
    - Infantile form.
    - Acute of adults; spinal paralysis, with rapid course and curable (Landouzy-Dejerine); subacute and chronic form; chronic mixed form (Erb); diffuse subacute, general spinal paralysis (Duchenne).
  - Syringomyelia.
- Facial hemiatrophy . . .
  - Multiple neuritis (amyotrophic form).
    - Lead paralysis.
    - Leprous neuritis.
    - Alcoholic neuritis.
- Muscular atrophy of cerebral origin . . . . . .
  - With secondary degeneration involving the anterior cornua.
  - Without secondary degeneration involving the anterior cornua.
- Muscular atrophy in hysteria.
- Muscular atrophy from systemic disease of the cord . . . . . .
  - Amyotrophic lateral sclerosis.
  - Glosso-labio-laryngeal paralysis.
- Atrophy complicating other disease of the cord . . . . . .
  - Atrophy in myelitis.
  - Atrophy in compression of the cord.
  - Atrophy in multiple sclerosis.
  - Atrophy in tabes dorsalis.

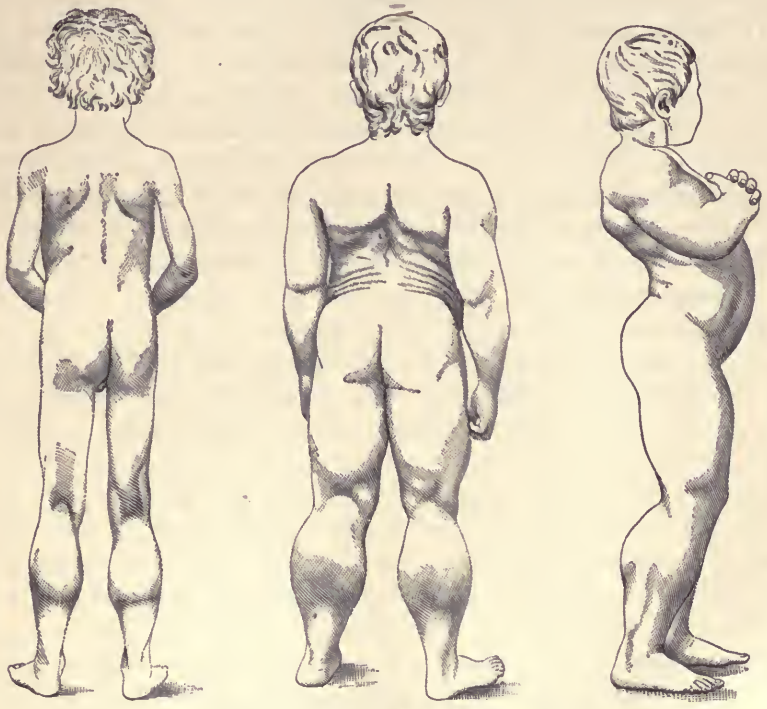
We shall here consider only the idiopathic muscular atrophy, the primary muscular dystrophy of Erb. The disease is characterized by muscular wasting with or without an initial hypertrophy. The essential change is in the muscles themselves, and as hereditary influences play an important rôle, it is probable that there is some inherent defect in the germinal tissues from which the muscular system is developed.

Etiology.—Congenital tendencies exist in many instances, and the disease is met with in family groups or many members are attacked through several

generations. As other family affections, the disease may be transmitted by a mother in whose family the disease exists, but who is herself exempt. As a rule, the disease appears during the stage of development, sometimes very early, just as the child is beginning to walk. In other instances the first symptoms may appear after adolescence. Other than hereditary no etiological factors are known.

Clinical Forms.—(*a*) *Pseudo-hypertrophic Type.*—The pseudo-hypertrophic muscular paralysis is a well-characterized and readily recognized affection, on account of the increase in size of certain of the muscle-groups. The child is first noticed to be clumsy in its movements, to fall easily, and to stumble in going up stairs. Nothing may at first be noticeable, but soon the attention is attracted to enlargement of certain of the muscles, particularly of the calves of the legs, which may stand out with extraordinary prominence. (See Fig. 58.)

FIG. 58.



Pseudo-hypertrophic Muscular Paralysis.

The extensors of the leg, the glutei, the lumbar muscles, the deltoids, the triceps, and infraspinati, are the next most frequently involved. The last-named may stand out with great prominence. The muscles of the face, neck, and forearm are rarely involved, and in marked contrast to the spinal forms of primary muscular atrophy the intrinsic muscles of the hand, in this as in other varieties of muscular atrophy, are spared. With the hypertrophy of certain muscles or of groups of muscles there is wasting of others, and there may be, for example, great enlargement of the calves, with wasting and weakness of the extensors

of the legs and of the muscles of the shoulder-girdle and of the back. In moderately advanced cases the attitude when standing is characteristic. The legs are far apart, the shoulders thrown back, the spine curved, and the abdomen protuberant. Owing to the loss of power in the extensors of the hip, the gait has a remarkable waddling, oscillating character, "in which the body is so inclined as to bring the centre of gravity over each foot, on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination toward the leg that is off the ground unless the balance is exact" (Gowers). In getting up from the floor the various positions assumed are quite pathognomonic, and have been rendered very familiar by the frequent reproduction of Gowers's well-known figures. The child turns over in the all-fours position, raises the trunk with the arms, gradually moves the hands along the ground until the knees are reached; then with one hand upon a knee he lifts himself up and grasps the other knee, and gradually pushes himself into the erect posture, climbing up his legs, as it is said.

The course of the disease is slow and progressive. The pseudo-hypertrophic muscles gradually waste. The greatest weakness, however, may be in muscle-groups which have not been primarily enlarged. At a late period contractures, lateral curvature, and various forms of talipes are present.

(*b*) *Primary Atrophic Form.*—A number of types of muscular atrophy have been described, chiefly according to the muscle-groups which they involve, all of which are merely varieties of one and the same affection, and differ only in their characters from the pseudo-hypertrophic type in an absence of the primary enlargement. They are all connected by intermediate forms with each other, and all occur in family groups. As a rule, the type which prevails in a family is the same in the different members affected, but occasionally in one family examples are met with of the pseudo-hypertrophic and the simple atrophic types. The following are the important types of the simple atrophic form:

Juvenile Form of Erb.—In this the affection begins as a rule about puberty, and involves the muscles of the upper arm and shoulder and of the gluteal and thigh groups. The deltoid is often spared. The calf muscles may be enlarged and hard. Later the back muscles are involved, and the patient assumes on standing the characteristic attitude mentioned in the pseudo-hypertrophic form. There are no special electrical changes, and even when the wasting is extreme the reaction of degeneration is not present. Several children in the same family may be attacked, and, as in other forms, the disease is with occasional interruptions progressive and leads to complete disability.

Infantile type of Duchenne, the *facio-scapulo-humeral* form of Landouzy and Dejerine, in which the disease sets in as a rule in childhood, but may be delayed until after the twentieth year. It is characterized by involvement of the facial muscles, so that the lower face and forehead are expressionless, and move slowly in laughing. The lips are often protuberant, thick, and everted, and cannot be pursed as in the act of whistling. The eyes cannot be com-

pletely closed. The muscles of the shoulder-girdle waste and the larger muscles of the thighs. Erb calls attention to the valuable test of the strength of the shoulder-girdle muscles in lifting the child by the arm-pits, when in weakness of these groups no resistance is offered and the shoulders are forced up almost to the child's ears.

Intermediate forms and types are met with, but do not, however, need special description.

In all forms the onset of the disease is gradual, and the wasting and weakness produced simultaneously in the various muscle-groups already mentioned. A striking feature is the absence of wasting in the intrinsic muscles of the hands—a contrast to the spinal form of muscular atrophy. The muscles of the tongue, pharynx, larynx, and eye are not affected. In all varieties the electrical irritability of the muscles is lessened in direct proportion to the wasting. There is no reaction of degeneration. Fibrillary twitching is rarely present. The sensation is unimpaired. The reflexes are weak; in the later stages lost, never increased. The sphincters are not involved. Late in the disease deformities occur, such as curvature of the spine and various forms of talipes.

The disease persists for an indefinite period, and it may be many years before the patient is bed-ridden.

Morbid Anatomy.—The spinal cord and peripheral nerves have been found normal, but Erb thinks that there are certain features which point to central dynamic changes as the cause of the atrophy, such as heredity, the special localization, and the existence occasionally of associated disorders of the brain, such as idiocy and epilepsy. In the pseudo-hypertrophic form the muscle-fibres present great variations in size. In the early stage there may be marked enlargement and the nuclei of the sarcolemma are increased. The fibres have sometimes been seen to be fissured longitudinally. The enlargement is chiefly due to the increase in the connective tissue and fat, by which in the later stages the muscle may be largely replaced. In the primary atrophic form a similar enlargement of the muscle-fibres has been noticed. The increase in the interstitial tissue is not so striking. The wasting of the fibres and the replacement by connective tissue and fat seem to be gradual processes.

Diagnosis.—The pseudo-hypertrophic form is recognizable at a glance. The striking contrast between the athletic appearance and the feeble condition, the attitude, gait, and mode of rising from the floor, make up an unmistakable symptom-group. It is to be remembered, however, that the gait and the mode of rising may be quite as characteristic of the simple atrophic forms. The occurrence in family groups is also a point of great importance. From myelopathic or spinal muscular atrophy the forms are usually easily separated. In the atrophy of chronic poliomyelitis anterior the small muscles of the hand are, as a rule, first attacked, whereas in the primary myopathies the muscles of the calves or of the shoulder-girdle or of the thighs are first involved. In spinal atrophy the reaction of degeneration is present and fibrillary twitch-

ings are more constantly met with. In addition to the wasting there is not infrequently increase in the reflexes and a spastic condition of the legs. The myelopathic atrophies come on late in life; the myopathic, as a rule, in childhood or adolescence. Heredity plays an important rôle in the primary muscular atrophies.

The Peroneal Type of Muscular Atrophy.—This form, which has been described by Charcot and Tooth, was at first regarded as a myopathy, but presents differences which make it doubtful whether it should come into this category. The wasting begins in the muscles of the legs, usually in the extensors of the great toe, afterward in the common extensors and in the peronei muscles. The small muscles of the foot may be early affected. Later the thigh muscles are involved. The unequal involvement of the legs in early childhood very commonly results in club-foot. The disease progresses slowly, and it may be years before the muscles of the upper extremities are involved. Here the order of attack is quite unlike the myopathic form, as the the thenar and hypothenar and interossei are first involved, often symmetrically, producing in some cases the claw-hand. Fibrillary contractions and the reaction of degeneration may be present. Sensory disturbances have also been noted, particularly impairment of tactile sensation, pains, more rarely vasomotor changes.

The essential nature of this form is still in doubt, but it may possibly be a neuritic disorder. Males are attacked much more frequently than females. Heredity has been present in many cases, usually through the mother.

The outlook in the primary myopathies is unsatisfactory, and it does not appear probable that any drug or form of treatment influences in the slightest degree the slow but progressive course of the disease. The general health should be attended to, moderate exercise allowed, and the muscles may be stimulated by massage and electricity. When the patient becomes bedfast care should be taken to prevent contractures in awkward positions.

Thomsen's Disease (Myotonia Congenita).

Definition.—An hereditary affection characterized by tonic cramps in the muscles on attempting to perform voluntary movements. The disease has received its name from the physician who first described it, in whose family it has been hereditary for several generations.

Etiology.—The disease appears to be most common in Scandinavia and in Germany. A majority of the cases have occurred in family groups. The sexes are equally involved.

Symptoms.—The disease sets in early, and it is noticed that on account of stiffness of the movements the child is unable to take part freely in games. In some instances the peculiar symptoms are not noticed until after puberty. The characteristic feature of the disease is only noticed in the performance of a voluntary movement. The muscles become rigid and fixed on attempting to move, or the contraction which the patient wills is very slowly accomplished and the relaxation is also slow. In walking the start is difficult: one leg

is put forward slowly, and for perhaps a second or two is stiff and can scarcely be moved. Gradually this rigidity wears off, the legs become limber, and the patient can walk for hours without fatigue. After a period of rest the same initial difficulty is experienced. The muscles of the arms and legs are those implicated. Motion and cold aggravate the condition. Sensation and the reflexes are normal. In some instances there have been mental changes, such as hypochondria. The patients are well nourished and appear muscular. In some instances there has been great increase in the muscular strength; in others the strength is scarcely in proportion to the size of the muscles. The muscles are very sensitive to mechanical stimuli, and pressure upon them may be followed by a tonic contraction lasting many seconds. Erb has described a characteristic reaction of the nerve and muscle to the electrical currents, the so-called myotonic reaction, the chief feature of which is that the contractions caused by either current attain their maximum slowly and relax slowly, and that vermicular wave-like contractions pass from the cathode to the anode. The condition persists throughout life. In a few instances it has been arrested temporarily, and there have been changes and variations in the intensity of the manifestations.

No post-mortem has yet been made upon the disease. Many examinations have been made of excised portions of muscle, and in all instances the fibres have been found to be greatly increased in size, and in some instances accompanied with an increase in the nuclei of the sarcolemma and of the interstitial tissue. The true nature of the disease is unknown, but it is usually placed among affections of the muscles.

No treatment for the disease has yet been found.

Affections which may be regarded as varieties of Thomsen's disease are on record; thus, Eulenberg reported a series of cases, the history of which could be traced through six generations, in which there were tonic spasms of variable duration, affecting chiefly the muscles of the face and less often those of the extremities. The contractions are followed by weakness. As it appeared in some members of the family shortly after birth, he termed it congenital paramyotone. In other instances, as in a case described by Gowers, the tonic spasm was associated with distinct ataxia.

Paramyoclonus Multiplex.

This is an affection first described by Friedreich, characterized by clonic contraction, chiefly of the muscles of the extremities, occurring usually in paroxysms.

A majority of the cases have been in male adults. The disease has most frequently followed fright or violent emotion or an injury. The clonic spasms usually begin in the muscles of the legs, and may at first not be severe enough to prevent the patient from working, and they can be to a certain measure controlled. They are, as a rule, bilateral, and vary from fifty to a hundred and fifty in a minute. In the intervals between the attacks there may be tremors in the muscles. Sometimes the contractions are definitely rhythmical.

In the severer attacks the spasms involve the muscles of the back and of the abdomen, and may become very violent, so that it is difficult to keep the patient in bed.

The course of the disease is variable. The spasms may recur at intervals for years, and in a majority of the cases recovery ultimately takes place. It may only be a manifestation of hysteria. Many of the cases have yielded promptly to powerful electrical currents.

VASO-MOTOR AND TROPHIC DISORDERS.

By WILLIAM OSLER.

Raynaud's Disease.

Definition.—A vascular disorder characterized by three stages, or, more properly, three grades of intensity: local syncope, local asphyxia, and local or symmetrical gangrene. The nature of the disease is still obscure, but it is, in all probability, a vaso-motor neurosis. Raynaud's original papers and an exhaustive summary of the literature will be found in Barlow's translation for the New Sydenham Society.

Local Syncope.—This is the most common manifestation, and produces in the extremities a condition properly known as dead fingers and dead toes. It is most frequently met with in women of a nervous temperament. The condition is present usually only in cold weather, and follows slight exposure. Occasionally an emotion causes it, as fright or an hysterical outbreak. Both hands may be affected, or one hand alone, or one or two fingers, sometimes the nose and the ear. The affected parts become cold, bloodless, and of an alabaster whiteness. The fingers and hand are stiff and move with difficulty, but are rarely painful except when the syncope is passing off. After persisting for a variable time the pallor gradually disappears, and the affected parts become either of a normal color, or the blood-vessels relax and there is a condition of great engorgement or local asphyxia. The two phenomena may be present in adjacent fingers, one being as white and as cold as marble, the other of a deep plum color. Local syncope is not often serious, and may recur for years during the winter season.

Local Asphyxia.—This may come on independently or follow local syncope. The fingers and toes are most often involved, then the ears and nose, and less often portions of the skin on the arms, legs, and trunk. The affected region becomes livid, deeply cyanosed, and the fingers and toes may be blue, cold, and on pressure the capillary circulation is almost absent, the white mark made by pressure of the finger disappearing very slowly. On the limbs and trunk the disease appears in livid or mottled patches, slightly raised, and sometimes surrounded by a zone of œdema or of hyperæmic skin. The hands may be involved, sometimes the hands and feet, and, as mentioned above, some of the fingers may be in the condition of local syncope, while others are deeply cyanosed. There are usually swelling and some pain, due to tension of the parts. In some cases there is marked anæsthesia and move-

ment is much impaired. The attacks last a variable time, and as the cyanosis passes off the parts become of a bright-red color, in which the circulation is extremely active. In adjacent fingers one may be in the reaction stage of a bright scarlet red, and the anæmia produced by pressure is instantaneously obliterated, whereas the other finger may be of a deep plum color, with almost complete stagnation of the capillary circulation. When the local asphyxia is persistent in the fingers or the ear-tips, there may be slight loss of substance, or in extreme grades the condition passes on into local gangrene. The attacks recur at irregular intervals, and are most frequent in cold weather. Some patients are entirely free in the summer months. Sometimes the condition recurs with striking periodicity. As in local syncope, the disease is perhaps most frequent in women. Some of the attacks are not unlike ordinary chilblains. The condition may recur for years without leading to serious trouble; thus, a woman of about thirty has had in the winter months for fourteen or fifteen years almost daily some grade of local asphyxia in the hands, which are sometimes livid and cold to the wrists. The fingers show only slight superficial losses on the tips. In some of the severer attacks mortification of the whole hand would appear to be almost inevitable.

Local or Symmetrical Gangrene.—The mildest grade of this condition is seen in those cases of local asphyxia in which small necrotic areas appear on the tips of the fingers or on the tips of the ears. Sometimes the terminal phalanges are quite cicatricial from repeated slight losses of this kind. In severer attacks the local asphyxia, which may be either primary or follow the syncope, persists. If in the extremities, the terminal phalanges become black, cold, and insensible. One finger only may be affected, or several fingers, or the entire hand or foot. More or less pain accompanies this. Instead of disappearing within twenty-four hours, the condition persists, and small blebs appear on the skin, which may be quite superficial. Sometimes without any formation of blebs the skin becomes dry and mummified. A line of demarcation is gradually formed, and a portion of one or more of the fingers sloughs away. In very severe cases the gangrene may involve several fingers, or the tip of the nose may be lost or a portion of an ear. It is important to note that the loss of substance is very much less than the appearance of the affected limb would indicate. The gangrenous blebs may be quite superficial and result in only necrosis of the skin. In one instance in which the feet were completely livid and superficial blebs appeared on the instep and the toes were livid and insensible, instead of an extensive loss of substance, as had been expected, the condition cleared and there were only superficial abrasions. Perhaps the most serious cases are those in which the patches of gangrene affect the symmetrical regions in different parts of the trunk and extremities. This severe type is most apt to occur in children. The affected regions are not necessarily symmetrical. Some of the cases have been preceded or accompanied with purpura of the skin and with hæmorrhages from the mucous surfaces.[1]

[1] Musser, "Grave Forms of Purpura Hæmorrhagica," *Transactions of the Association of American Physicians*, vol. vi.

A majority of these cases are fatal, and death may follow within three or four days.

The **prognosis** as regards death is, as a rule, good, except in the more malignant forms, and, as already mentioned, the destruction of tissue is, as a rule, very much less than the appearance of the affected part would warrant. The outlook for complete recovery is not very hopeful.

Associated Conditions.—There are very remarkable concomitant symptoms in Raynaud's disease to which of late much attention has been paid. Hæmoglobinuria may occur during an attack or may take the place of an outbreak. The blood-coloring matter is not always present. There may be only albuminuria. In a case which has been at my clinic on several occasions the local asphyxia with slight loss of substance in the ears recurred for three successive winters, always in association with hæmoglobinuria. The attacks were usually preceded by a chill, and during them he had a peculiar sallow, subicteroid hue. The relations between paroxysmal hæmoglobinuria and Raynaud's disease is evidently very close, and some have regarded them as manifestations of one and the same affection.

Colicky pains, diarrhœa, nausea, and vomiting occasionally occur in Raynaud's disease, but are not so common as in intermittent hæmoglobinuria and angio-neurotic œdema.

Peripheral neuritis has been found in a few instances of symmetrical gangrene, and there are instances in which the affection was associated with wrist-drop. In a recent case the patient, an alcoholic, had had for some time numbness and tingling and formication in the hands and feet. This was followed by a condition of the most intense local asphyxia of the hands and of the toes, and scattered patches resembling erythema nodosum upon the skin of the extremities and the trunk. Together with these symptoms there were slight articular pains and swelling of the right knee, so that the condition resembled somewhat peliosis rheumatica. Urticaria, erythema nodosum, and scleroderma have been described in connection with this affection.

Among the most remarkable are the cerebral manifestations. Mental torpor and transient loss of consciousness have been described. In the case above mentioned with hæmoglobinuria the patient had epileptic seizures with the attacks. Exposure on a cold day would bring on a fit, with local asphyxia of the ear-tips and bloody urine. Acute mania has developed, and delusions. A case has recently been under observation in which during the attack there was aphasia with temporary hemiplegia. Dimness of vision has been noted, and retinal changes, chiefly great narrowing of the arteries, have been described.

The **pathology** of this remarkable disease is not very clear. Raynaud suggested that the local asphyxia was caused by contraction of the vessels, which probably in the extreme grades of local syncope involves arteries, veins, and capillaries. The asphyxia is dependent upon dilatation of the capillaries and small veins, probably with the persistence of some spasm in the arterioles. Necrosis only follows when the condition is persistent, and in the prolonged

stasis the vitality of the tissues becomes lowered beyond power of restitution. In all probability the remarkable cerebral symptoms are caused by local spasm in special vascular territories.

Treatment.—So far as my own experience goes in a number of protracted cases, internal remedies have little or no influence. When hæmoglobinuria has been present and anæmia develops, iron should be given. Galvanism is recommended by Barlow, who advises immersing the affected limb in a basin of warm salt water, in which the negative electrode is placed, while the positive is applied over the spine. This sometimes relieves the pain. In an attack the affected limb should be raised and kept wrapped in cotton wool. The pain may be intense enough to require morphine. As the condition improves systematic frictions with sweet oil will be very useful.

Angio-neurotic Œdema.

(Giant Urticaria.)

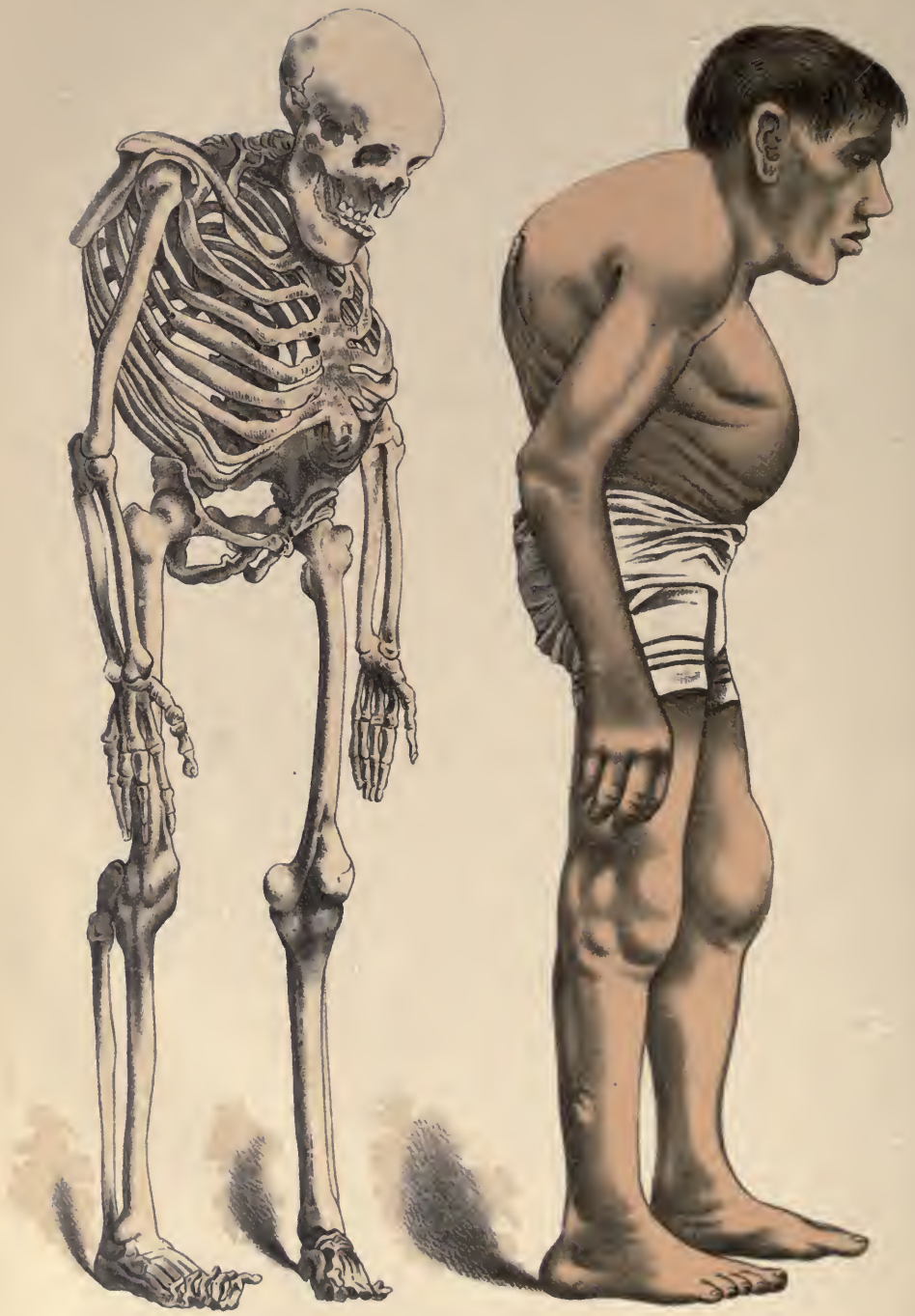
Definition.—A disorder characterized by an outbreak of œdematous swelling of variable extent, sometimes accompanied by gastro-intestinal crises. The disease may show a marked hereditary disposition.

Symptoms.—The most common situation for the œdema is the face, and particularly on the eyelids; next in order, the nose. In obstinate cases it may appear on any part of the body. The mucous membrane of the lips and pharynx may be attacked, and in a few instances there has been sudden and fatal œdema of the glottis. The œdema may reach rapidly a very high grade, and the skin is tense, shiny, pale, and on pressure pits readily. Usually the condition is transient and the œdema disappears within a few hours. The onset may be abrupt without any previous distress. In many instances the attack only occurs when there is gastric disturbance. The disease may recur with curious periodicity; thus in the case described by Matas the attack came on every day at eleven or twelve o'clock. The hereditary form is very remarkable. In the family described by me twenty-two members were affected in five generations. The swellings appeared in various parts, usually on the hands, face, or genitalia. Heat, redness, itching, and in some instances true urticaria, preceded the outbreak. Two members of the family died of œdema of the glottis. In all the cases in this group the gastro-intestinal symptoms were most pronounced—colicky pains, nausea, vomiting, and sometimes diarrhœa. The colic was severe enough to require hypodermics of morphine.

Quincke regards the condition as a vaso-motor neurosis causing sudden increase in the permeability of the vessels. The affection has close affinity with urticaria and with the form of purpura associated with gastro-intestinal crises and arthritis.

The **treatment** should consist of general tonics. Strychnine, antifebrin, and antipyrine may be tried, as they sometimes seem to have marked influence in checking the outbreaks of urticaria. The diet is of great importance in the

PLATE III.



Case of Acromegaly (Marie).

Geo.S Harris & Sons Lith. Phila

cases, and the outbreaks may be definitely connected with indigestion or with overeating. Local treatment is not of much value. When the œdema is very great and the tension painful, careful puncturing gives relief.

Acromegaly.

Definition.—An affection characterized by hypertrophy of the hands, feet, and face. The name, which signifies large extremities, was given by Marie of Paris.

Etiology.—The condition occurs more frequently in women. Of 38 cases in the monograph of Sousa-Leite, 22 were in women and 16 in men. The affection begins, as a rule, at about the twenty-fifth year, occasionally earlier, in some instances as late as the fortieth. It has no apparent connection with sexual processes. Heredity has apparently played a part in some cases. Syphilis, the specific fevers, and rheumatism have occasionally preceded the development of the disease, but probably have no special connection with it. In this country only a few cases have been reported.

Symptoms.—A well-marked case presents a very characteristic appearance. (See Plate III.) The hands and feet are enlarged, the increase in size involving both bones and soft parts and giving a spade-like character to the hands. The wrists may be enlarged, but the arms are not often affected. The feet are uniformly enlarged, and the size of the big toe has been in some cases out of proportion to the others. The nails are usually broad and large and vertically grooved. Although they look clumsy and unwieldy, the hands can be used freely, and even such a delicate operation as threading a needle can be well performed. The long bones, as a rule, are unaffected, but in some instances there has been thickening of the extremities of the femur and of the tibia and the fibula. The enlargement involves the head and face, particularly the latter, which becomes elongated and broadened in consequence of the increase in size of the superior and inferior maxillary bones; the latter in particular increases, and may project beyond the upper jaw. The alveolar processes become greatly thickened and the teeth are separated. The lower lip is thickened and the ears greatly hypertrophied. The tongue has in some instances been greatly enlarged. Increase in the size of the bones of the skull may be present. The neck looks short and thick, chiefly on account of the elongation and depression of the chin. The skin may look normal or is coarse and flabby. It rarely has the harsh appearance of myxœdema, and the subcutaneous tissues are not infiltrated. The bones of the thorax may slowly and progressively enlarge, and in a late stage of the disease the spinal column may be involved and there may be marked kyphosis. The clavicles and sternum may also increase in size. The muscles are sometimes wasted. The genitalia have sometimes been hypertrophied.

The thyroid has been normal in some cases, atrophied in others, and in a third group enlarged. Erb has noticed an area of dulness over the manubrium sterni which he thought to be possibly due to persistence of the thymus. In women menstrual disturbance may be an early feature, and there may be com-

plete suppression. The voice changes, partly on account of the enlargement of the tongue, partly because of changes in the laryngeal cartilages. Patients often complain of headache. There may be increasing dimness of vision, owing to a progressive atrophy of the optic nerve. Less commonly the sense of hearing and smell are deficient. The disease may last from ten to twenty years: a condition of cachexia ultimately develops and the patient dies of exhaustion.

The **morbid anatomy** of the disease has been studied in several cases. The enlargement of the bones is a true hypertrophy. The increase in the size of the face is largely due to dilatation of the antrum. The lesions are essentially different from those of the osteitis deformans of Paget, in which the shafts of the bones are chiefly involved, and also from arthritis deformans. The pituitary body has been found hypertrophied; less commonly changes occur in the thymus and thyroid. The peripheral nerves have been found degenerated in several cases. The nature of the disease is still in doubt. According to Marie, it is a systemic dystrophy analogous to myxœdema, and associated possibly with the changes in the pituitary body, just as myxœdema is connected with disease of the thyroid gland. The most exhaustive description of the pathological anatomy and a discussion of the relation of the affection to other forms of enlargement of the bones will be found in the essay of Arnold of Heidelberg.[1]

Diagnosis.—The disease must not be confused with myxœdema, in which the bones are not enlarged. It is most likely to be confounded with the osteitis deformans of Paget, in which disease, however, the shafts of the long bones are chiefly affected, and in the head the cranial, not the facial, bones are enlarged. In the cases of congenital progressive hypertrophy, the so-called giant growth, as a rule, only a single member is involved and the shafts of the long bones are affected. According to Marie, the face in Paget's disease is triangular, with the base upward; in acromegaly it is ovoid or egg-shaped, with the large end downward; in myxœdema it is round and full-moon-shaped. Marie has separated a group of cases characterized by hypertrophy of the bones of the extremities and of the shafts, associated in some way with disease of the lungs. The condition of the fingers differs from that of acromegaly, as the phalanges are bulbous and enlarged and the nails curved, very different from the flattened terminal phalanges of acromegaly. Curvature of the spine is also common. The cases have been met with in connection with purulent pleurisy, with new growths in the lungs, and with chronic bronchitis. Marie terms it osteo-arthropathie pneumique. Arnold does not, however, regard this as a form which can be definitely separated from acromegaly.

A curious dystrophy, met with only in women, involving the fingers and toes, is the sclerodactyle. The lesions are, as a rule, symmetrically distributed. The fingers are atrophied and shortened, the skin is thickened and waxy, and the nails are deformed and small and often curved. The

[1] *Ziegler's Beitrage*, 1891.

fingers may be dislocated, and the joints become ankylosed in irregular positions, so that the fingers are distorted. Ankylosis of the wrist and of the ankle have been observed. In some instances there have been diffuse sclerodermatous changes in the skin in other parts. The causation of the disease is unknown. Many of the patients have suffered severely with cold, and the condition is much worse during the winter. In the only autopsy which has been made no clue was obtained as to the nature of the trouble. A good description of the disease has been given by Gordinier.[1]

No **treatment** has been found of any avail in acromegaly or the allied conditions.

Scleroderma.

A condition in which the skin becomes tense and hard, either in circumscribed patches or in extensive diffuse areas. These two forms, the circumscribed and the diffuse, may exist together or separately. The circumscribed scleroderma corresponds to the affection known as morphœa and to the keloid of Addison. In patches ranging from half a centimetre in diameter to the size of the hand the skin is hard, brawny, inelastic, and has a waxy, dead-white appearance. These patches occur most frequently in women about the breasts and neck, occasionally in the course of the nerves either in the trunk, the intercostal, or lumbar, and on the face in the branches of the fifth. A preliminary hyperæmia may precede the development of the patches, and in some instances there are changes in color due to increase in the pigment of the skin. In other cases there is complete atrophy of the pigment and leucoderma. Preceding the onset there may be itching or irritation of the skin, and, when fully developed, anæsthesia is occasionally present. The sweat secretion is either diminished or completely abolished. There are instances in which the disease begins with the development of small linear, cicatricial-like spots—lineæ atrophicæ. A curious, remarkable feature is the rapidity with which the patches appear and their variability. They may persist for months or years with but slight change, and after lasting for some time may disappear in a few weeks. According to Crocker, "The pathology appears to be that, owing probably to some defect in innervation, an exudation occurs round the vessels, narrowing the lumen, obstructing therefore the blood-flow, and leading to thrombosis and sometimes to a real rupture and effusion. Each atrophic spot seen near a growing patch is the base of a cone from which the blood-supply is cut off, the violet zone being due to collateral hyperæmia round an anæmic area. The patch or atrophic spot thickens by the fibrillation of the effused cells. Where the arterial supply is completely cut off an atrophic spot is produced; where it is merely diminished partial atrophy, with connective-tissue hyperplasia, or morphœa, is the result."

The diffuse scleroderma is more rare. It appears first in the extremities or on the face, and the skin becomes hard and tense, so that the patient has great difficulty in performing ordinary movements. When fully developed there is a brawny induration of such a degree of firmness that the skin cannot be

[1] *American Journal of the Medical Sciences*, Jan., 1889.

picked up or pinched. The wrinkles are effaced and the skin looks stretched, dry, and glossy. Dinkler, who has recently made a careful study of the condition from Erb's clinic, states that of 44 cases, in 24 the first appearances were on the arms; in 7 on the legs, in 1 on both; in 10 on the face and neck; and in 2 on the trunk. Gradually extending, the disease may involve the greater portion of the skin of the trunk or that of an entire limb. Occasionally it becomes universal. The joints are fixed in semiflexion, and movements are impossible on account of the hidebound condition. The face is expressionless, immobile, and it may be almost impossible for the patient to chew his food. The sensory changes are not marked, but during the development of the affection there may be great itching. The mucosa of the mouth and pharynx has been occasionally involved. The disease persists for months or years, and there are instances on record of its persistence for more than twenty years.

The disease is sometimes arrested, and in a few instances recovery has followed. Death usually results from intercurrent pulmonary affection or from nephritis. The nature of the disease is unknown. Some cases have been preceded by rheumatism; others have been met with in connection with endocarditis and rheumatic nodules. It is generally regarded as a tropho-neurosis, possibly depending upon changes in the arteries of the skin, and so leading to connective-tissue overgrowth.

As the patients are particularly sensitive to changes in the weather and to cold, they should be warmly clad, and when possible live in a mild climate. Frictions with oil and electricity have been recommended, and in the local forms galvanism seems to have been beneficial.

Allied to scleroderma is the remarkable affection known as sclerema neonatorum, in which, either at birth or shortly after, there are large areas of induration of the skin, which is tense and glossy and does not pit on pressure. It is sometimes associated with œdema of the subcutaneous tissues. The disease may spread rapidly, and the congenital cases are usually fatal. Recovery, however, is not impossible. In a remarkable case recently seen with Dr. Ellis of Elkton a healthy, well-grown child of two and a half months had an acute pleuro-pneumonia, during which, on the sixth day, it developed general scleroderma, the entire skin becoming hard and leathery, the legs stiff, and the whole body looking rather like a model of a child in wax.

Facial Hemiatrophy.

This is a rare affection, and less than one hundred cases have been reported in the literature. The wasting is on one side of the face—hence the name—is progressive in character, and involves bones and soft tissues. It begins, as a rule, in childhood, and the onset may be accompanied by pains and paræsthesia. It may start at one or two spots on the skin and gradually spread, or begin diffusely and gradually involve soft parts and bones. The atrophy is strictly confined to one side, and when fully developed gives a remarkable appearance to the patient, whose face looks made up of two unsymmetrical halves. The atrophy is strictly limited to the middle line. Sensibility is not

affected. The skin may be darker in color and the hair falls out. The teeth may become loose, owing to wasting of the alveolar processes. The movements of the muscles are rarely affected, though in Sachs' patient there were tonic and clonic contractures of the temporal and masseter muscles. The nature of the disease is still doubtful. In the autopsy in Homan's case, which came on rapidly and can scarcely be regarded as a typical illustration, a tumor was found pressing upon the Gasserian ganglion and the fifth nerve. In Mendel's case there was an interstitial neuritis in all the branches of the trigeminus from its origin to the periphery, most advanced in the superior maxillary branch. The disease is probably due to involvement of the trophic fibres of the fifth nerve.

The **prognosis** is unfavorable, as the affection is progressive. The cases of facial asymmetry in children associated with congenital wry-neck must not be confounded with progressive facial hemiatrophy.

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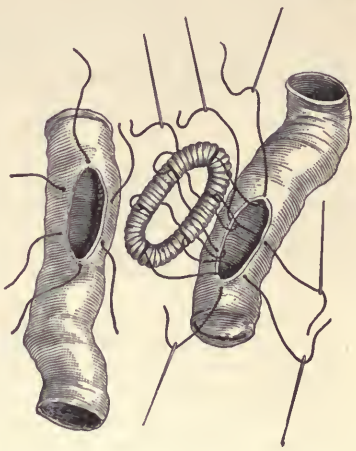

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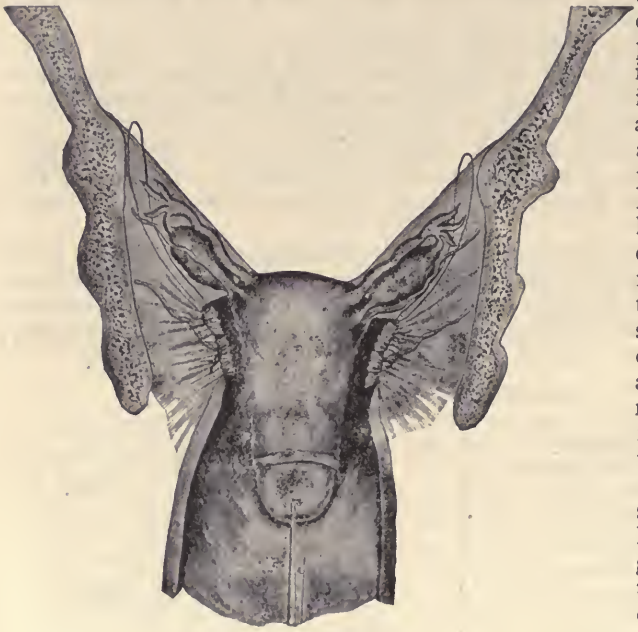

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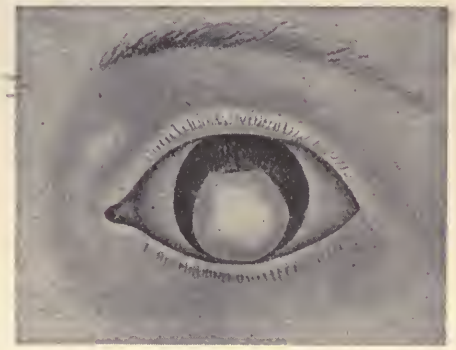

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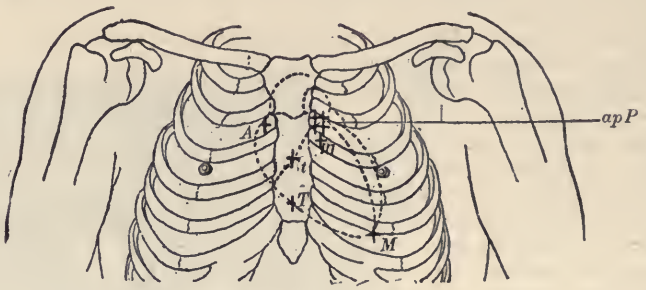

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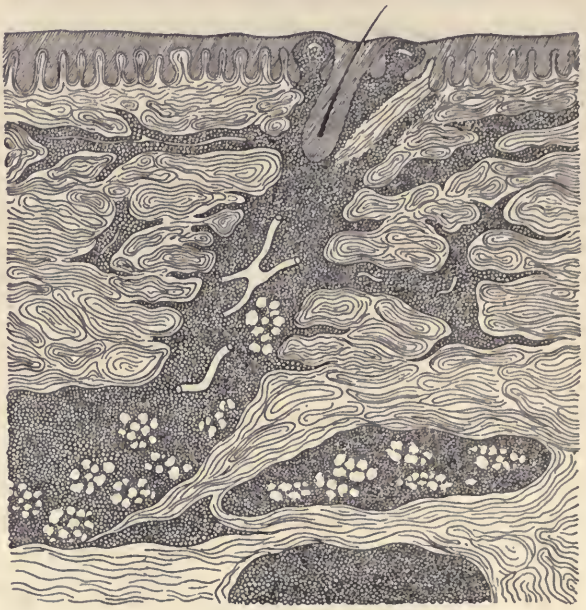

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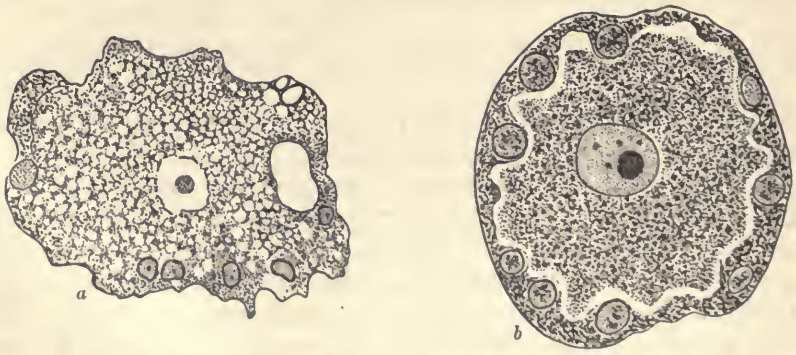

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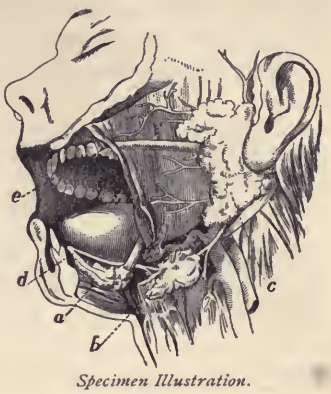

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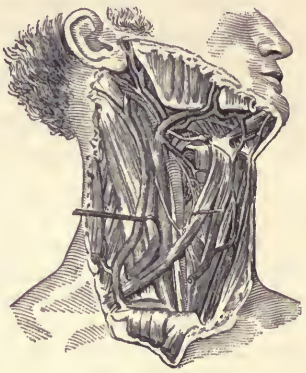
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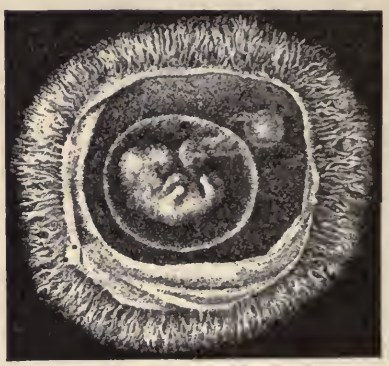

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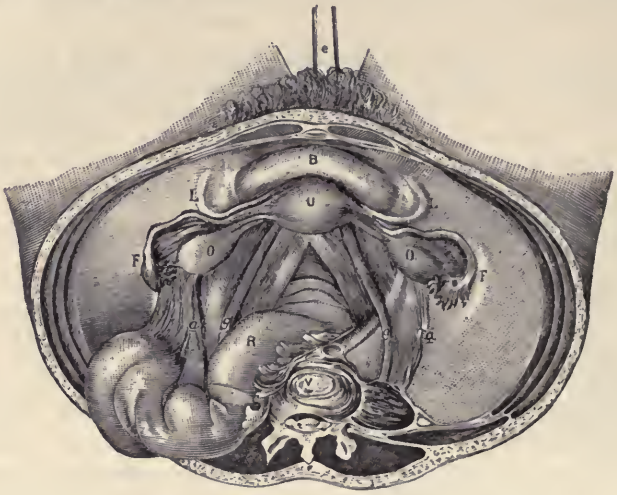

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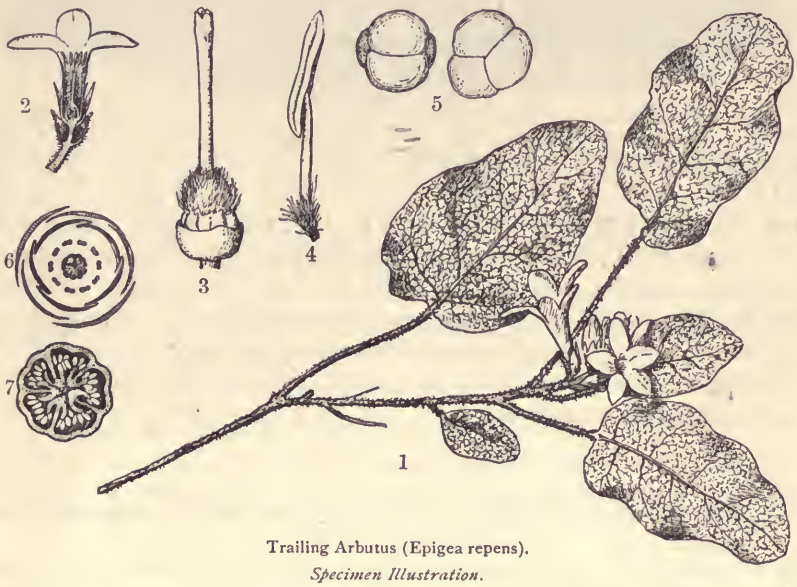

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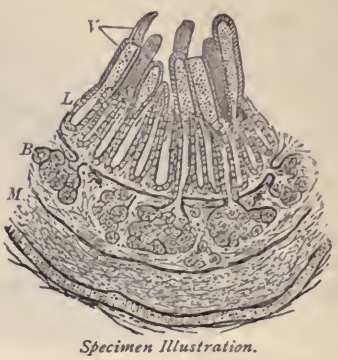

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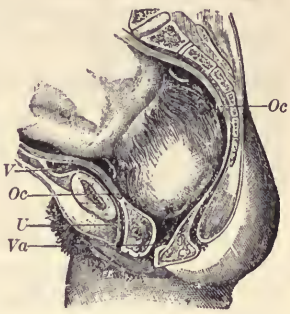

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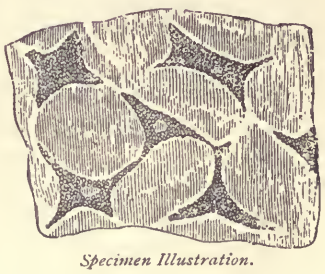
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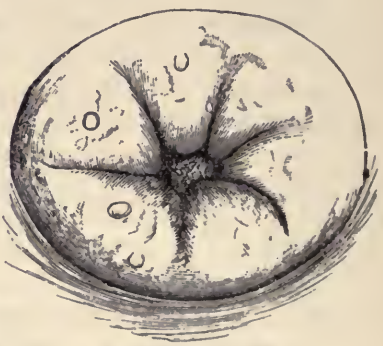

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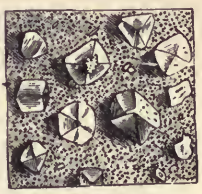

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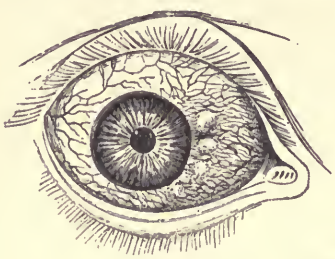

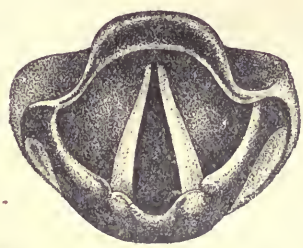

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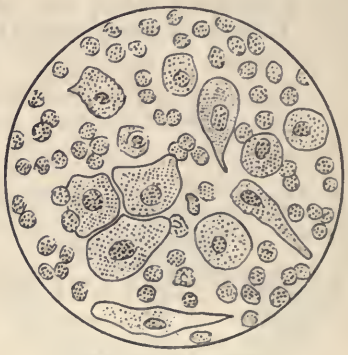

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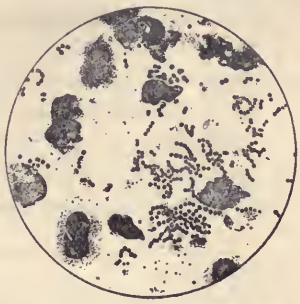

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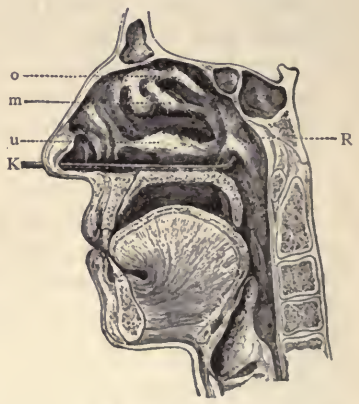

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